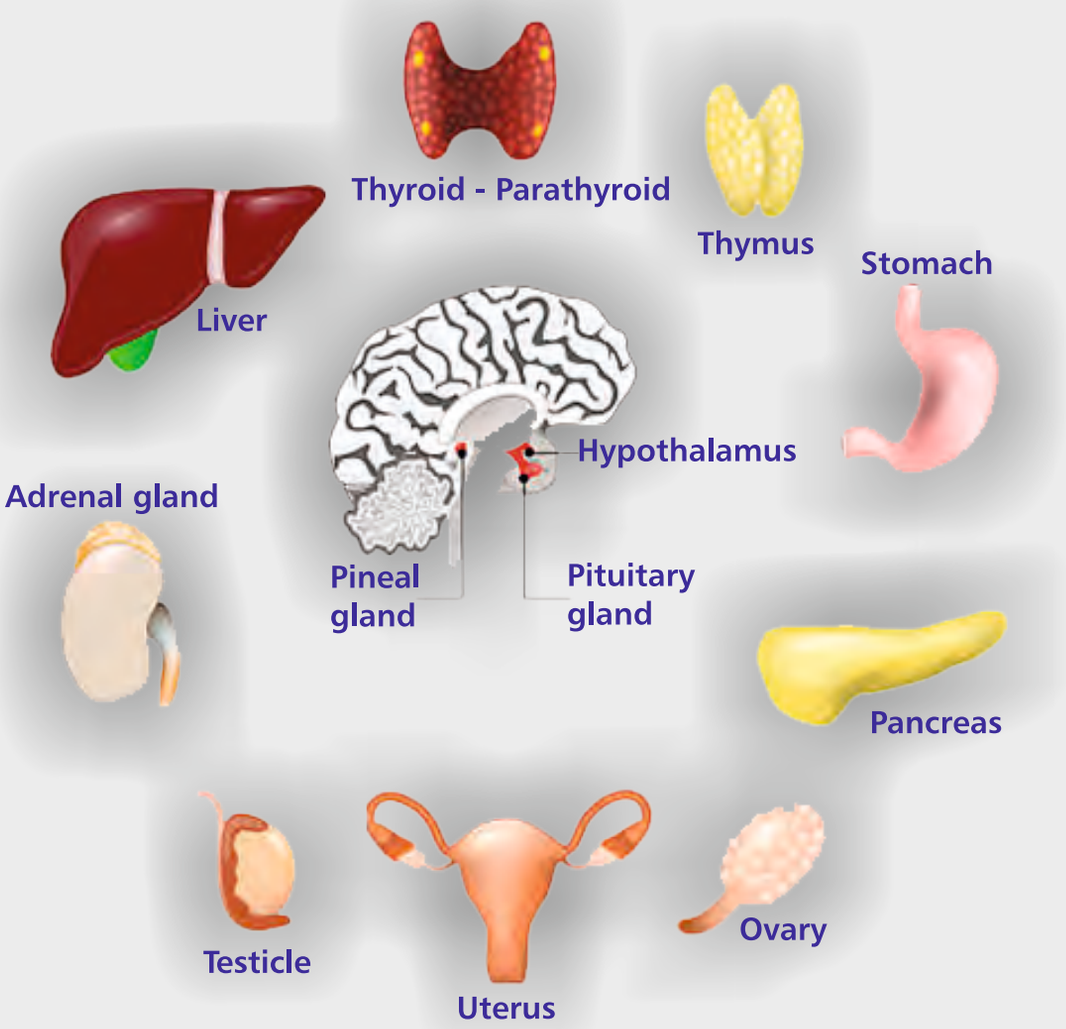
Pulse

Volume 19 March 2019

HORMONES, HEALTH AND MORE



CONTRIBUTORY HEALTH SERVICES SCHEME

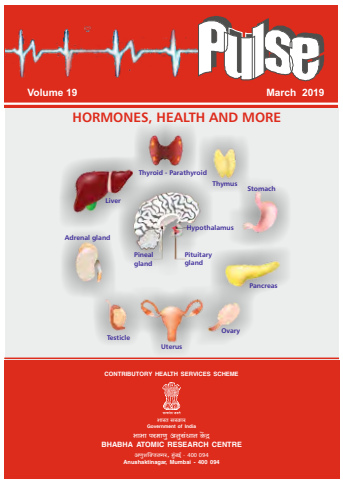
सत्यमेव जयते
भारत सरकार
Government of India
भाभा परमाणु अनुसंधान केंद्र
BHABHA ATOMIC RESEARCH CENTRE
अणुशक्तिनगर, मुंबई
Anushaktinagar, Mumbai - 400 094

MMC Accredited CMEs held in the year 2018

Sr No	Date	MMC CODE	Dept	Topics	Speakers	Venue
1	16.02.2018	MMC/MAS/2018/F-009635	DISPENSARY	Fever - I	Dr. Hakim P	Conference Hall - 1,
				Fever - II		
				Whats new in Diabetes	Dr. S Chandalia	BARC Hospital
2	20.04.2018	MMC/MAS/2018/F-10038	PATHOLOGY	"Diagnosis and advances in Management of Epilepsy"	Dr. Joy Desai	Conference Hall - 1,
			OPTHALMIC	"Interesting cases in Neuro Opthalmology"	Dr. Hemalini Sawant	
			MEDICINE	"Newer Biomarkers in Neuroimmunology"	Dr. Rajesh Bendre	BARC Hospital
3	06.07.2018	MMC/MAS/2018/F-10522	SURGERY	"CT Scan in Acute Abdomen"	Dr. Nimeesh Kamat	Conference Hall - 1,
			ANAESTHESIA	"Anaesthesia in non-OR setting"	Dr. Jitendar Bapat	
			RADIOLOGY	"Current Concepts in Management of Deep vein Thrombosis"	Dr. R Sekhar	BARC Hospital
4	07.09.2018	MMC/MAS/2018/F-11034	PSYCHIATRY	"Positive Psychiatry"	Dr. Vihang Vahia	Conference Hall - 1,
			PAEDIATRIC	"Infection control measures in tertiary care hospital"	Dr. Hiren Doshi	
			ENT	"COUGH: ENT Surgeon's perspective"	Dr. Samir Bhobe	BARC Hospital
5	26.10.2018	MMC/MAS/2017/F-11486	PALLIATIVE CARE	"Disease experience and serious health related suffering"	Dr. Jayita Deodhar	Conference Hall - 1,
				"Palliative care in Chronic Neurological conditions"	Dr. Roopkumar D G	
				"Concept of End of Life Care"	Dr. Seema Rao	BARC Hospital
6	21.12.2018	MMC/MAS/2018/F-11796	ANAESTHESIA	"Anaesthesia for Obese Patients"	Dr. Tasneem Dhansura	Conference Hall - 1,
			OBST. & GYN	"Diabetes in Pregnancy"	Dr. Uday Thanawala	
			PATHOLOGY	"Recent Trends in Transfusion Medicine"	Dr. Joyce Regi	BARC Hospital

Pulse

Volume 19 March 2019



CONTENTS

Clinical Meet Year 2018

Date	Unit/Section	Topic	Speaker
28.12.2018	Radiology	Interesting cases in CT scan	Dr Shubhra Gupta
14.12.2018	Ophthalmology	Retinopathy of prematurity	Dr Ronak Parekh
09.11.2018	ENT	Temporal Bone Fracture	Dr Sen P. A.
12.10.2018	Orthopaedic	Vit. D and Calcium preparations	Dr Hardik Shah
28.09.2018	Dispensary	Gestational Diabetes	Dr Vaishali Wadhe, (Kharghar)
14.09.2018	Anesthesia	Low Back Pain - How can a pain specialist help?	Dr Sanjog Mekewar, Dr Jalpa Kate
24.08.2018	Gynaecology	Ovarian Hyperstimulation Syndrome - OHSS	Dr Nigamananda Mishra, Dr Devika
10.08.2018	Pathology	Sepsis & Biomarkers in Sepsis	Dr Raja Silvan
27.07.2018	Surgery	Inflammatory Conditions of Breast	Dr Pratima Pimpalkar
13.07.2018	Psychiatry	Frontotemporal Dementia	Dr Yogesh Motwani, Dr Vijay Teli, Dr Apurva Ungratwar, Dr Krishna Prakash
22.06.2018	Dispensary	Constipation in Elderly	Dr Shailaja Madake, (Ghatkopar)
08.06.2018	Medical	1) Recent advances in epilepsy 2) Reversing neurological disability, stroke and treatable dementia	Dr Jayanti Mani, Dr Tushar Raut
11.05.2018	Ophthalmology	Eye Donation	Dr Monali Rathod
27.04.2018	ENT	Obstructive Sleep Apnoea	Dr Kumarswamy Salimath
13.04.2018	Gynaecology	Presentation on Chemical, Biological, Radiological and Nuclear (CBRN) Disaster Management	Dr Nigamananda Mishra
23.03.2018	Paediatric	Iron Deficiency Anaemia in Children	Dr Amol Chavan
09.03.2018	Orthopaedic	Bone Infection	Dr Praveen Bande
23.02.2018	Dispensary	Overview of Diabetes	Dr Sonali Shejul, (Andheri-W)
09.02.2018	Anaesthesia	Oxygen Therapy - Concepts and Current Guidelines	Dr Hemesh Shewale

Dear Readers,

After a prolonged interlude, the editorial team is back with the current issue of 'Pulse'. As the team compiled various articles and case reports for this issue, it became increasingly evident that most of the articles address endocrine dysfunction and their health impact. Thus, this issue is broadly based on the theme of 'endocrine health'. The word 'endocrine' is derived from Latin and means 'internal secretion'. The endocrine system includes all hormone secreting glands of the body; and affects almost every cell and organ of the body. It helps regulate growth, development, metabolism, sexual function, reproduction, sleep, mood and homeostasis among other functions.

In this issue, our guest article discusses the much ignored problem of 'Male Sub-fertility'. New age challenges like Ovarian Hyper Stimulation Syndrome associated with artificial reproductive techniques and hurdles in localizing parathyroid adenoma have been analyzed in our case report section. The write ups on role of computed tomography in diagnostics of endocrine dysfunction and anaesthetic management of adrenal tumors are highly informative. Other articles in this issue touch upon a variety of topics like retinopathy, dental care, culture contaminants, interesting cases and more.

Our 'Academic and Extracurricular Achievements' section showcases the accomplishments of medical division.

The editorial team strives to make 'Pulse' informative but above all, useful. I hope you enjoy reading this issue. Do let us know your feedback, suggestions and any topic you would like to see in future issues, at pulse@barc.gov.in

*Lectiobeatus!**

Dr. Shrividya Chellam
Chief Editor, Pulse.

**Happy Reading Latin*

The Indian Government has set a goal of universal health coverage for all by 2022. This concept was envisaged by Dr. Homi J. Bhabha way back in 1960 when he customized a unique contributory health scheme which gave universal health coverage, irrespective of job designation, seniority, financial status, socio-cultural factors or gender. Thus, the CHSS came into existence in 1962 for the benefit of all employees (present and retired) and their dependents.

Taking this vision further, the Medical Division, consisting of a 390 bedded multi-specialty hospital with 24/7 Casualty, 13 zonal dispensaries and 3 occupational health centers, has been consistently serving the ever increasing population of CHSS beneficiaries, which currently stands at 103064 persons. Of this, the geriatric population (>60yrs) is 29%, far more than the average geriatric population % of the developed countries like Japan, Europe, UK and US, while the Indian national average stands at a mere 9%. CHSS beneficiaries are well taken care of, with our per capita expenditure (2017-18) at Rs.24021 (average expenditure per person per year) while the Indian average per capita expenditure on public health is approx Rs.1112 per person per year (National Health Profile 2018, released by Union Min for health and family welfare, June 2018).

Over the years, the attendance at OPD, Casualty, Dispensaries as well as indoor admissions has steadily risen. Keeping the increasing demand for diagnostics services in mind, in house facilities have been increased. Keeping pace with the times, Medical Division & BARC Hospital has introduced new facilities.

1) *Blood collection facilities at zonal dispensaries and Occupational Health Center, Trombay.*
2) *Infertility Clinic since March 2018*
3) *Dental and Radio-diagnostics at Kharghar dispensary.*
4) *CT scan facility at BARC Hospital*
5) *Home care facility for palliative care patients in and around Anushaktinagar.*
6) *MRI facility is in the wings.*
7) *Picture Archiving and Communication System (PACS) is in the process of being installed which will replace hardcopy based medical scans, providing space advantage and will enable remote access of images for off-site viewing and reporting.*
8) *To augment the healthcare facilities and to serve the increasing beneficiaries, the new hospital building with state of the art amenities is coming up rapidly. Additional facilities of cardiac catheterization lab, cardiac surgery, upgraded AKD with dialysis unit and increased medical & surgical intensive care beds will be made available.*

We, at Medical Division, hope to continue providing healthcare service by following the winning combination of clinical excellence backed by cutting edge technology and medical ethics.

Dr. Kaustubh Mazumdar
Head, Medical Division

Male Subfertility

Subfertility is defined as an unwanted delay in conception after 1 year of regular unprotected intercourse. Most couples presenting with a fertility problem do not have absolute infertility (i.e. no chance of conception), but rather have relative subfertility with a reduced chance of conception in each cycle, due to one or more factors in one or both partners. Approximately 15% of the population, i.e. 1 in 6-7 couples, experience subfertility.

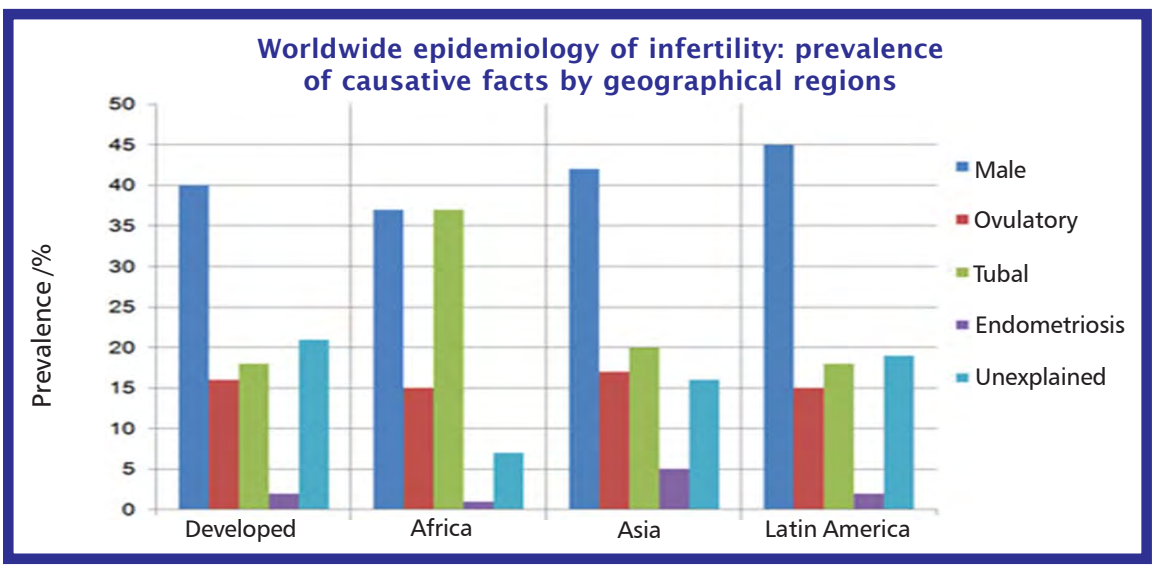
For many decades, the term subfertility is being used with the constant assumption of the word female in front of it, but now male subfertility has joined the mainstream medical discourse.

Reports of declining sperm counts and increasing incidence of urogenital abnormalities and testicular cancer from many different countries have stimulated public interest and concern about male subfertility. While modern lifestyles and environmental pollution have been incriminated in subfertility, whether there is a true deterioration of human semen quality is still a matter of some controversy.

Dr. Deepak Bhenki,
Infertility Specialist, BARC Hospital

The incidence of ovulatory disorders is about 15%. Tubal disease and male factor infertility are becoming more prevalent. Male factors now account for up to 40% of cases of infertility, and in about 20% of cases both male and female factors may coexist.

Although a battery of tests and treatment have been described and continue to be used in evaluation of female infertility, the male has been essentially neglected. Many factors accounts for this disparity. Gynaecologists have little training in the evaluation of the subfertile male. Many times examination of male is not



done as gynaecologists are not trained for it and urologist, who are trained, rarely take lead in this. Hence male factor remains neglected even if it is contributing about 40% of total subfertility issue.

What is Normal?

In 2016, the World Health Organization (WHO) revised the reference values for human semen characteristics that evaluate the male fecundity to 95th and 5th percentile from men who had fathered a child within 1 year of trying for a pregnancy. These values seem to lean towards lower sperm counts, low motility and lower morphology (compared with the previous WHO reference values in 1987, 1992 and 1999). This poses the question – is the male fecundity on the decline or is the fecundity maintained at these lower reference values?

Etiology of Male Subfertility:

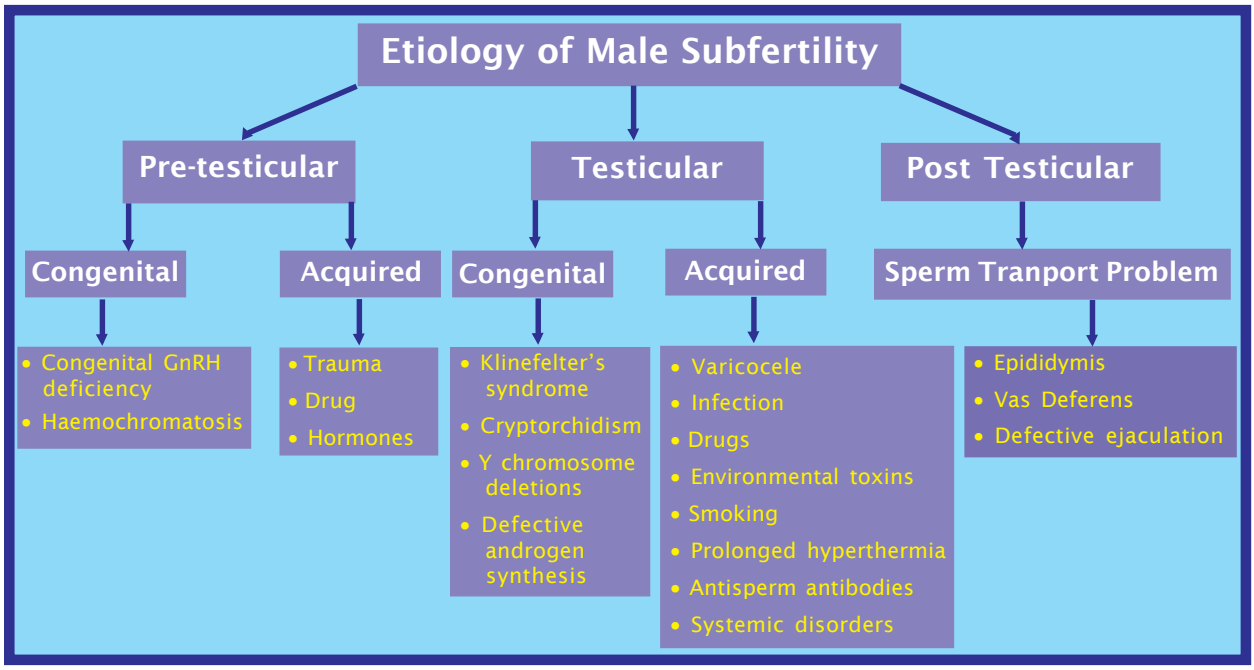
A quagmire of epidemiology, lifestyle, environmental pollution, systemic illnesses, medications, gene polymorphism and partner's fertility can all affect male fecundity.

WHO Reference Value (2016):

Volume	1.5ml (1.4-1.7)
Sperm concentration	15(12-16) million
Total sperm count	39(33-46) million
Total motility	40% (38-42)
Progressive motility	32% (31-34)
Vitality	58% (55-63)
pH	7.2
Morphology	4%(3-4)

The reason behind the male factor problems is unknown/idiopathic in a considerable proportion of cases. Causes can be broadly classified as:

- **Pre-testicular i.e. hypothalamic-pituitary disease**
- **Testicular**
- **Post-testicular**



- **Pre-testicular i.e. hypothalamic-pituitary disease**

Congenital	Congenital GnRH deficiency (Kallmann's syndrome), Genetic disorders that affect multiple organs (e.g. Prader–Willi and Laurence–Moon–Biedl syndromes), Haemochromatosis
Acquired	Pituitary and hypothalamic tumours, Trauma (surgery, irradiation) Vascular Infiltrative disorders (sarcoidosis, tuberculosis) Drugs/hormonal (prolactin, ↑ testosterone ↑ estrogen) Systematic disorders (chronic illness, malnutrition, morbid obesity)

- **Testicular**

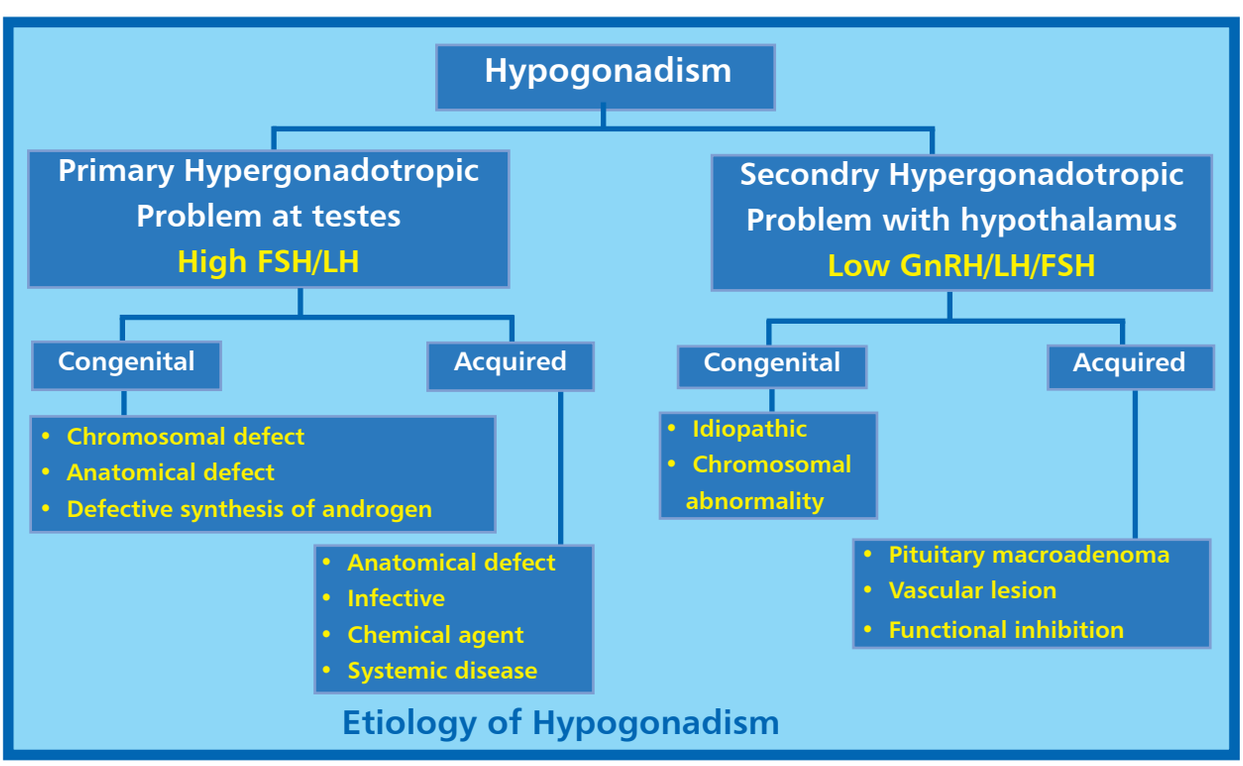
Congenital	Klinefelter's syndrome, Cryptorchidism, Y chromosome deletions, Defective androgen synthesis or response (5a-reductase deficiency, androgen insensitivity)
Acquired	Varicocele, Infection, Drugs, Environmental toxins, Smoking, Hyperthermia, Anti-sperm antibodies, Systematic disorders

- **Post-testicular**

Sperm transport problems	Epididymis, Vas Deferens, Defective ejaculation

Sometimes, men with subfertility have **Hypogonadism**. Hypogonadism is a combination of impaired spermatogenesis and testosterone production in the testicles after puberty. These men present with impaired semen analysis and decreased testicular volume.

Hypergonadotrophic (primary) hypogonadism (where the testes don't produce sperm/testosterone despite



Etiology of Hypogonadism

being stimulated by high levels of FSH and LH) is more common than hypogonadotrophic (secondary) hypogonadism (where adequate GnRH from the hypothalamus or LH and FSH from the pituitary aren't being secreted despite low circulating levels of testosterone).

- **Pre-testicular i.e. hypothalamic - pituitary disease (Secondary Hypogonadism)**

Congenital causes

Idiopathic hypogonadotrophic hypogonadism (commonly known as hypo-hypo) is characterised by isolated gonadotrophin deficiency. Hypo–hypo can be caused by either anatomic or functional problems. This results in sexual underdevelopment, eunuchoid body habitus and sometimes anosmia (Kallmann's syndrome). Other genetic disorders of gonadotrophin secretion include haemochromatosis, the Prader–Willi syndrome and the Laurence–Moon–Biedl syndrome.

Chromosomal abnormalities

Subfertile men with poor semen quality (i.e. low count, low motility and morphology) have increased frequency of chromosomal abnormalities. A sperm count of 10 million/ml has a ten-time higher incidence of autosomal abnormalities, which are numerical or structural (translocations). In a large series of subfertile men (Vincent MC et al 2002) the incidence of chromosomal abnormality was 5.8%. Sex chromosome abnormality was 4.2% and autosomal abnormality was 1.5% (Johnson MD 1998)

Acquired causes of hypothalamic-pituitary disease (Secondary Hypogonadism)

GnRH neuron destruction or hypothalamic - pituitary circulation interruption can be due to pituitary macroadenomas (mainly prolactinomas), craniopharyngiomas as well as the surgical therapy of these. Less common causes include vascular lesions and infiltrative diseases such as sarcoidosis and tuberculosis. Functional inhibition of GnRH or gonadotrophin secretion can be caused by hyperprolactinaemia, androgen, estrogen, or cortisol excess. Any cause of hyperprolactinaemia, drugs, hypothyroidism and prolactinomas may be incriminated. Androgen excess may be due to the abuse of anabolic steroids or due to congenital adrenal hyperplasia or tumours of the testis or adrenal glands. Estrogen excess may be due to estrogen producing testicular tumours. Any serious systemic illness or chronic nutritional deficiency can cause hypo-hypo and subfertility. Massive obesity has a similar effect.

- **Testicular disease (primary hypogonadism)**

Primary hypogonadism (hypergonadotrophic) can also be due to congenital or acquired pathology.

Congenital causes of primary Hypogonadism

- **Klinefelter's syndrome** has an incidence of one in 500–1000 in new-born males. An extra X chromosome (XXY) is the most frequent reason although cases of chromosomal mosaicism (e.g. XY/XXY) have been described. These men are tall, with cognitive and behavioural difficulties being common.
- **Cryptorchidism** is failure of descent of the testes into the scrotum during fetal development. This results in the testes being within the abdomen, inguinal canal, or other locations. The incidence is 2-5% in new-born baby boys and is higher in preterm infants. Both unilateral and bilateral cryptorchidism, are associated with impaired spermatogenesis and an increased risk of testicular tumours (relative risk of testicular cancer 7.5 compared with the general population). Whilst the

testes may descend spontaneously after birth (incidence decreases to 1-2% by 3 months) orchidopexy is recommended if the testicular descent has not occurred by 1 year of age.

- **Y chromosome deletions** are increasingly recognised as a genetic cause of azoospermia and severe oligozoospermia. Published pick up rates of Y chromosome deletions range from 2-10% depending on referral criteria. Azoospermic men were found to have Y chromosome deletions involving the distal part of the long arm of the chromosome (Yq11 region). This area of the Y chromosome is referred to as the azoospermia factor (AZF) region. Deletions vary from 1-3Mb. Three AZF regions have been defined: a, b and c. These regions are required for normal spermatogenesis. Men with deletions of AZFa and AZFb are generally azoospermic and are unlikely to benefit from testicular sperm extraction (TESE). Men with deletions of AZFc, which are the majority, show a more variable phenotype and in 50% of cases, sperm will be obtained with TESE (increasing to 70% with micro-TESE). These deletions may also be detectable in men with disorders such as cryptorchidism, varicocele, and obstructive lesions of the vas deferens. The deletions, therefore, may cause abnormalities in testicular development as well as spermatogenesis
- **Defective androgen synthesis or response**, congenital androgen insensitivity and 5α-reductase deficiency. Reifenstein's syndrome is partial androgen insensitivity characterised by varying degrees of ambiguous external genitalia, hypogonadism, and subfertility. 5α-reductase deficiency presents as pseudo-hermaphroditism with increased virilisation at puberty, small phallus, severe hypospadias and cryptorchidism.

Acquired causes of primary hypogonadism

- **Varicoceles** are dilatations of the pampiniform plexus of spermatic veins in the scrotum. Left-sided varicoceles are ten times more common than right-sided ones, perhaps because of a longer left spermatic vein and the right-angle insertion into the left renal vein, which causes a higher hydrostatic pressure and dilatation in the left spermatic vein. Varicoceles are found in 11.7% of adult men and 25.4% of men with abnormal semen analysis. The reversibility of subfertility with surgery is controversial and the 2013 NICE guidlines) recommend that men should not be offered surgery to correct a varicocele to improve fertility, as it does not improve pregnancy rates. However, the European Association of Urology Guideline on Male Infertility) (2012) does suggest that DNA damage may improve after varicocele ligation.
- **Infection,** especially mumps, is a well-recognised cause of subfertility. While rare before puberty, clinical orchitis occurs in 15–25% of adult men. In mumps and other viral causes of orchitis (echovirus and arbovirus) sperm production problems are much more common than androgen deficiency. Other infectious causes of orchitis include sexually transmitted diseases, such as gonorrhoea and chlamydia, as well as tuberculosis and leprosy.
- Many **drugs** are associated with impaired spermatogenesis and/or Leydig cell dysfunction. The alkylating drugs (cyclophosphamide and chlorambucil), and anti-androgens (flutamide, cyproterone, spironolactone, ketoconazole, and cimetidine) cause testicular dysfunction by inhibiting testicular androgen production or action.
- **Environmental toxins** may be an underappreciated cause of subfertility.

The pesticide dibromochloropropane is a well-known cause, as are lead, cadmium and mercury. Chemicals with estrogenic or antiandrogenic activity, including insecticides and fungicides, may lower sperm counts, although direct proof of an effect in men is lacking. Radiation should also have adverse effect.

- In a meta-analysis of 20 observational studies, men who **smoked** cigarettes were more likely to have low sperm counts but the evidence that it reduces fertility is lacking.
- **High testicular temperature** may explain the subfertility associated with varicoceles and spinal cord injuries. Studies in rodents and non-human primates have shown that small increases in testicular temperature accelerate germ cell loss. However human data on this topic are lacking.
- Some subfertile men have **antisperm antibodies**, which presumably could impair spermatogenesis. These antibodies can occur spontaneously or after some testicular injury (vasectomy included).
- Oligospermic men have **increased DNA damage** in spermatozoa, which may reduce the chances of natural conception, as well as assisted reproductive techniques. It is reported to be associated with increased early pregnancy loss.
- **Systemic diseases** like: Chronic renal insufficiency, cirrhosis, or malnutrition may cause hypogonadism. Subfertility is common in men with sickle cell anaemia, presumably due to ischaemia inside the testes. Sperm motility and morphology abnormalities have been reported in men with celiac disease.
- **Post-testicular disease/sperm transport problems**
- **Epididymis** is site of sperm maturation. Its absence, obstruction or dysfunction leads to subfertility even though testicular sperm production is normal. Drugs (e.g. triptolide), chemical toxins (chlorohydrin) or intrauterine exposure to estrogens may cause epididymal dysfunction. It is presumed that some men with isolated asthenozoospermia have epididymal function defects.
- **Vas deferens** transports sperm from the epididymis to the urethra. One to 2% of subfertile men have congenital absence of the vas deferens. Most are carriers of cystic fibrosis mutations. Obstruction may also result from infection (e.g. gonorrhoea, chlamydia, tuberculosis). Vasectomy, although potentially reversible, generates an immune response and can cause men to remain subfertile despite adequate reanastomosis. Another genetic defect that may lead to abnormal transport of sperm is Young's syndrome (bronchiectasis, rhinosinusitis, reduced fertility) in which inspissated secretions within the vas deferens and epididymis interfere with transport of sperm.
- **Seminal vesicles and prostate** produce secretions. These secretions contribute to the spermatic fluid. It is unknown if abnormal function can cause subfertility.
- **Defective ejaculation:** Spinal cord or autonomic disease (e.g. diabetes) can interfere with normal ejaculation. Erectile dysfunction and premature ejaculation may be contributing factors.

Unexplained male factor subfertility

Oligo-Astheno-Teratozoospermia Syndrome (OATS) is the most common diagnosis and is classified as unexplained subfertility. Environment pollution, genetic aberrations and reactive oxygen species (ROS) are forerunners in the most possible causes of unexplained male subfertility.

- **Environment pollution:** Men exposed to pesticides have higher serum estradiol concentrations, and men exposed to solvents had lower LH concentrations with impaired androgen production. This leads to lower Testosterone: Estradiol (T: E) ratio and impaired spermatogenesis (Oliva et al 2001).
- **Genetic aberrations:** Understanding of spermatogenic genetic subfertility is gradually widening with the advances in molecular diagnostic technology. An exciting pilot study detecting genetic variants single - nucleotide polymorphisms (SNPs) in men with oligozoospermia and azoospermia identified 21 novel SNPs related to male subfertility. This is very promising and further larger studies are required to give insight in male spermatogenic subfertility (Ferlin et al 2007, Aston et al 2009).
- **Reactive Oxygen Species (ROS) and oxidative stress:** ROS (H_2O_2 – hydrogen peroxide, OH-hydroxyl radicals, O_2-oxide anions) are normal metabolites of sperm metabolism and leucocytes in the semen. At low levels, ROS enhance sperm capacitation, sperm hyperactivation and promote acrosomal reaction. At higher levels, ROS damage the sperm membrane reducing sperm motility, acrosomal reaction and zona pellucida binding of the spermatozoa. In addition, ROS directly damage the sperm DNA (DNA fragmentation) leading to failure of conception and miscarriages. Antioxidant therapy, i.e. vitamin E, vitamin C, folate, lycopene, zinc, selenium and garlic have shown to significantly increase pregnancy rates for couples where the men have higher levels of ROS and sperm damage. A Cochrane analysis showed a significantly higher birth rate in subfertile men taking oral antioxidants compared to men taking the control.

Empirical treatments for unexplained male factor subfertility

Empirical treatments like antiandrogens, androgens, gonadotropins, hCG (human chorionic gonadotropin), aromatase inhibitors, corticosteroids, bromocriptine, á-blockers and magnesium supplements have no scientific evidence and are not effective in the treatment of OATS. Recommendation for medical treatment of male subfertility is only for cases of hypogonadotrophic hypogonadism.

Evaluation of Male Subfertility:

History

A detailed history gives us important information about the following:

- **Developmental** Testicular descent Puberty, Change in shaving frequency, Loss of body hair.
- **Infections** Mumps, Sexually transmitted diseases, Prostatitis, Sinopulmonary symptoms, Surgical Hernia, hydrocele or varicocele repair, Vasectomy.
- **Drugs / environmental** Alcohol, Smoking, Anabolic steroids, Chemotherapy, Drugs that cause hyperprolactinaemia, Exposure to toxic chemicals (e.g. pesticides), Radiation Recreational drugs.
- **Sexual history** Libido, Frequency of intercourse, Previous fertility assessment, Chronic medical illness.

Investigations:

Semen analysis is a basic test needed to be done for all subfertile male. If report is abnormal then again repeated after 3 months. If gross spermatozoa deficiency identified then repeat semen analysis should be done as soon as possible.

Endocrine tests

If repeated semen analyses demonstrate severe oligozoospermia (<5 million

FSH	Testosterone	Diagnosis
Normal	Normal	Obstructive (Post-Testicular)
High	Low	Primary hypogonadism (Testicular)
Low	Low	Secondary hypogonadism (Pre testicular)
Low	High	Anabolic steroid abuse

spermatozoa/ml) or azoospermia, then basal serum FSH, LH, testosterone and estradiol levels should be measured. These can give an indication as to the cause of the male factor problem.

If serum concentrations of FSH, LH, and testosterone are normal and the man has absent or low volume semen at ejaculation and severe oligospermia or azoospermia, a postejaculatory urine sample can provide evidence about the possibility of retrograde ejaculation. Men with low sperm counts and low FSH and LH who are well-androgenised should be suspected of anabolic steroid abuse.

The serum testosterone can be normal, or high and rarely low depending upon the specific substance taken. Sperm production recovers in most men when they stop using anabolic steroids; however, this process can take a few months to a few years depending on the type and duration of anabolic steroid use.

Prolactin

To be measured in men who complain of reduced libido and have low serum testosterone. Low serum inhibin B may be a more sensitive indicator of primary testicular dysfunction than high FSH.

Treatment of Male subfertility:

Many treatments have initially been reported to improve male fertility only to be later proven ineffective. There are two main reasons for this; firstly, the use of semen quality as the outcome measure, and secondly, the non-inclusion of controls. Pregnancy should be the main outcome in male fertility studies because semen quality can show considerable background variability. A control group should be included, as it is possible for untreated men with severe deficiencies in their semen analysis to make their partners pregnant.

Specific treatments of proven benefit

Men with hypogonadotrophic hypogonadism should be offered gonadotrophin drugs because these are effective in improving fertility. Men with hypogonadotrophic hypogonadism desiring fertility should not be given testosterone replacement as that will further exacerbate the situation.

Hyperprolactinaemia, if present, should be reversed, either by stopping the offending medication or using a dopamine agonist such as Cabergoline or Bromocriptine. Spermatogenesis takes 3 months to achieve, so restoration of a normal sperm count does not occur until at least 6 months (or more) after serum prolactin and testosterone levels have normalised. If serum testosterone does not increase to normal within 6 months of prolactin normalisation, hCG or pulsatile GnRH is indicated.

If the serum Testosterone: Estradiol ratio (T: E) is <10, then anti-oestrogen therapy may be beneficial in improving the semen analysis.

Where appropriate expertise is available, men with obstructive azoospermia (azoospermia, normal size testes, normal FSH and testosterone) should be offered surgical correction of the epididymal blockage because

it is likely to restore patency of the duct and improve fertility. Surgical correction may be considered as an alternative to surgical sperm retrieval and IVF. Results are variable and depend on the site of re-anastomosis, the skill of the operator and the duration of obstruction. Results are better with vasectomy reversals, with pregnancy rates of approximately 50%. Vasectomy reversal has found to be more successful and cost effective than microsurgical sperm aspiration followed by Intra Cytoplasmic Sperm Injection (ICSI). In contrast, for obstructions due to other epididymal lesions, the results of surgical re-anastomosis are not as good as those with aspiration and ICSI. It should be noted however that vasectomy may be associated with the production of anti-sperm antibodies, meaning that subfertility may persist despite adequate re-anastomosis.

Retrograde ejaculation can be treated with IUI using the male partner's spermatozoa collected after alkalinisation of the urine and washing of the sperm. Alternatively, the spermatozoa can be used for IVF or ICSI.

Treatment of ejaculatory failure can restore fertility without the need for invasive methods of sperm retrieval or use of assisted reproductive procedures. However, further evaluation of different treatment options, specific to treat the cause, is needed.

Conclusion:

Male subfertility is many times neglected as not much attention is paid to it. Detailed history and examination can give us diagnostic information. Carefully selected investigation from battery of tests, allow us to decide timely management for subfertile male. Depending on investigation results, we can treat with simple IUI or ICSI by Percutaneous Epididymal Sperm Aspiration (PESA) and Microsurgical Epididymal Sperm Aspiration(MESA).

References:

1. Chia S-E, Lim S-TA, Tay S-K, Lim S-T. Factors associated with male infertility: a case–control study of 218 infertile and 240 fertile men.BJOG 2000; 107:55–61.
2. Jequier AM. Male fertility. BJOG 1993;100:612–4, commentary.
3. Nieschlag E, Behre HM, Nieschlag S, editors. Andrology: Male Reproductive Health and Dysfunction. 3rd edition. Springer; 2009.
4. Johnson MD. Genetic risks of intracytoplasmic sperm injection in the treatment of male infertility: recommendations for genetic counseling and screening. Fert Steril 1998;70:397–411).
5. Vincent MC, Daudin M, De MP, Massat G, Mieusset R, Pontonnier F et al. Cytogenetic investigations of infertile men with low sperm counts: a 25 year experience. J Androl 2002; 23:18–22.
6. Swerdlow AJ, Higgins CD, Pike MC. Risk of testicular cancer in cohort of boys with cryptorchidism. BMJ 1997;42:1507–11.
7. Oliva A, Spira A, Multigner L. Contribution of environmental factors to the risk of male infertility. Hum Reprod 2001;16:1768–76.).
8. Ferlin A, Raicu F, Gatta V, Zuccarello D, Palka G, Foresta C. Male infertlity: role of genetic background. Reprod Biomed Online 2007;14:734–45).
9. Aston KI, Carrell DT. Genome-wide study of single nucleotide polymorphisms associated with azoospermia and severe oligospermia. J Androl 2009;30:711–25.).
10. Tremelen K. Oxidative stress and male infertility – a clinical perspective. Hum Reprod Update 2008;14:243–58.
11. Showell MG, Brown J, Yazdani A, Stankiewicz MT, Hart AJ. Antioxidants for

male subfertility. Cochrane Database Syst Rev 2011;(1):CD007411.

12. National Institute for Health and Care Excellence. Fertility: Assessment and Treatment of People with Fertility Problems. CG156. RCOG; London; 2013.)

13. Vine MF, Margolin BH, Morrison HI, Hulka BS. Cigarette smoking and sperm density: a meta-analysis. Fertil Steril 1994;61:35–43.

Successful Management of Severe Grade Ovarian Hyperstimulation Syndrome: Case Report

Dr N. Mishra*, Dr G. Savani*, Dr D. Desai *, Dr P. Toal, Dr K. Dalal**, Dr H. Shewale****

**Dept. of Gynaecology and Obstetrics*

***Dept. of Anaesthesia*

BARC Hospital

Introduction:

Ovarian Hyper Stimulation Syndrome (OHSS) is a known complication in this present era of artificial reproductive techniques. The various modalities presently used for infertility treatment are ovulation induction with intrauterine insemination, in vitro fertilisation techniques like embryo transfer and intracytoplasmic sperm insemination. These all have led to increased incidence of OHSS which may be life threatening too.

Dr Nigamananda Mishra

Case Report:

A 34 years female patient married since 7 years, case of primary infertility underwent In Vitro Fertilisation (IVF) with embryo transfer. Patient was obese with BMI-33kg/m^2& a known case of PCOS (Polycystic ovarian syndrome). Previously, she had undergone myomectomy in view of fibroid uterus with no other significant co-morbidities. On day 7 of embryo transfer, patient presented with complaints of pain in abdomen, vomiting and dyspnoea at rest. Ultrasound showed enlarged bilateral ovaries with mild ascites and pleural fluid. Baseline tests done included haematocrit, coagulation profile, Human Chorionic Gonadotropin (HCG), liver and renal function tests (Table 1). Patient was started on conservative management-

Table 1:

Parameters	At Admission	On Day 5
Haematocrit	40	35
WBC	8000	21000
Coagulation profile	Normal	Normal
RFT	Normal	Normal
LFT	Raised liver enzymes with low albumin	Progressively increasing liver enzymes with low albumin
Chest X-ray	Bilateral Basal Pleural effusion (Rt>Lt)	Bilateral Basal Pleural effusion (Rt>Lt)
USG Abdomen and pelvis	Moderate Ascites Rt ovary-98cc, Lt ovary-92cc	Severe ascites Rt ovary-100cc, Lt ovary -110cc
Beta HCG	160IU/ml	767IU/ml
AG	102cm	112cm

hydration, progesterone supports for pregnancy, analgesics with cabergoline 0.5 mg per vaginally with subcutaneous low molecular weight heparin 40 mg for prophylaxis against Deep Vein Thrombosis (DVT). Patient's symptoms deteriorated with worsening of dyspnoea, intolerance to food, abdominal distension, pedal oedema with deranged liver function tests and normal coagulation profile. Repeat ultrasound done suggested bulky ovaries (volume-90cc, 100cc) with moderate ascites and bilateral pleural effusion (Table-1). Patient was diagnosed as a case of severe OHSS.

Patient was shifted to surgical intensive care unit for further management. Along with routine investigations serial blood gas monitoring was done. Her vitals were as follows: heart rate 110/min, blood pressure 130/90 mm Hg, respiratory rate 30/min, SpO_2 on room air was 96%. She was put on supplemental oxygen at 5 litres/ min following which she maintained a saturation of 99%. Her baseline ABG readings were pH 7.42, pCO2 29.1 mm Hg, pO2 135 mm Hg, HCO3 21 mmol/L. ECG was suggestive of sinus tachycardia with no other abnormalities. On clinical examination, she had decreased air entry bilaterally and had tenderness over abdomen with an abdominal girth of about 108.5 cm. Ultrasound of chest and abdomen were suggestive of bilateral pleural effusion and moderate grade ascites respectively. Sequential compression device and enoxaparin 40 mg subcutaneous injection per day were started for DVT prophylaxis. 2D echo was done to rule out cardiac involvement and it was also within normal limit. All blood investigations were normal except for raised liver enzymes (SGOT - 240IU/L, SGPT - 438 IU/L) and bilirubin levels (Direct - 2.3 mg%, Indirect - 2.7 mg%). Gradually her abdominal girth increased to 116 cm and she complained of respiratory discomfort following which ultrasound guided ascitic tapping was done. Around 1800 ml of fluid was drained after which she was comfortable. Adequate hydration was given to maintain her urine output. During the course she had fever episodes which were not responding to paracetamol and thus she was shifted from ceftriaxone to piperacillin with tazobactam following which her fever subsided. Decision of termination of pregnancy by medical method was taken in view of maternal morbidity. A dose of 100 mg of Inj methotrexate (calculated based on body surface area) was given to terminate the pregnancy. She was closely monitored for vitals and blood investigations. Gradually symptoms subsided, patient improved and HCG titre showed a decreasing trend from 509 IU/ml to489 IU/ml. Patient was discharged with follow up ultrasound and investigations. Patient underwent dilation and curettage one month later in view of thickened endometrium-13mm with plateau of HCG values. Post procedure patient resumed regular menstruation.

Discussion:

The pathophysiology of OHSS is not fully understood, but increased capillary permeability with the resulting loss of fluid into the third space is its main feature. In the susceptible patient, Human Chorionic Gonadotropin (HCG) administration for final follicular maturation and triggering of ovulation is the pivotal stimulus for OHSS, leading to overexpression of Vascular Endothelial Growth Factor (VEGF) in the ovaries, release of vasoactive - angiogenic substances, increased vascular permeability, loss of fluid to the third space, and full-blown OHSS.

OHSS is an iatrogenic and potentially life-threatening condition that affects young, healthy patients. In addition, there is an important economic burden associated with OHSS due to absence from work, bed rest, or hospitalization and intensive medical management of severe cases.

There are two clinical forms of OHSS, both HCG related: the early-onset form (occurring in the first eight days after HCG administration) and the late - onset form (occurring nine or more days after HCG administration, related to pregnancy - induced HCG production).

Risk factors:

Several factors independently increase the risk of developing severe OHSS. These include age < 30 years, polycystic ovaries or high basal antral follicle count on ultrasound, rapidly rising or high serum estradiol, previous history of OHSS, large number of small follicles (8 to 12 mm) seen on ultrasound during ovarian stimulation, use of hCG as opposed to progesterone for luteal phase support after IVF, large number of oocytes retrieved (>20), early pregnancy.[1]

Classification of OHSS:

Mild OHSS (20-33% prevalence)	◆Mild Abdominal Pain ◆Bloating ◆Ovarian size <8 cm
Moderate OHSS (3-6% prevalence)	◆Moderate Abdominal Pain ◆Nausea ±vomiting ◆Ascites (ultrasound) ◆Ovarian size 8-12 cm
Severe OHSS (0.1-2% prevalence)	◆Ascites (clinical) ◆Oliguria ◆HCT >45 ◆Ovarian size >12 cm
Critical OHSS (multisystem failure)	◆Tense ascites/large hydrothorax ◆HCT >55% ◆WBC >25,000 ◆Oligo/anuria ◆Thromboembolism ◆ARDS

RCOG guidelines for management:

Mild OHSS (20-33% prevalence)	◆Analgesics for pain (APAP or opiates - NSAIDs controversial) ◆Patient education – oral intake >1L/day; I&O recording; weight and abdominal circumference measurement; no strenuous activity (including intercourse). ◆Follow up in 24 hours
Moderate OHSS (3-6% prevalence)	◆Any hemodynamic/laboratory abnormalities or patients who are symptomatic will require hospitalization
Severe OHSS (0.1-2% prevalence)	◆Fluid resuscitation (NS or LR +D5W) to maintain UO no less than 20-30cc/hr ◆If hypoalbuminemic, 25-50g of albumin q2-12h.
Critical OHSS (multisystem failure)	◆Same as above with supportive treatment of complications (e.g. paracentesis)

Prevention:

It is essential to identify patients who are likely to develop OHSS. Ovarian stimulation protocol with individualised treatment should be followed. All patients undergoing ovarian stimulation for treatment of infertility should be counselled regarding possibility of development of OHSS and its signs and symptoms so that early detection will minimize the associated morbidity among these patients.

References:

1. Golan A., Weissman A. A modern classification of OHSS. Reprod Biomed Online. 2009 Jan 1;19(1):28–32.
2. Mathur R., Kailasam C., Jenkins J. Review of the evidence base strategies to prevent ovarian hyperstimulation syndrome. Hum Fertil 2007;10:75–85.
3. The Management of Ovarian Hyperstimulation Syndrome. 2016; Greentop Guideline No.5.

Localisation of Parathyroid Adenoma - A Challenging Case Report

Dr Jayesh Kalbhande
Dept. of Surgery
BARC Hospital

A 62 year old non-diabetic, non-hypertensive female patient, presented with extreme weakness, multiple joint pain more in small joints of both hands and difficulty in getting up from squatting position since one month.

Her blood investigations showed Hb13 gm/dl, WBC 10,400/cu.mm, S. Creatinine 0.8 mg/dl, liver function test was normal, Vitamin D 43 ng/ml (Normal), B 12 level 233pg/ml (Normal), RA factor negative, S. Calcium 12.7 mg/dl (Raised), S. Phosphorus 2.9 (Low), S. Para Thyroid Hormone (PTH) level 365 pg/ml (Raised)

In view of raised PTH levels with raised S. Calcium levels, hypersecreting parathyroid pathological lesion was considered as cause of symptomatic hyperparathyroidism. "SestaMiBi" scan was done which showed increased tracer uptake in a large hypodense nodule measuring 1.7 x 1.5 cm posterior to the upper part of left lobe of thyroid gland suggestive of parathyroid adenoma posterior to upper pole of left lobe of thyroid gland. Dexa Scan of total body and lumbar spine femur was done which showed T. Score of minus 1.7, suggestive of hyperparathyroidism causing ostopenia in entire skeleton. Based on these investigations, diagnosis of left superior parathyroid adenoma was reasonably tenable.

Endocrinology opinion was advised for further management. A focussed 2D computerised tomography from neck to pelvis showed nodular thyroid goitre and no evidence of arterial enhancing mass lesion. However, focussed USG of neck demonstrated adenoma in the right superior parathyroid gland.

Fluid aspirate from right thyroid gland was done which had PTH level of more than 1900 pg/ml while aspirate from left thyroid gland had PTH levels of 5.93 pg/ml. Skeletal muscle aspirate from sternocleidomastoid had PTH levels of < 3 pg/ ml thereby localising the parathyroid adenoma to the right superior parathyroid gland.

The patient underwent right parathyroidectomy under general anaesthesia. After removal of right superior parathyroid gland, intraoperative serum parathyroid hormone level showed normalisation thus confirming removal of pathological parathyroid gland. Intraoperative frozen section showed parathyroid carcinoma as diagnosis. Hence, she underwent radical central and right lateral compartment lymphadenectomy. Left parathyroid gland was neither explored nor removed. Serum calcium level normalised, in addition to significant symptomatic improvement after surgery.

Discussion

Hyperparathyroidism is caused by hypersecretion of parathyroid hormone from parathyroid gland. It is associated with symptoms of muscle weakness, abdominal pain, symptoms related to kidney stones and bone pain secondary to osteoporosis. Increased parathyroid hormone can be due to.

1) Primary hyperparathyroidism: Due to parathyroid adenoma, hyperplasia or carcinoma.
2) Secondary hyperparathyroidism: Secondary to another condition that lowers serum calcium e.g. chronic renal failure, severe vitamin D deficiency.
3) Tertiary hyperparathyroidism: In a few cases of secondary hyperparathyroidism due to chronic renal failure, parathyroid gland becomes autonomous.

This patient had symptomatic hyperparathyroidism with hypocalcaemia. Her initial investigation revealed a left superior parathyroid adenoma. However, further investigation confirmed that she actually had right superior parathyroid adenoma which later turned out to be carcinoma. Therefore surgery on left parathyroidgland based on initial investigation would not have helped the patient.

Conclusion

"Sesta-MiBi" scan for localisation of parathyroid adenoma may sometimes be inadequate. Ultrasound scan and intrathyroid parathyroid hormone levels may give additional information to help in localisation of parathyroid adenoma/ carcinoma.

Suggested reading

1) Rodgers SE, Hunter GJ, Hamberg LM, et al, Improved preoperative planning for directed parathyroidectomy with 4-dimensional computed tomography. Surgery 2006;140:932–40.
2) Krausz Y, Bettman L, Guralnik L, et al, Technetium-99m-MIBI SPECT/CT in primary hyperparathyroidism. World J Surg 2006; 30:76–83.
3) Rasmussen K, Larsen LP, Arveschoug A, et al. Predictive value of parathyroid Scintigraphy in the preoperative evaluation of patients with primary hyperparathyroidism.Scand J Surg. 2006; 95(3):199-204.

Trigeminal neuralgia- A challenge to treat!

Dr Jalpa Kate, Dr Kailash Kothari, Dr Sheetal Chiplonkar
Dept of Anaethesia,
BARC Hospital

Trigeminal neuralgia(TN) has been known since many years. It was first described by Arateus in the first century AD.

Dr Jalpa Kate

Clinical features:

The pain experienced is severe, almost always unilateral, and neuropathic in nature. It is located within the distribution of the trigeminal nerve (Figure 1) and may involve one or more divisions of trigeminal nerve which are ophthalmic(V1), maxillary (V2) and mandibular (V3). There are episodes of stabbing pain followed by a refractory period, a period of relief that lasts seconds, minutes, or even hours. The pain is often characterized as an "electric shock" and is typically accompanied by a unilateral grimace due to muscle twitching hence the designation 'tic douloureux'. The attack may occur spontaneously and/or can be triggered by stimulating a specific area of the face known as a trigger zone. Trigger zones can exist anywhere in the trigeminal distribution, including intraoral mucosa. Due to this reason, patients characteristically avoid touching the face, washing, shaving, biting or chewing, or any other maneuver that stimulates the trigger zones causing pain. These episodes may become more intense and frequent with time. They last for weeks or months and return after a period of remission.

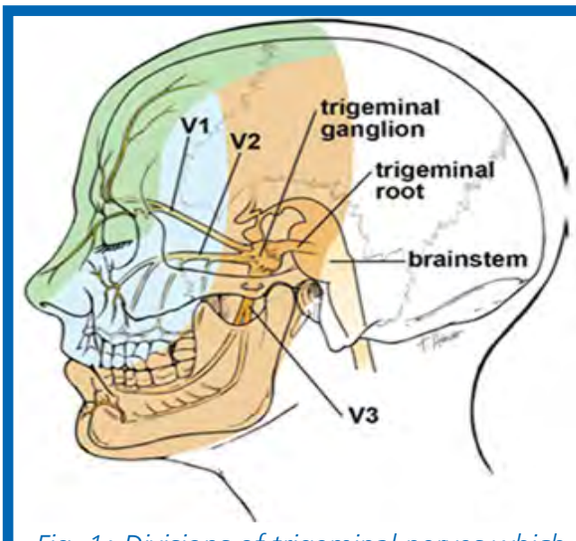


Fig. 1: Divisions of trigeminal nerves which can get affected in TN and trigger zones

Etiology:

Currently there are three main etiological theories behind TN. Disease related, injury related and vascular related etiology. Dysfunction of the trigeminal sensory system is the pathology suspected in most of the cases. Disturbed integrity of myelin sheath as a cause for trigeminal neuralgia is under investigation.

Vascular compression theory of TN, attributes presence of a groove or distortion of the trigeminal nerve root by vessels, or rarely tumors. Disease related theory for TN suggested that the paroxysmal neuralgic pain of TN with associated trigger zones is consistent with

minor mechanical or pulsatile compression superimposed upon axonal degenerative changes due to hypertension, diabetes atherosclerosis, or Multiple Sclerosis.

Classification:

Trigeminal neuralgia is classified as "classical" and "symptomatic." The term classical refers to trigeminal neuralgia of unknown etiology. Classical rather than primary has been applied to those patients with a typical history. The term symptomatic or secondary can be used for those patients in whom a cause of trigeminal neuralgia has been demonstrated like a neuroma, tumor, multiple sclerosis (MS) etc.

Diagnosis:

Accurate diagnosis of TN is essentially clinical as objective laboratory tests are usually normal in these patients. The International Headache Society recently established new clinical diagnostic criteria (Table 1) for TN as part of the International Classification of Headache Disorders, Second Edition (ICHD-II).

Treatment:

The primary objective of treatment in TN is complete relief of pain preferably with preservation of sensation on the face and avoiding major complications associated with surgical decompression.

Medical treatment is usually the first line of treatment. Carbamazepine, a tricyclic antidepressant is the drug of choice for TN. Other medications like colanzepam which depresses excitatory transmissions in the nucleus caudalis can also be used. Those patients who do not respond to monotherapy may benefit from combination therapy with gabapentin, lamotrigine, topiramate, or baclofen. But, up to 30% of patients become refractory to medical therapy after 2 years.

Table 1: ICHD-II Diagnostic Criteria for Classical TN

A	Paroxysmal attacks of pain lasting from a fraction of a second to 2 minutes, affecting one or more divisions of the trigeminal nerve and fulfilling criteria B and C
B	Pain has at least one of the following characteristics:
	1. Intense, sharp, superficial, or stabbing. 2. Precipitated from trigger areas or by trigger factors
C	Attacks are stereo typed in the individual patient
D	There is no clinically evident neurologic deficit
E	Not attributed to another disorder

Surgical treatment, microvascular decompression (MVD) is a neurosurgical procedures with a low recurrence rate and minimal sensory disturbance. But it is a major surgery done under general anaesthesia and is associated with a risk of significant morbidity. This more aggressive surgical option may be best reserved for younger patients who can tolerate the procedures and benefit most from the better long-term relief of pain or for any patient who is a good surgical candidate and who strongly wishes to avoid the risk of sensory loss. Surgical treatment is also required for patients with structural lesions.

Other percutaneous techniques including retrogasserian glycerol rhizolysis and balloon compression have also been used with good results. Though these procedures are relatively simple and safe but have higher recurrence rates and result in extensive sensory loss.

Gamma knife radiosurgery is a minimally invasive technique to treat trigeminal neuralgia. It is claimed to be having low risk of facial

paresthesias, with approximate 80% rate of significant pain relief, and a low recurrence rate. However longer-term evaluations are warranted for this new modality.

If medical therapy fails and surgery is not possible due to coexisting medical conditions, old age or recurrence, treatment with percutaneousretrogasserian-thermocoagulation is an option. Thermocoagulation with radiofrequency current is fairly safe and simple requiring only minimal sedation and hospitalization.

The aim of radiofrequency is to produce trigeminal sensory loss, but only to pin prick not to light touch. Controlled increment of radiofrequency currents allows preferential destruction of fibers that conduct pain (finely myelinated A delta fibers and unmyelinated C fibers) with some reservation of the heavily myelinated A beta fibers which conduct touch sensation. Recent literatureshows that thermocoagulation of the gasserian ganglion is actually performed with a technical success of 98–100%. Immediate pain relief is reported as high as 90–95% in multiplestudies.Radio frequency ablation/ thermocoagulation techniques have been found to provide complete pain relief without medication in a median of88% in patients till 6-month and in 61% patients it may last for the 3-years. The common side effects are hypoaesthesia (50%), dysaesthesia(6%), anaesthesia dolorosa (4%) corneal anaesthesia with a risk of keratitis (4%).

A 71 year old female patient was suffering from trigeminal neuralgia since 1995. She received carbamazepine, which only worked partially in her case MRI in 2003 revealed incidental pituitary adenomaand hence she underwent transnasal removal of nonfunctioning pituitary adenomain 2008 followed by microvascular decompression of trigemional nerve 8 days later. However following surgery she did not get complete relief from neuralgia and continued medical management. Over next 10 years the pain worsened and inspite of very high doses of carbamazepine (1200 mg) and gabapentine (400mg), she was in excruciating pain day and night. She underwent an MRI again in 2018 which showed stable pituitary adenoma slightly abutting optic chiasma superiorly and small vessels in close proximity of bilateral trigeminal nerves at the root entry, so she underwent retromastoid suboccipiotal craniectomy with microvascular decompression of trigeminal nerve under general anaesthesia. However her symptoms recurred within 48 hours. Her pain was very severe with visual analogue scale of 10/10 with rightV2 distribution. She was referred to Anaesthesia Pain clinic in BARC for treatment.

Percutaneous radiofrequency ablation of the trigeminal nerve was successfully done in operation theatre under fluoroscopic guidance. A 22 G, 10 cm RF cannula with 5 mm active tip was used. After administration of local anaesthesia with minimal intravenous Propofol sedation the cannula was advanced towards the foramen ovale. The depth and position in the Meckel's cave was confirmed on lateral fluoroscopy. (Fig. 2 and 3). The mandibular nerve was stimulated at 2 Hz between 0.5 and 1.5 mA and muscle contraction at lower jaw was confirmed. This confirmed that the needle tip was lying close to the mandibular nerve in the lateral portion of foramen ovale. After confirming the paraesthesia in the V2 distribution on sensory stimulation, RF ablation was carried out at 70° for 60 seconds. The procedure was repeated once more to get better lesioning so that higher current would be required to get the baseline motor response.

Post procedure her pain score was 2-3/10 and 0-1 by 1st week. Medications were slowly tapered off over next few days. With complete pain relief and intact corneal reflexes, her

pain relief and intact corneal reflexes, her quality of life improved drastically. In her follow up visit she had hypoaesthesia of right cheek but no complaints about it! Precise ablation of the involved division and reproducibility of the results makes minimally invasive radiofrequency ablation a promising low risk procedure in patients with trigeminal neuralgia.

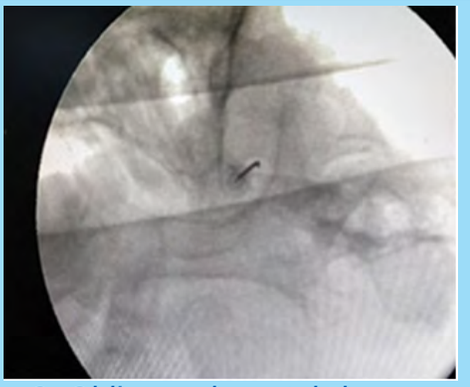


Fig. 2: Oblique submental Fluoroscopic image showing needle directed towards foramen ovale

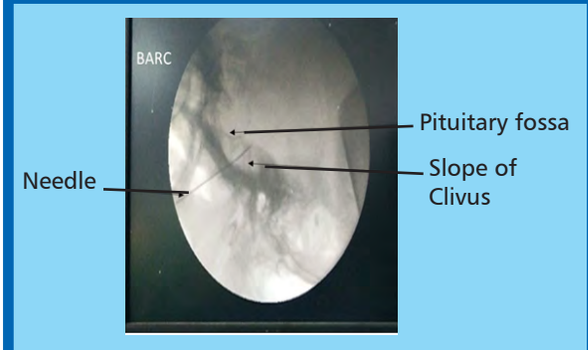


Fig. 3: Lateral Fluoroscopic image showing needle at the entrance of foramen ovale

Suggested reading:

1. Stiles Mand J Evans J. Pain Management. 1st Ed. Philadelphia: Saunders; 2007. Chapter 46, Trigeminal Neuralgia: p. 502-510.
2. Hickey A, Scrivani S, and Bajwa Z. Cranial neuralgias in: Fishman S, Ballantyne J, Rathmell J editors. Bonica's Management of Pain. 4th Ed. Baltimore. Lippincott Williams & Wilkins. 2010. Chap 66. p. 953-971.
3. Yizhong H, Jiaxiang NI, Baishan WU, et al. Percutaneous radiofrequency thermocoagulation for the treatment of different type of trigeminal neuralgia: evaluation of quality of life and outcome. J HuazhongUnivSciTechnol Med Sci 2010;30(3): 403–7.
4. Fouad W. Management of trigeminal neuralgia by radiofrequency thermocoagulation. Alexandria Journal of Medicine 2011; 47: 79–86.
5. Lopez BC, JamlynpJ, Zakrzewska JM. Systematic Review of ablative neurosurgical techniques for the treatment of trigeminal neuralgia. Neurosurgery 2004;54:973-983.
6. Broggi G, Ferroli P, Franzini A, Servello D, Dones I. Microvascular decompression for trigeminal neuralgia: comments on a series of 250 cases, including 10 patients with multiple sclerosis. J NeurolNeurosurg Psychiatry 2000;68:59–64.

Adrenal tumors: Current concepts in Anaesthetic Management

Dr Poorva Magarkar, Dr Sandeep S., Dr Sheetal Chiplonkar
Dept. of Anaesthesia
BARC Hospital

Introduction:

Adrenal gland surgery needs a multidisciplinary team approach. The indications for surgery are hormone secreting and non-hormone secreting tumors. The surgical techniques vary from open laparotomy, laparoscopic and now, to robotic approaches. Hormone secreting adrenal tumors present unique challenges to the Anaesthesiologists. This article attempts to address the peri-operative issues in the management of these challenging tumors with an emphasis on anaesthesia technique, hemodynamic monitoring and postoperative concerns.

Pheochromocytoma:

These tumors originate from chromaffin cells of sympathoadrenal system. The characteristic feature is the production of catecholamines predominantly noradrenaline, adrenaline and rarely dopamine. The tumors of extra-adrenal sympathetic and parasympathetic ganglia are classified as Paragangliomas. The condition is potentially life threatening unless diagnosed and treated. The site of origin helps in surgical dissection and prognostication of malignant potential which is different according to the site of origin.

CLINICAL FEATURES

Clinical features depend upon the relative proportions of adrenaline or noradrenaline. Classic triad includes

Cardiovascular system

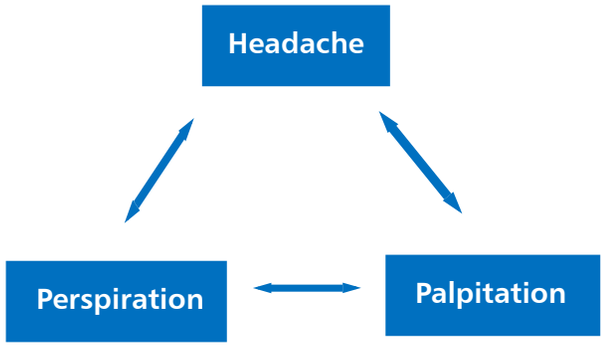
- Hypertension
- Tachyarrhythmias, bradycardia
- Heart failure

Dr Poorva Magarkar

- Myocardial infarction
- Cardiomyopathy
- Pulmonary oedema
- Vasoconstrictionand marked decrease in circulatory blood volume causes raised haematocrit and apparent polycythemia.

Central nervous system

- Tremors, anxiety, feeling of impending doom, nervousness and psychosis,
- Cerebrovascular accidents as intracranial haemorrhage or an embolic episode,
- Hypertensive encephalopathy,



Metabolic disturbances

Catecholamine induced glycogenolysisim pairs glucose control, also there is an impaired release of insulin. Excessive catecholamines cause hyper-metabolism resulting in weight loss. Excess of dopamine causes nausea and vomiting.

DIAGNOSIS

Biochemical tests

- Free catecholamine level in a 24 hr urine sample: best confirmatory test,
- Plasma or urine catecholamine metabolites such as metanephrines,
- Plasma free metanephrines,
- Urinary vanillylmandellic acid (VMA) levels-nonspecific,

Localisation

- MRI and CT – gives accurate diagnosis.
- MIBG scan - Helps to localize recurrent tumors, metastases and tumors in unusual sites.
- PET scan.

Preoperative Management

Multidisciplinary involvement is required from the starting to optimize the outcome. Due to the meticulous preoperative management morbidity(23.6%) and mortality(2.4%) associated with these tumors is drastically reduced. The mainstay of optimization includes control of hypertension and vascular expansion.

Criteria for optimal control include:

- Blood pressure readings consistently < 160/90 mmHg
- Presence of orthostatic hypotension (not < 80/45)
- ECG should be free of ST-T changes
- No > 1 premature ventricular contraction every 5 minutes
- Nasal congestion

Control of hypertension

Nonselective, irreversible, α-receptor antagonists

Traditionally phenoxybenzamine has been used. It is a irreversible, nonselective and non-competitive α-receptor antagonist. It is initiated 14 days before the surgery at doses of 10 mg every 6–12 h, increased to 30–40 mg every 6 hourly to a maximum dose of 240 mg/day. Disadvantages are tachycardia and persistent α blockade which can cause resistant hypotension after tumor removal, soit may be advisable to even stop phenoxybenzamine administration 48 hrs prior to surgery.

Selective α-1 receptor antagonists

Drugs like prazosin, doxazosin, and terazosin cause vasodilatation without causing much tachycardia. They are not as efficient as phenoxybenzamine in preventing rise in blood pressure during tumor manipulation. The side effects are malaise, vertigo, mild headache, dizziness, gastrointestinal symptoms such as nausea, vomiting, diarrhea, postural hypotension.

Prazosin is the most commonly used drugwhich is initiated with 0.5–1 mg/dose every 4–6 h, titrated to maximum 20–24 mg/day. Because of shorter half-life, it is relatively ineffective in the intraoperative control of blood pressure especially if the last dose is given on the night before surgery.

Doxazosin is a longer acting drug and is usually given as once daily or twice daily dose. It is initiated at the dose of 1–2 mg/day and titrated to a maximum dose of 16 mg/day.

β-receptor antagonists

Tachycardia is secondary to α 2 receptor blockade from phenoxybenzamine or due to secretion

of adrenaline or dopamine. Suppression of β-1 mediated cardiac sympathetic drive before adequate arteriolar dilatation can cause acute cardiac insufficiency and pulmonary edema so β-blockers are started after initiation of α-blockade. Metoprolol, Atenolol, Carvedilol, Bisoprolol are commonly used.

Calcium channel blockers

They are especially used in normotensive patientsand in paroxysmal hypertension. Amlodipine (5–20 mg/day), diltiazem (90–240 mg/day), nifedipine (30–90 mg/day), nicardipine (60–90 mg/day), verapamil (180–540 mg/day) are commonly used.

Vascular expansion

The Endocrine Society Clinical Practice Guidelines also recommend a high-sodium diet and fluid intake to reverse catecholamine - induced blood volume contraction and to prevent severe hypotension after tumour removal. Serial haematocrit values guide to the effectiveness of volume expansion.

Other drugs

α-methylparatyrosine (Metyrosine) - inhibits tyrosine hydroxylase enzyme and decrease catecholamine production by 50 – 80% but its use is limited in malignant and inoperable tumors due to side effects like diarrhea, crystalluria, anxiety, depression, extra pyramidal symptoms and galactorrhoea.

ACE inhibitors can be used for preoperative control of blood pressure.

Oral hypoglycemic drugs and / or insulin for hyperglycemia may be required.

Preoperative Investigations

- CBC, RFT, LFT, serum electrolytes,
- Serial haematocrit,
- Coagulation profile,
- Blood sugars,
- Chest x-ray,
- ECG - presence and/or extent of left ventricular strain, hypertrophy, bundle branch blocks, and ischemia,
- 2D echocardiography-assess global systolic and diastolic function, valve function, dilated cardiomyopathy, heart failure.

Intraoperative Management

The anaesthesia goals are to:

- Relief anxiety before anesthetic induction with benzodiazepines night prior to surgery and before entering in operation theatre
- Avoid drugs or maneuvers which produce a catecholamine surge especially at laryngoscopy, surgical stimulation, peritoneal insufflation, ligation of venous drainage and tumor handling.
- Deliver anesthetic agents to provide stable hemodynamics during catecholamine surges, followed by the opposite scenario after removal of tumor.
- Maintain normovolaemia and hemodynamics after tumor resection.

Monitoring

Standard ASA monitoring which includes 5 lead ECG, Pulse oximeter, EtCO2, Temperature probe, NIBP, Invasive lines – Arterial line before induction of anaesthesia and CVP monitoring, Cardiac output monitoring in patients with cardiomyopathy (Pulmonary artery catheter, TOE), Large bore peripheral venous access, Urinary catheter, Blood glucose monitoring, Forced air warming devices to prevent hypothermia, Depth of anaesthesia monitoring like BIS, entropy, Intermittent pneumatic Sequential compression device.

All patients are given general anaesthesia with endotracheal intubation with Epidural analgesia.

Agents commonly used include propofol and etomidate. Airway instrumentation should be attempted only after adequate anaesthetic depth and pressor response to laryngoscopy can be abolished with fentanyl in small doses, IV lidocaine 1-1.5mg/kg, esmolol 0.5 mg/kg bolusand infusion, dexmeditomedine, nitroglycerinor sodium nitroprusside.

Drugs considered safe include: Propofol, Etomidate, Fentanyl, Alfentanil, Remifentanil, Benzodiazepines, Vecuronium, Rocuronium, Isoflurane, Sevoflurane, Nitrous oxide.

Drugs avoided include: sympathetic stimulants like histamine releasing drugs (Atracurium, Morphine). Suxamethonium can produce a catecholamine surge by virtue of muscle fasciculatioN and drugs like pancuronium, metoclopramide, ephedrine, chlorpromazine, ketamine, halothane, desflurane.

Pharmacological options for intraoperative hemodynamic control

Communication with the surgeon should be maintained during tumor manipulation which can result in blood levels of catecholamines up to 200,000 to 1,000,000pg/ml. This should be anticipated and treated with drugs like Phentolamine, nitroglycerin, sodium nitroprusside, magnesium sulphate, volatile anaesthetics, â blockers, anti-arrhythmic drugs. Hyperglycemia can occur as a result of catecholamine excess and insulin infusion therapy may be required.

After venous drainage ligation refractory hypotension can occur due to sudden drop in catecholamines, residual á blockade, down regulation of adrenoceptors, suppression of the normal contralateral adrenal gland, myocardial dysfunction and hypovolaemia from blood and fluid loss. This can be pre-empted by giving 2-3 L of crystalloid and /or colloid bolus prior to tumor ligation and all vasodilators have to be discontinued. Persistently hypotensive patients may require adrenaline, noradrenaline, phenylephrine support. Vasopressin does not rely on peripheral adrenergic receptors for its effect, it is particularly effective for refractory hypotension. Once all agents have been used with little or no effect and fluid therapy has been fully optimized, the use of IV methylene blue may be considered. Steroid replacement following bilateral adrenalectomies should be considered. After ensuring haemodynamic stability patients are extubated at the end of the procedure.

Post Operative Management

Ideally, these patients are managed post operatively in surgical ICU for intensive monitoring. Along with fluid management, vasopressor infusion is usually required for a short duration only. Sudden catecholamine withdrawal after tumorremoval leads to rebound hyperinsulinemiawhich can cause severe hypoglycemiaso hourly blood sugar monitoring is required in first 24-48 hr period. Steroid supplementation is required if bilateral adrenalectomy is carried out. Sustained hypertension after surgery could be due to renal ischaemia, residual tumor, or underlying essential hypertension.

Epidural analgesia along with regular oral medications provide multimodal analgesia.

Cushing Syndrome

It is caused by increased cortisol production or by exogenous glucocorticoid therapy.

Clinical Features

Includes moon facies, buffalo hump, central obesity, skin thinning, bruising, abdominal striae, proximal muscle weakness, fatigue, spontaneous bone fractures, amenorrhea, menstrual irregularities, decreased libido, infertility, water retention, leading to hypertension, hyperglycemia, metabolic alkalosis and hypokalemia.

Diagnosis

Laboratory tests include blood and urinary cortisol level, urinary 17-hydroxycorticosteroids and plasma ACTH level, dexamethasone suppresion test. Imaging modalities include ultrasonography, angioCT, and MRI. Ectopic glucocorticoid secretion can be detected by technetium 99 labeledoctreotidescintigraphy.

Anaesthetic Management

Preoperative optimization includes control of hypercortisolism, hyperglycemia, hypokalemia, hypertensionand prevention of hypercoaguablestate. Utmost care is required during positioning to prevent bone fractures and skin damages. Deep sedation is avoided due to risk of hypoxia and difficult airways. Wide bore IV cannulation,central vein accesses are required to facilitate the fluids and drugs administration. Epidural anaesthesia for pain relief is useful. Postoperatively pain management, early mobilization, control of hypertension, hyperglycemia and cortisol level monitoring is required.

Conn's Syndrome

This syndrome is caused by excessive secretion of aldosterone. Majority of cases (60%) have unilateral adenoma.

Clinical *Features*

Patients usually present with fatigue, muscle cramps, skeletal muscle weakness, systemic hypertension,arrhythmias, myocardial ischemia, metabolic alkalosis, increased urinary excretion of potassium, hypokalemia, hypernatremia.

Diagnosis

Laboratory tests will show decreased blood renin and increased aldosterone levels, hypokalemia, hypernatremia. Imaging modalities include ultrasonography, CT Angiography, MRI.

Anaesthetic Management

Intraoperative hemodynamics changes and hypokalemia are the main concerns. Manipulation of adrenal gland during resection may lead to catecholamine releasewhich can cause hemodynamic fluctuations. It can produce brisk and untreatable intraoperative hypertension. The anesthesiologistshould also deal with hypervolemia, and depending on the case cortisol supplementation may be needed.

Factors which can cause intraoperative major blood loss include size of tumor, preoperative preparation time, proximity to vessel and other organs.

This year, we came across a 29yr old male patient reported to surgery OPD with complaints of facial puffiness swelling of whole body and history of recent onset hypertension. The patient was investigated and diagnosed with a left suprarenal mass with normal levels of metanephrine, normetanephrine and VMA but was metabolically active on FDG PET scan and raised a suspicion of pheochromocytoma. The patient was started on tab. Telmisartan for hypertension and was advised to undergo open adrenalectomy. After attaching all ASA standard monitors, patient wasadministered general anaesthesia withendotracheal intubation and thoracic epidural after securing a central venous access and arterial line for invasive hemodynamic monitoring. Ionotropes and vasodilators were kept ready to tackle BP fluctuations. But on contrary to what was expected, there was minimal hemodynamic fluctuations with tumor handling. Free thrombus was seen in left adrenal vein and another in left renal vein. Hence the patient underwent left adrenalectomy along with left nephrectomy with blood loss of 4 liters which required ionotropic support in addition to blood and blood products. Patient was extubated and observed in surgical intensive care unit for 2days and later shifted

to ward. Pathology report revealed that the tumor was Adrenocortical carcinoma and patient was sent for chemotherapy.

Conclusion

Preoperative optimization has played an important role in decreasing the incidence of perioperative morbidity and mortality associated earlier with these surgical procedures. Patients with these tumors are best managed by an experienced team with good communication between the surgeon, endocrinologist and anaesthetist. Careful perioperative hemodynamic monitoring and control should be vigilantly continued in the postoperative period.

Suggested Readings

- Ramakrishna H. Pheochromocytoma resection: Current concepts in anesthetic management. J Anaesthesiology clinical pharmacology 2015;31:317-23.
- Rubin Domi, Hector Sula, Myzafer Kaci. Anaesthetic consideration on adrenal gland surgery. J clin Med Res. 2015;7(1):1-7.
- Rashmi Ramachandran, Vimi Rewari. Current perioperative management of Pheochromocytoma. Indian journal of urology 2017;33: 19-25.
- Anju Gupta, Rakesh Garg, Nishkarsh Gupta. Update in perioperative anesthetic management of pheochromocytoma. World J Anesthesiol. Nov 27, 2015; 4(3): 83-90.
- Hongju Lou, Bin Li, Xuerong Yu. Preoperative risk factors for massive blood loss in adrenalectomy for Pheochromocytoma. Oncotarget 2017; 8(45):79964-79970.

Polycythemia Vera – A Rare Blood Disorder

Introduction:

Polycythemia Vera is one of the chronic myeloproliferative disorders (neoplasms). It is a slow growing bone marrow disease that leads to an abnormal increase in the number of red blood cells. It occurs more often in elderly men than in women. The diagnosis is often suspected on the basis of laboratory tests. Common findings include an elevated hemoglobin level (>18.5gm/dl in male and >16.5gm /dl in female) and hematocrit (>60% and >55% respectively) reflecting the increased number of red blood cells, the platelet and white blood cell count may also be increased. The problem is often linked to JAK2V617F gene defect, mutation of which is observed in up to 95% of patients with Polycythemia Vera. Following is a case of patient with Polycythemia Vera who had various clinical manifestations on different occasions as the disease process was progressive.

Dr Debjani Pal,
Mandala Dispensary

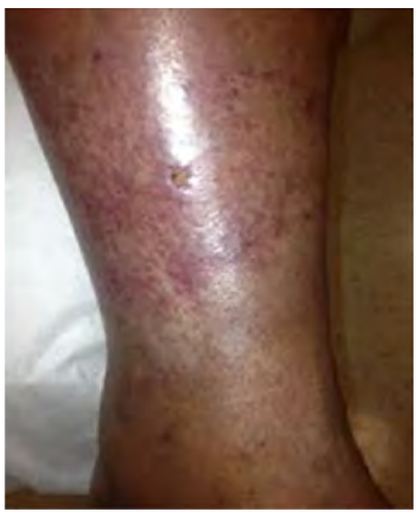
Clinical Presentation:

A 58year old male presented with history of congestion of eyes, giddiness and fatigability. After initial investigation he was found to be a case of Polycythemia Vera with positive JAK2 mutation. Subsequently he underwent phlebotomy on regular basis to maintain PCV < 45. Five years after initial diagnosis of the disease, he reported with history of significant weight loss and dysphagia with development of Hiatus hernia. Endoscopic evaluation revealed duodenitis.

2 years later, patient also had an episode of chest pain and was diagnosed as Ischemic Heart Disease. He was started on B blockers, ACE inhibitors. As he was at risk of developing thrombotic events antiplatelet therapy was also initiated. In view of increased requirement of repeated phlebotomies to maintain hematocrit value, he was advised Tablet Hydroxyurea with a dose of 500 mg once daily.

Two years following this, he presented with features of superficial thrombophlebitis and cellulitis. This was treated with injectable broad spectrum antibiotics and anti inflammatory agents.

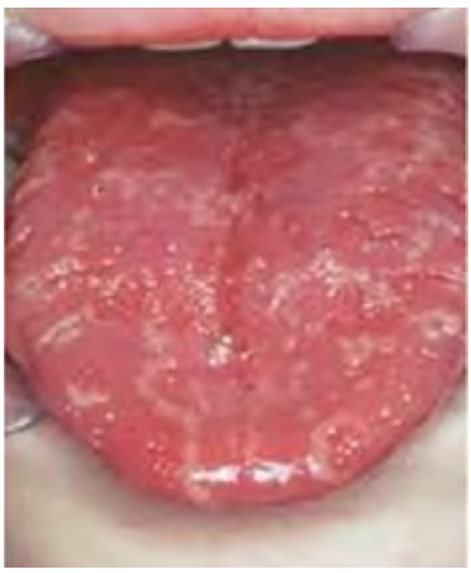
After 8 months, he presented with soreness of mouth, glossitis and after few weeks developed oral thrush. He also had leukocytosis and thrombocytosis. His general condition deteriorated.



Superficial Throbophlebitis and Cellulitis of right lower limb

Tablet Hydroxyurea was stopped and he was started on Mercaptopurine (6MP).

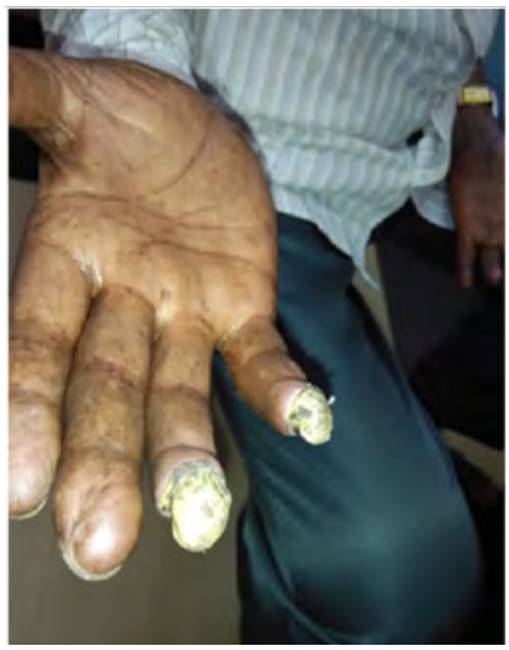
But after 4 months of treatment with 6MP, he got admitted in view of pancytopenia and received Packed Cell Volume transfusion and 6MP was withdrawn. As a consequence of the disease process, this time he developed facial puffiness, bilateral pedal edema and dyspnoea on exertion. 2D echo revealed cardiac ejection fraction of 50% with mild diastolic dysfunction. CT Chest was performed which showed evidence of cylindrical bronchiectasis. He was



Soreness of Mouth, Glossitis

detected to have pulmonary hypertension. He also developed blackish discoloration of right hand 3rd, 4th and 5th fingers due to dry gangrene of distal phalanges secondary to Deep Vein Thrombosis.

Treatment was initiated with Heparin drip for 3days followed by oral anticoagulant, Warfarin with close monitoring of PT and INR. After 1 month repeat Venous Colour Doppler showed resolution of DVT.



Dry gangrene of right phalanges

Discussion:

Polycythemia occurs in all age groups although the incidence increases with age. People with Polycythemia Vera can be asymptomatic. Sometimes, symptoms are related to blood hyper viscosity, sluggish blood flow or thrombosis that leads to poor oxygen delivery and symptoms include

- Headache, dizziness, vertigo, tinnitus, visual disturbances, angina pectoris or intermittent claudication.
- Bleeding complication 1%.
- Thrombotic complications 1% include venous thrombosis and thromboembolism and an increased prevalence of stroke and other arterial thromboses.
- Abdominal pain due to Peptic Ulcer Disease(increased Histamine levels & gastric acidity)
- Splenomegaly 75%
- Pruritus 40% results from increased histamine levels.

Untreated, Polycythemia Vera can be fatal. As the condition cannot be cured, treatment focuses on treating symptoms and reducing thrombotic complications by reducing the erythrocyte levels.

Major goal of treatment is to prevent thrombotic events

- Phlebotomy / bloodletting: mainstay of treatment – is aimed at reducing the hyper viscosity by decreasing hematocrit level to < 45%.
- Hydroxyurea: is an effective agent for myelosuppression.
- Recombinant interferon alfa2b reduces myeloproliferation, splenomegaly and alleviate pruritus symptoms.
- Splenectomy is indicated in patients with painful splenomegaly or repeated episodes of thrombosis causing splenic infarction.

References:

1. Mayo Clinic staff ''Polycythemia Vera – MayoClinic.com". Polycythemia Vera: Definition. MayoClinic Retrieved 2011-09-03.
2. What is "Polycythemia Vera?" National Heart, Lung and Blood Institute, Retrieved 2011-09-03.
3. Polycythemia Vera Follow – up. Retrieved 2011-09-03.
4. Björkholm, M; Derolf, AR; Hultcrantz, M; et al. (10 June 2011). "Treatment-related risk factors for transformation to acute myeloid leukemia and myelodysplastic syndromes in myeloproliferative neoplasms". Journal of Clinical Oncology. 29 (17): 2410–5. doi:10.1200/JCO.2011.34.7542.

Role of Computed Tomography in Endocrine Dysfunction

Dr Shubhra Gupta,
Dept of Radiology
BARC Hospital

Endocrine glands are major glands which have multiple functions. Increase or decrease in the secretion of endocrine glands lead to disorders in the endocrine system. Major glands such as pituitary, thyroid, pancreas and adrenals have been studied and their imaging plays an integral role in the evaluation of endocrinopathies involving these glands. Magnetic resonance imaging is currently the study of choice in evaluating the pituitary gland. As for the adrenals, computed tomography and magnetic resonance are probably equally sensitive in detecting adenomas and carcinomas. The endocrinologic and radiologic evaluation of the thyroid gland involves the use of mainly nuclear medicine scanning and ultrasonography. Imaging plays a vital role in evaluation of Endocrine Hypertension (EH) that can occur due to hormone secreting tumour in adrenal, thyroid or pituitary gland.

Computed tomography (CT) is the imaging modality of choice for evaluating adrenal gland morphology and masses associated with it. High-resolution CT of upper abdomen, using 1–3 mm thick slices to reduce volume averaging, is the most accurate technique for identifying adrenal lesions. Contrast-enhanced CT and delayed images help in further characterization of the lesions. Adenomas are most common lesions of adrenal. They can be asymptomatic or symptomatic. Adenomas appear as small (<4 cm in size), homogenous, well- circumscribed masses with smooth margins (Figure 1). Calcification, necrosis, and haemorrhage are uncommon in benign adenomas. A large amount of intracytoplasmic lipid within the

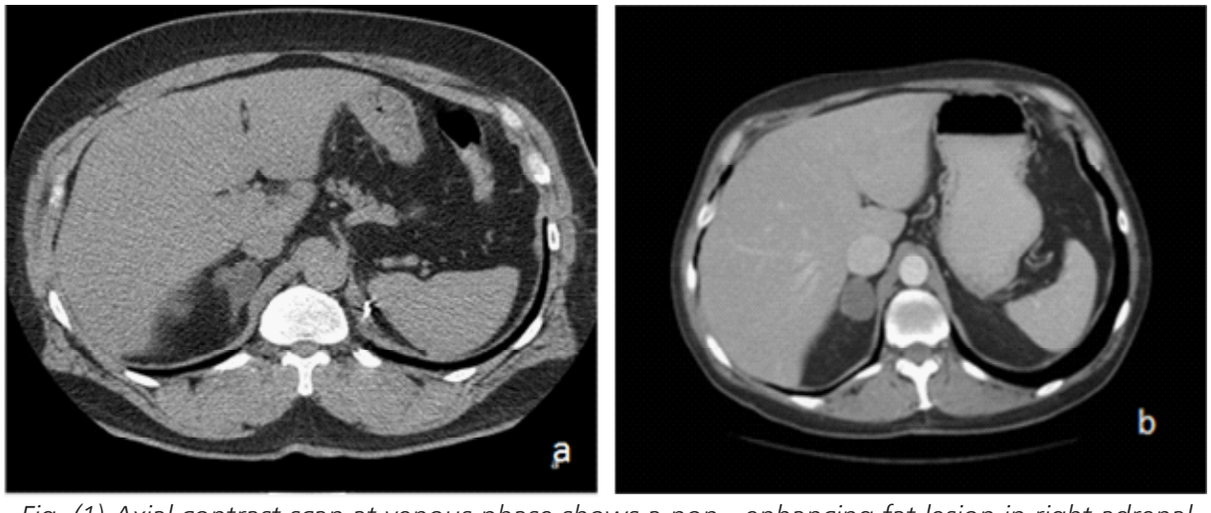


Fig. (1) Axial contrast scan at venous phase shows a non - enhancing fat lesion in right adrenal gland - adenoma (b)

adenoma allows for a quantitative evaluation by measuring the attenuation value of the lesion [1]. An attenuation value of 10 HU (Hounsfield units) or less on unenhanced image is diagnostic of adrenal adenoma with 79% sensitivity and 96% specificity, and no further investigations are required. However, 30% adenomas are lipid-poor showing the attenuation value greater than 10 HU. A dynamic (at 60 s) and delayed (at 10 min) contrast-enhanced CT scan is obtained. The washout of the contrast is a measurement of the percentage decrease between the initial enhancement and the delayed enhancement. A washout of greater than 50% is specific for benign adrenal adenoma, and a washout of less than 50% is specific for metastasis or malignancy. Thus, the contrast-agent washout measurement yields 98% sensitivity and 100% specificity and can reliably determine if an adrenal mass is benign or malignant [2, 3].

Cushings' syndrome(CS): Hypertension is one of the most distinguishing features of Cushing's syndrome. In ACTH-dependent and ACTH-independent CS patients, CT, and/or MRI is the primary imaging modality for localization of the lesion, while scintigraphy is a useful confirmatory imaging method. CT imaging of pituitary typically shows a hypodense lesion that fails to enhance post contrast. Unfortunately, normal-appearing pituitary may occur in some patients with Cushing disease due to both diffuse hyperplasia of ACTH-producing cells and small microadenomas that are not detected on imaging studies [4, 5, 6].

Pheochromocytoma (PH) is an important but rare cause of EH, occurring in about 0.1–0.9% of hypertensive individuals. On CT they are large (2–5 cm), heterogeneous masses with areas of necrosis and cystic change, they typically enhance avidly and may wash out similar to an adrenal adenoma, but they tend to have a greater enhancement in the portal venous phase than the arterial phase. A 110 HU of enhancement on the arterial phase is compatible with pheochromocytoma (Figure 2). [7]. Upto 7% demonstrate areas of calcification. CT and MRI are generally indicated as primary imaging modalities for localization of these tumors. Sensitivities vary between 75% and 100% depending on location at adrenal or extra-adrenal sites and whether the tumor is primary, recurrent, or metastatic. Both imaging methods have poor specificity.

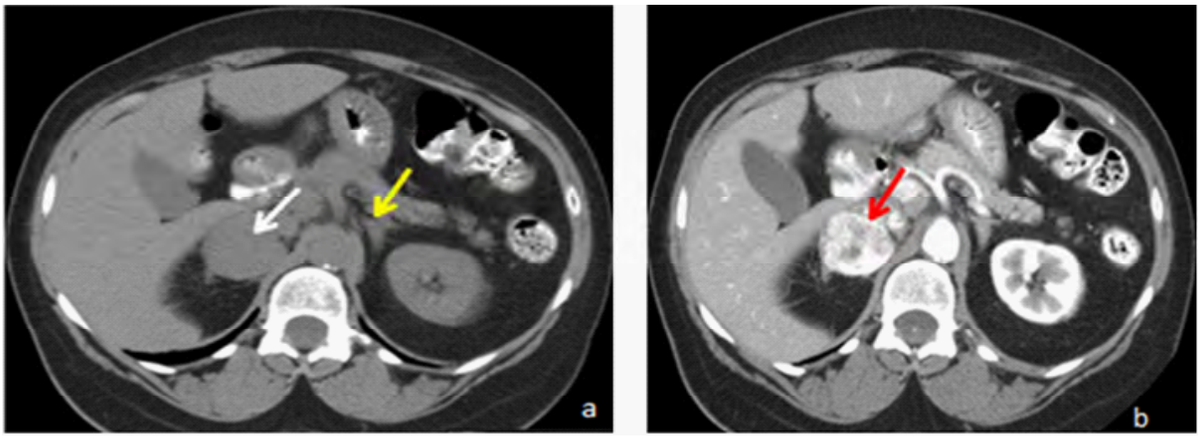


Fig. 2 (a) and (b) Pheochromocytoma. Axial plain and contrast images of right adrenal shows an intensely heterogeneous enhancing nodular lesion in right adrenal gland

However, MRI is more sensitive than CT in detecting extra - adrenal pheochromocytomas (paragangliomas), and is considered as the anatomic imaging of choice because of its excellent anatomic detail, potential for better tissue characterization and multiplanar image capability [8]. Metastatic spread is the only reliable criterion for diagnosis of malignant PH. Skeleton, lymph nodes, lung and peritoneum are the most common sites for metastases.

Acromegaly is another important cause of EH. Approximately 95% of cases are the results of a pituitary adenoma. Pituitary macroadenomas (75%) are more frequent than microadenomas (25%) in patients with acromegaly [9]. MRI of sella is the investigation of choice for evaluation of growth hormone-secreting pituitary adenoma but CT of sellar masses can also be done [10].

CT scan, though less frequently used for evaluating sellar and parasellar lesions, is a useful examination depicting soft tissue calcification, bony destruction, and surgically relevant bony anatomy. CT scans are valuable, particularly when MRI is contraindicated, such as in patients with pacemakers or metallic implants in the brain or eyes [11]. However, less optimal soft tissue contrast and radiation exposure are two important drawbacks that limit the judicious use of CT scan for evaluating pituitary lesions (Figure 3). [10]. Imaging with chest and abdominal CT to detect rare extracranial GHRH-secreting tumor is only indicated if pituitary MRI is normal and there is a definitive biochemical evidence of acromegaly.

Anatomical imaging in hyperparathyroidism: Thinsection, triple phase contrast - enhanced CT (CECT) is used for localization of parathyroid adenoma with sensitivity ranging from 46-87%. CECT demonstrates intensely enhancing nodule posterior to the thyroid gland.

Neuroendocrine tumour of pancreas:

Pancreatic neuroendocrine tumours (pNETS) are a part of a heterogeneous group of tumors, neuroendocrine gastroentero- pancreatic tumors (GEP-NETs), with their origin in

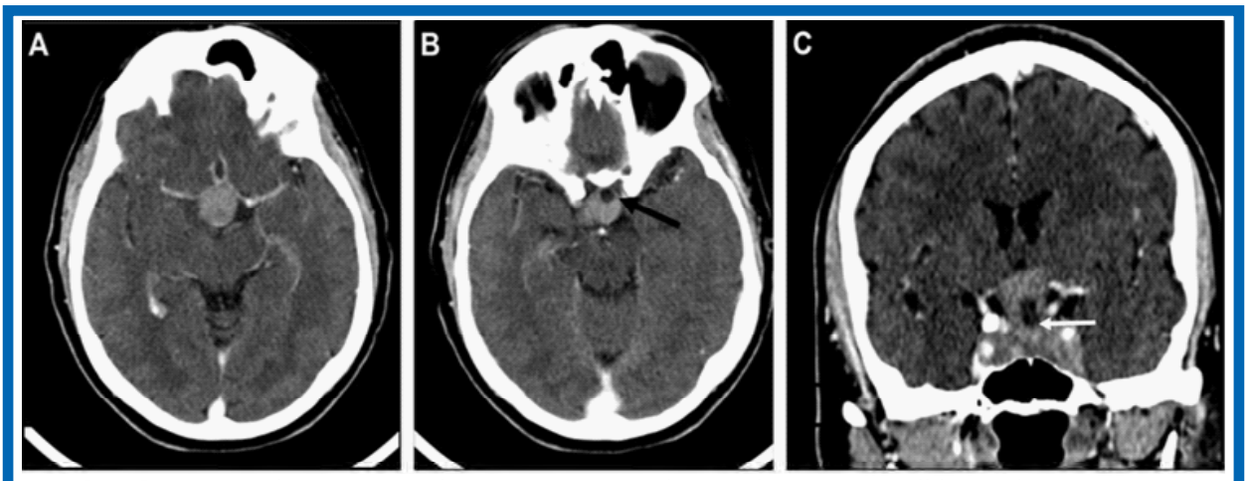


Fig. 3A. CT scan with contrast enhancement in axial view showing a well limited pituitary tumor with suprasellar extension. B. A 29 x16mm tumor involving an anterior cystic component (black arrow) C. Tumor compressing optical chiasma, reaching the inferior part of third ventricle and laterally displacing the cavernous sinus with a part of the posterior-superior cyst (white arrow)

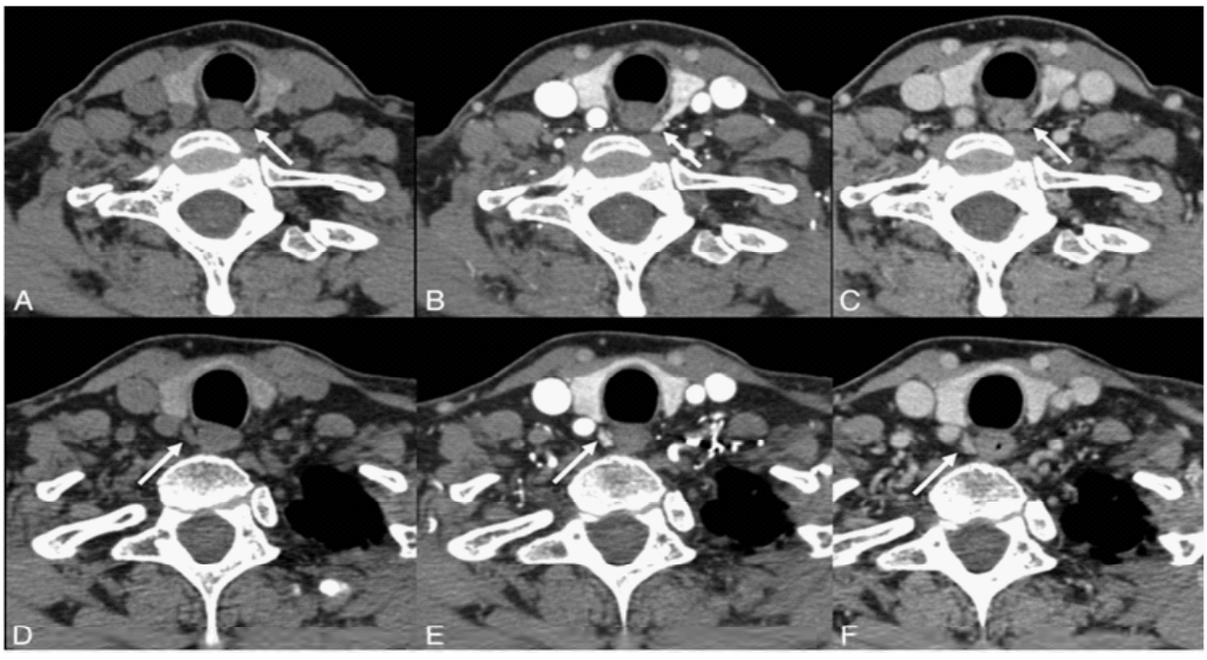


Fig. 4A Axial CT showing avidly enhancing lesions in the orthotopic superior location (arrows) bilaterally with rapid washout of contrast greater than that of the adjecent thyroid gland (A and D: noncontrast phase; B and E: initial postcontrast "arterial" phase; C and F: delayed post-contrast phase).

neuroendocrine cells of the embryological gut. Most commonly, primary lesions are located in gastric mucosa, small and large intestine, rectum and pancreas. The overall incidence of all GEP-NETS has increased over the past decade, with pNET incidence of 0.32/100 000/year. Patients with multiple endocrine neoplasia type 1 (MEN-1) or von Hippel–Lindau's disease (VHL), have pNETS 15–20 years earlier than patients with sporadic pNETS [13]. Pancreatic islet cell tumors (ICTs) are neuroendocrine neoplasms that produce and secrete hormones to a variable degree. These neoplasms can present a diagnostic challenge, both clinically and radiologically. ICT can be syndromic or nonsyndromic. Multi–detector row computed tomography (CT) plays an important role in the diagnosis and staging ICTs. Preoperative detection of ICTs with dual phase CT, demonstrates a sensitivity exceeding 80%. They are typically hyperenhancing and are usually best seen on CT scans obtained during the arterial phase (Figure 5) [14]. Nonsyndromic ICTs tend to be larger than syndromic ICTs at presentation and are more likely to be cystic or necrotic. CT helps is diagnosing these lesion, mass effect cause by lesion, assessment of local invasion of mesenteric vessel and presence of distant metastasis to liver.

Conclusion:

Multi detector row CT is valuable in the diagnosis and staging of neoplasm associated with endocrine dysfunction. These neoplasms can present a diagnostic challenge, both clinically and radiologically. It is important for the radiologist to be familiar with appropriate CT protocol for imaging patients with suspected endocrinopathies and to understand the variable CT appearances of these neoplasms.

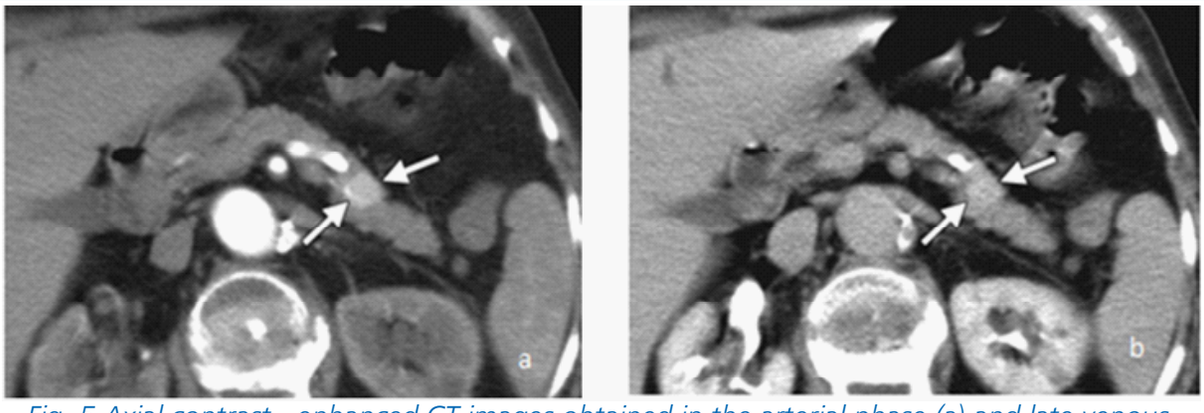


Fig. 5 Axial contrast - enhanced CT images obtained in the arterial phase (a) and late venous phase (b) show a 1.5-cm homogeneous hyperenhacing mass at the junction of the pancreatic body and tail (arrow), The mass is more conspicuous on the arterial phase image than on the venous phase image.

Refrences:

1) Lockhart ME, Smith JK, Kenney PJ. Imaging of adrenal masses. Eur J Radiol. 2002;41:95–112.[PubMed]

2) Blake MA, Kalra MK, Sweeney AT, Lucey BC, Maher MM, Sahani DV, et al. Distinguishing benign from malignant adrenal masses: Multi-detector row CT protocol with 10-min delay. Radiology. 2006;238:578–85. [PubMed]

3) Peña CS, Boland GW, Hahn PF, Lee MJ, Mueller PR. Characterization of indeterminate (lipid-poor) adrenal masses: Use of washout characteristics at contrast-enhanced CT. Radiology. 2000;217:798–802.[PubMed].

4) Nieman L, Cutler GB., Jr . Cushing's syndrome. In: DeGroot LJ, Besser M, Burger HG, et al., editors. Endocrinology. 3rd ed. WB Saunders: Philadelphia Pa; 1995. pp. 1741–69.

5) Sohaib SA, Hanson JA, Newell-Price JD, Trainer PJ, Monson JP, Grossman AB, et al. CT appearance of the adrenal glands in adrenocorticotrophic hormone-dependent Cushing's syndrome. AJR Am J Roentgenol. 1999;172:997–1002. [PubMed].

6) Orth DN. Cushing's syndrome. N Engl J Med. 1995;332:791–803. [PubMed].

7) Reiser MF. Magnetic Resonance Tomography. Springer Verlag. (2007) ISBN: 354029354X.

8) Maurea S, Cuocolo A, Reynolds JC, Tumeh SS, Begley MG, Linehan WM, et al. Iodine-131-metaiodobenzylguanidine scintigraphy in preoperative and postoperative evaluation of paragangliomas: Comparison with CT and MRI. J Nucl Med. 1993;34:173–9. [PubMed].

9) Chaudhary V, Bano S. Imaging of the pituitary: Recent advances. Indian J EndocrMetab. 2011;15:S216–23. [PMC free article] [PubMed].

10) Nimsky C, Ganslandt O, von Keller B, Fahlbusch R. Intraoperative high-field MRI: Anatomical and functional imaging. ActaNeurochir Suppl. 2006; 98:87–95. [PubMed].

11) Cheemum L, Walter K, Walter JM, Laurence EB. Magnetic resonance imaging of the brain and spine.Vol. 2. Philadelphia: WW Lippincott Co; 2002. The sellaturcica and parasellar region; pp. 1283–362.

12) Tziakouri C, Eracleous E, Skannavis S, Pierides A, Symeonides P, Gourtsoyiannis N. Value of ultrasonography, CT and MR imaging in the diagnosis of primary hyperparathyroidism. ActaRadiol. 1996;37:720–6. [PubMed].

13) Yao JC, Hassan M, Phan A, et al.: One hundred years after "carcinoid": epidemiology of and prognostic factors for neuroendocrine tumors in 35,825 cases in the United States. J ClinOncol 2008, 26: 3063–3072.

14) Fidler JL, Fletcher JG, Reading CC et. al.. Preoperative detection of pancreatic insulinomas on multiphasic helical CT. AJR Am J Roentgenol 2003;181(3):775–780. Crossref, Medline, Google Scholar.

Newborn Screening Guidelines for Congenital Hypothyroidism (CH)

Introduction:

Congenital hypothyroidism (CH) is one of the most common preventable causes of mental retardation. In most cases, the disorder is permanent and results from abnormality in thyroid gland development (dysgenesis or agenesis) or a defect in thyroid hormonogenesis. Hence the main objective of CH screening is eradication of mental retardation after congenital hypothyroidism is diagnosed.

The fetal hypothalamic-pituitary-thyroid axis begins to function by mid-gestation and is mature in the term infant at delivery. Despite the critical importance of TH on multiple organ systems, especially the brain, most infants with CH appear normal at birth. The hypothyroid fetus appears to be protected at least in part by placental transfer of maternal Thyroid Hormone.

Normal or near-normal cognitive outcome is possible in even in the most severely affected infants with CH. This is true as long as postnatal therapy is early and adequate and maternal thyroid function is normal. In contrast, when both maternal and fetal hypothyroidism are present, whether attributable to severe iodine deficiency, potent thyrotropin receptor (TSH-R)-blocking antibodies (TRBAbs) (or TSH-blocking immunoglobulins), or maternal-fetal PIT1 deficiency, there is a significant impairment in neuro-intellectual development despite adequate therapy soon after birth.

In addition to the profound clinical benefit, it has been estimated that the cost of screening for CH is much lower (less than rupees 100) than the cost of diagnosing CH at an older age. The overall incidence of CH ranges from 1 in approximately 1000 Indian newborns.

Dr Santosh Kumar,
Dept. of Paediatrics
BARC Hospital

The Sample for Newborn Screening (NBS)

Cord Blood vs. Postnatal Sample:

The crucial point in screening for CH is the neonatal surge of TSH and T4. The TSH surge starts 30 min after birth (T4 some hours later), is most marked for the next 24 h, but may persist for 48 to 72 h. Thus, cord blood is largely spared of the neonatal surge. If the screen sample is taken during the surge, a false positive result will follow, leading to a large number of infants being recalled for a confirmatory sample (a high "**recall rate**"). Parents will be made unnecessarily anxious and the system will be over burdened. The screen sample should therefore be taken either from the cord (placental end, immediately after delivery) or postnatally after 72 h of life. If the hospital stay is shorter, it may be taken after 48 hours of life. Cord blood NBS for CH is an effective strategy to reduce missed opportunities for screening due to early discharge. On the other hand, postnatal samples offer the advantage of screening for other treatable inborn errors of metabolism like congenital adrenal hyperplasia, biotidinase deficiency and those dependent on feeding (galactosemia, Phenylketonuria), and should be used wherever feasible and economically viable.

Recommendation: Either cord blood or postnatal day 3-day 5 samples should be used for screening CH.

The Screening Test:

Two screening strategies for the detection of CH have been evolved: a primary TSH/backup T4 method and a primary T4/backup TSH method. In addition, an increasing number of programs use a combined primary TSH plus T4 approach.

Primary TSH with Backup T4 Measurements:

Most programs in Europe, Japan, Canada, Mexico, and the United States screen by using primary TSH measurements, supplemented by T4 determinations for infants with elevated TSH values. With this approach, delayed TSH elevation in infants with thyroid-binding globulin (TBG) deficiency, central hypothyroidism, and hypothyroxinemia will be missed. Delayed TSH elevation is particularly common in infants with low birth weight (LBW) and very low birth weight (VLBW).

Current TSH assay techniques (enzyme-linked immunoassays, chemiluminescent assays, and fluoroimmunoassays) use nonradioactive labels and have improved sensitivity with the potential for better separation between normal and abnormal TSH concentrations. Thus, many screening programs are considering switching to a primary TSH approach.

The primary T4 approach:

An initial T4 measurement is followed by a measurement of TSH for filter-paper specimens with low T4 values, will detect primary hypothyroidism in infants with low or low-normal T4 with elevated TSH concentrations (prevalence ranging from 1 in 3000 to 1 in 4000 newborn infants). In addition to detecting primary hypothyroidism, the primary T4/backup TSH approach can also identify infants with TBG deficiency (prevalence ranging from 1 in 5000 to 10 000 newborn infants) and central hypothyroidism (low or low-normal T4 with normal TSH concentration; prevalence: l in 50 000 newborn infants). Programs that quantify high T4 values also have the potential to identify infants with hyperthyroxinemia (1 in 20 000 to l in 40 000 newborn infants). This approach, however, will miss the condition in an infant with an initially normal T4 concentration and delayed increase in TSH.

Combined Primary TSH Plus T4 Measurements:

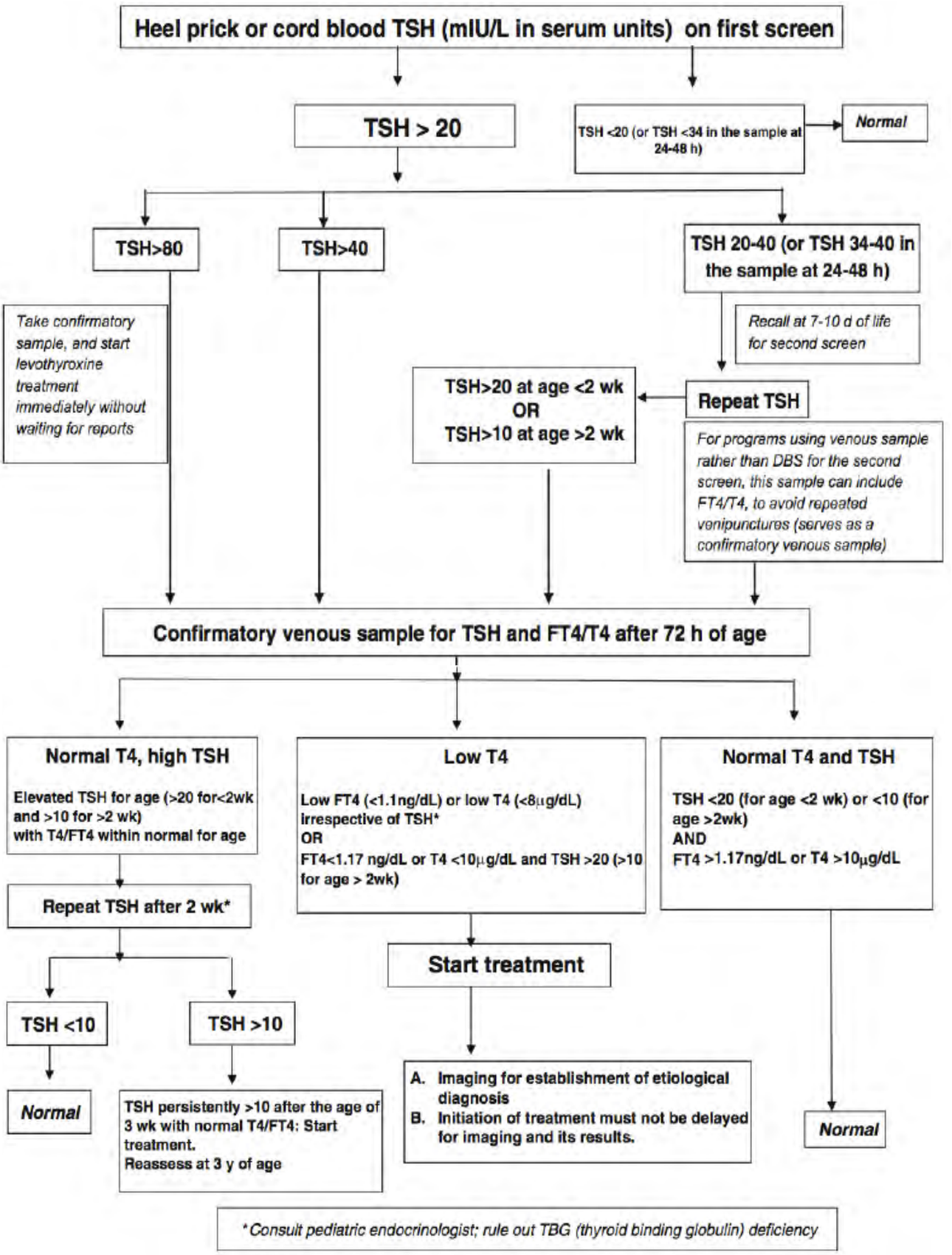
Methods for the simultaneous measurement of T4 and TSH are available. This represents the ideal screening approach.

Until T4 and TSH determinations can be performed practically for all infants, physicians should be aware of the potential limitations of each method of screening for CH.

TSH Cut-Offs to be used for the Screening Test:

Only 1 in a few babies with an abnormal screen TSH value will finally have true CH. To identify the true positives, confirmatory test must be done for all babies whose screen TSH is above a chosen cut-off. Various cut-offs have been used in different studies across the world. A TSH cut-off of >20 mIU/L for recall has been shown to be associated with reasonable specificity and recall rate. Studies which examined the outcome of shifting to lower cut-offs for recall showed substantial increase in recall rates to diagnose a few additional cases of mild CH

Therefore, it is recommend a screen TSH cut-off of >20 mIU/L for recall. Mildly elevated


Heel prick or cord blood TSH (mIU/L in serum units) on first screen
TSH > 20
TSH <20 (or TSH <34 in the sample at 24-48 h)
Normal
TSH>80
TSH>40
TSH 20-40 (or TSH 34-40 in the sample at 24-48 h)
Take confirmatory sample, and start levothyroxine treatment immediately without waiting for reports
Recall at 7-10 d of life for second screen
TSH>20 at age <2 wk
OR
TSH>10 at age >2 wk
Repeat TSH
For programs using venous sample rather than DBS for the second screen, this sample can include FT4/T4, to avoid repeated venipunctures (serves as a confirmatory venous sample)
Confirmatory venous sample for TSH and FT4/T4 after 72 h of age
Normal T4, high TSH
Elevated TSH for age (>20 for<2wk and >10 for >2 wk)
with T4/FT4 within normal for age
Low T4
Low FT4 (<1.1ng/dL) or low T4 (<8μg/dL) irrespective of TSH*
OR
FT4<1.17 ng/dL or T4 <10μg/dL and TSH >20 (>10 for age > 2wk)
Normal T4 and TSH
TSH <20 (for age <2 wk) or <10 (for age >2wk)
AND
FT4 >1.17ng/dL or T4 >10μg/dL
Repeat TSH after 2 wk*
Start treatment
TSH <10
TSH >10
Normal
TSH persistently >10 after the age of 3 wk with normal T4/FT4: Start treatment.
Reassess at 3 y of age
A. Imaging for establishment of etiological diagnosis
B. Initiation of treatment must not be delayed for imaging and its results.
Normal
*Consult pediatric endocrinologist; rule out TBG (thyroid binding globulin) deficiency

screen TSH (between 20 and 40 mIU/L) dictates recall early in the second week of life for a repeat screening TSH (most of the mildly high TSH reports due to unresolved neonatal TSH surge or other reasons would have normalized in a few days). However a clear-cut high screen TSH >40 mIU/L necessitates immediate recall (after 72 h of age) for a confirmatory venous sample (Fig.). The recall rates are high when cord blood samples are used because of its high standarddeviation, therefore these guidelines may need to be revised in future when false negative rates are available using this cut-off level. In centres where the second TSH screen is done using a venous sample rather than a heel prick dried blood sample, both TSH and T4 may be performed on this sample itself, as it may not be cost-effective to recall the family once again for T4/FT4 if needed.

Recommendation:

1. TSH >20 mIU/L (serum units) is recommended as the cut-off for recall for cord blood and postnatal screen samples after 48 h of age.
2. Screen TSH >40 mIU/L is recommended for defining screen positive cases for immediate recall for venous confirmatory test, whereas those with mildly elevated TSH from 20 to 40 mIU/L should have a second TSH screen at 7 to 10 d of age.
3. Age-related TSH cut-off (>34 mIU/L) is suggested for screen samples taken between 24 to 48 h of age.

Decision Making for Recalled Infants:

For clearly elevated screen positive neonates (TSH >40 mIU/L), parents should be contacted immediately by an appropriate health worker or doctor for obtaining the confirmatory venous sample, and the baby should be evaluated by a pediatrician or pediatric endocrinologist and levothyroxine therapy initiated if indicated. Measurement of venous serum T4/FT4 and TSH are done by chemiluminescence or ELISA assay. Before 2 wk of age, venous TSH >20 mIU/L and after 2 wk of age, >10 mIU/L, is indicative of primary CH. Serum T4 < 10 μg/dL(<128 nmol/L) or FT4 < 1.17 ng/dL (<15 pmol/L) is considered low in infancy (in contrast to lower levels in older children and adults). Babies with screen TSH >80 mIU/L serum units are highly likely to have low T4 or FT4 levels, therefore commencement of therapy is recommended as soon as the confirmatory sample is taken, without waiting for the results unless these results are available on the same day. For mildly elevated TSH results as defined above, a repeat sample should be obtained as early as possible, in the second week of life. This will ensure treatment initiation within two weeks of life for better neurodevelopmental outcome. The reason for not taking the repeat sample immediately is to allow the neonatal factors causing a false positive result to settle down. If the second screen TSH is high (>20 mIU/L for age<2 wk and >10 mIU/L for age>2 wk), immediate confirmatory venous sample for T4/FT4 and TSH measurement should be taken. Where a venous sample has been taken for the second screen, the TSH and T4/FT4 in that sample will serve as the confirmatory venous sample.

Decision Making for Borderline Thyroid Function Reports:

For elevated venous TSH and normal FT4 levels, the baby may be retested after 2 wk (treatment may be started immediately if there is any doubt about compliance to instructions). At thispoint, referral to a pediatric endocrinologist is made wherefeasible. After 3 wk of age, if TSH remains persistently >10 mIU/L even with normal range of T4/FT4, levothyroxine treatment may be started to avoid insult to the developing brain.

Thyroid imaging (Ultrasonography and Nuclear scan) may also help to get a definitive

diagnosis. Re-evaluation is recommended after 3 y of age, by which age the phase of rapid brain development has been achieved.

Recommendation:

1. Infants with persistently elevated TSH >10 mIU/L (after 3 wk of age) with normal T4/FT4, or normal confirmatory sample TSH with clearly low T4 (<8 μg/dL) / FT4 (<1.1 ng/dL) may be treated with levothyroxine, and a reevaluation off therapy should be planned after 3 y of age.
2. Referral to a pediatric endocrinologist is suggested for all babies diagnosed with CH especially those with borderline results of thyroid function.

Special Situations (Twins, IUGR, Preterm and Sick Neonates):

High risk neonates such as preterm, low birth weight (LBW) (1500–2499 g), very-low birth-weight (VLBW) (1000–1499 g) and sick neonates, multiple births, particularly same sex twins are at increased risk for an inappropriate TSH level at initial screening (both false positive and false negative). The postnatal TSH surge and rise in thyroid hormones seen in term infants are present in preterm infants as well, but attenuated owing to immaturity of the Hypothalmic- Pituitary- Thyroid (HPT) axis. Moreover, preterm and sick infants often have a fall in serum T4 and T3 in first week of life, which may be due to poor nutrition, decreased hepatic TBG production, immaturity of the HPT axis, use of iodine antisepsis and increased tissue utilization of T4 or sick euthyroidism. Sick euthyroid syndrome (low serum T4 with normal TSH) resulting from associated medical problems, such as respiratory distress syndrome or the consequences of intrauterine growth retardation (IUGR) may persist until the infant recovers from the acute illness or gains weight. No causal relationship has been established between hypothyroxinemia of prematurity and problems in neuro development and intellectual disability; current evidence does not indicate benefit from therapy of hypothyroxinemia of prematurity in the absence of raised TSH.

In the majority of preterm infants, T4 rises into the normal range when a repeat screening test is performed at two to four weeks of age, as the HPT function matures. Occasionally, recovery from sick euthyroidism may lead to mild elevation of TSH and give rise to false positive screen results. Conversely, preterm infants with true CH may not be ableto mount an appropriate TSH response in the first 2 weeks of life due to immaturity of the HPT axis or treatment with glucocorticoids or dopamine, leading to false negative initial screen. Various studies have reported a higher incidence of CH (both transient and permanent) in preterm, VLBW and LBW infants, who may be missed in the first screen. A second screen done after 2 weeks of age will pick up the delayed rise of TSH. On the contrary, the cases of CH detected by a second screen often represent mild or transient CH, and many question the policy of re-screening at 2 weeks.

Another group at high risk of CH consists of newborns with Down syndrome - not only do these infants have a higher incidence of CH picked up by NBS, but they may also have mildly elevated TSH levels that can be missed by screening and therefore, require careful follow-up and re-testing before 6 months of age.

As for all newborns, preterm and LBW/VLBW infants should undergo routine screening for CH only at 48–72 hours postnatal age, not earlier. Only in instances of acute hemorrhage or hemolysis, when transfusion is warranted,

they may be screened before 24–48 h of birth.

With sick infants in NICUs, screening should be performed at least by 7 d of postnatal life. It is suggested to do a second screening test at 2–4 wk of age for high-risk babies, though not at the expense of missing infants with severe CH in an already overburdened system. The final TSH cut-offs for preterm, LBW/VLBW infants and twins remain the same as for term infants.

Recommendation:

1. As for all newborns, preterm and LBW/VLBW infants should undergo routine screening for CH at 48–72 hours postnatal age.
2. Sick neonates should be screened at least by 7 days of age.
3. High risk neonates such as preterm, LBW and VLBW babies, ill neonates admitted to NICU and multiple births (particularly same sex twins) may have a second screen at 4 weeks of age (or at 2 weeks of age if discharged early).

Conclusions:

NBS for CH is one of the most successful interventions in preventive pediatrics worldwide. Till such time as NBS is integrated in national health programmes in India, pediatricians and health authorities should make concerted efforts to initiate NBS for CH for all newborns in their care.

References:

1. Newborn Screening Guidelines for Congenital Hypothyroidism in India: Recommendations of the Indian Society for Pediatric and Adolescent Endocrinology (ISPAE). The Indian Journal of Pediatrics (June 2018) 85(6):440–447.
2. Update of Newborn Screening and Therapy for Congenital Hypothyroidism. PEDIATRICS Volume 117, Number 6, June 2006.

Retinopathy of Prematurity: An Overview

Dr Ronak Parekh, Dr Sayali Bhedasgaonkar, Dr Snehal Nadkarni, Dr Akshita Patel
Dept. of Ophthalmology,
BARC Hospital

Introduction

Retinopathy of prematurity (ROP) is a multifactorial vaso-proliferative retinal disorder seen in premature infants with low birth weight. Worldwide incidence of the disease is 60% in premature babies. In India, the incidence rate is reported to be around 24 to 47%. This may be due to underdiagnosis. Around 90% of early stage ROP cases regress spontaneously with nil or minimal visual disturbances. The estimated risk of blindness without treatment in threshold ROP is 50%.

Dr Ronak Parekh

History

ROP was first described by Terry in 1941. It was originally known as Retrolental Fibroplasia. The term Retinopathy of Prematurity was coined by Health in 1951. Campbell suggested the relationship of intensive oxygen therapy & subsequent development of ROP. Kinsey suggested that ROP is inversely proportional to birth weight.

Risk factors

Prematurity in terms of low birth weight or gestational age is the most important risk factor for development of ROP. High concentration of oxygen administration for a long period is also considered as an important risk factor. Other factors are hyperoxia or hypoxia, postnatal sepsis, mechanical ventilation, intraventricular hemorrhage, apneic attacks, patent ductus arteriosus and multiple blood transfusions.

Pathogenesis

Normally, retinal vascular development occurs in two phases. Phase 1 is independent of Vascular Endothelial Growth Factor (VEGF) and occurs from 8 to 21 weeks of fetal development. Spindle cells (mesenchymal precursor cells) appear around optic disc region and then cords of spindle cells advance

Classification of Retinopathy of Prematurity: Locations

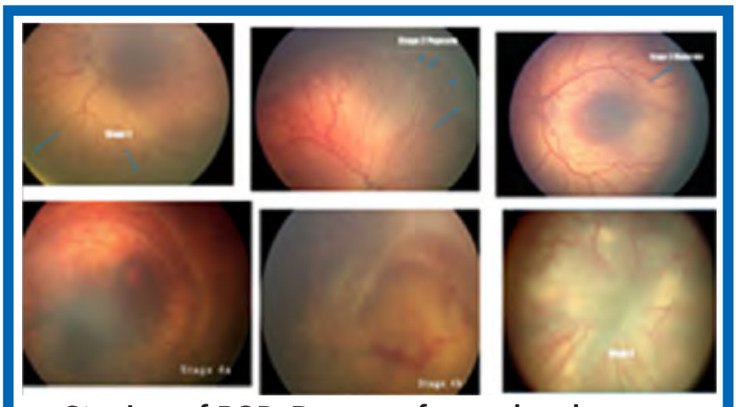
towards oraserrata and differentiate into arterioles and venules. Phase 2 dependent on VEGF occurs from 22 to 40 weeks of development in which proliferating endothelial cells migrate from existing blood vessels to form new capillaries. Premature babies exposed to high oxygen after birth, which leads to vaso-obliteration and cessation of vessel growth due to down-regulation of VEGF. When oxygen exposure is reduced there is a pathological release of VEGF from avascular retina that leads to neovascularization.

Classification

The International Classification of Retinopathy of Prematurity (ICROP 1984-1987, revised in 2005) has following components:

1. Location: It refers to how far the developing retinal vessels have progressed.

- Zone 1 consists of an imaginary circle with optic nerve at the center and a radius twice the distance from optic nerve to the macula.
- Zone 2 extends from the edge of zone I to the oraserrata on the nasal side of the eye and to the equator on the temporal side.
- Zone 3 consists of the outer crescent shaped area extending from the Zone II out to the oraserrata temporally.

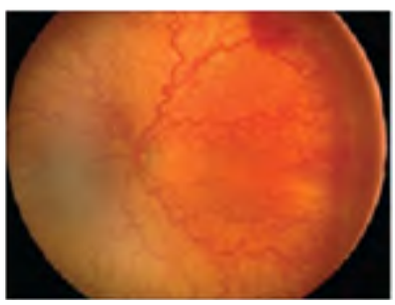


Staging of ROP: Degree of vascular changes

2. Extent of the disease: It is described by the number of clock hours involved.

3. Staging: It is defined according to the degree of vascular changes.

- Stage 1 - A demarcation line appears as a thin white line that separates the normal retina from avascular retina.



Plus disease

- Stage 2 - A ridge of fibro-vascular tissue with height and width replaces the demarcation line. It extends inwards from the plane of retina.
- Stage 3 - The ridge has extra-retinal fibrovascular proliferation. Abnormal blood vessels and fibrous tissue develop on the edge of the ridge and extend into the vitreous.
- Stage 4 - Partial retinal detachment may result when the scar tissue pulls the retina.
- Stage 5 - It indicates complete retinal detachment. The retina assumes a funnel shaped appearance and is described as open, narrow or closed funnel.

4. Plus disease: It is an additional designation that refers to the presence of vascular dilatation and arteriolar tortuosity of posterior retinal vessels in at least 2 quadrants. This indicates

the severe form of the disease. It may be associated with iris vascular engorgement, pupillary rigidity and vitreous haze.

Pre-plus disease: It is defined as vascular abnormalities of posterior pole that is insufficient for the diagnosis of Plus disease but demonstrates more arterial tortuosity and more venous dilatation than normal.

Screening strategies

Screening is recommended in all the babies with birth weight <1750gm and gestational age <30 weeks. The babies born after 30 weeks of gestation may be considered for screening if they have been ill (RDS, hypotension, surgery).

It should be performed at 34 weeks of gestation or 4 weeks of post natal age, whichever is earlier. The procedure is performed in Neonatal Intensive Care Unit (NICU) by Ophthalmologist specialized in ROP, under the supervision of neonatologist. The pupils are dilated with a mixture of Phenylephrine 2.5% and Tropicamide 0.4% eye drops or Cyclopentolate 0.5% eye drops instilled 3 times at 15 minutes interval before the scheduled examination. Indirect Ophthalmoscopy is performed with 20D / 30D lens using fresh sterile instruments. Scleral depression is done to stabilize the eye, rotate it, indent it and contrast the retina. RETCAM can be used to provide real time video display of images. Topical anesthetic eye drops should be used to reduce discomfort. Atleast two fundal examinations should be performed after dilatation using binocular Indirect Ophthalmoscope.

Screening should be stopped in following situations:

- Complete vascularization.
- Vascularization in Zone III (till 1 DD of temporal oraserrata) if no previous ROP in zone I & II.
- Regressed ROP between 40-44 weeks PCA.
- 45 weeks PCA with less than pre-threshold disease.

Management pearls of ROP

Treatment of ROP is indicated in following cases:

Threshold ROP-It is coined by CRYO ROP Study.

- Zone I stage III with Plus
- Zone II stage III with Plus
- 5 contiguous or total 8 clock hours

Pre-Threshold ROP-It is coined by Early Treatment of ROP Study (ETROP)

High risk Pre-threshold (Type1)

- Zone I any stage with plus or
- Zone I stage 3 without plus or
- Zone II stage 2 and 3 with plus

Low risk Pre-threshold (Type 2)

- Zone I, stage 1 or 2 ROP without plus or
- Zone II, stage 3 ROP without plus

The main principle of the treatment is to ablate the ischemic peripheral retina to stop the release of angiogenic factors. Following modalities of treatment are available:

- Cryotherapy-Multiple applications are made to treat the entire avascular retina anterior to the ridge. However, cryotherapy requires general anesthesia, has more local complications like severe lid edema and is not feasible for zone 1 cases.
- Laser Photocoagulation - It is a practical alternative after the advent of the Indirect Laser System. The main advantages are that it can be performed under topical anesthesia, with less systemic and local complications compared to cryotherapy and

can be done as an out-patient procedure. Also, posterior retina in zone 1 can be reached easily.

- Anti VEGF intravitreal injections – Anti VEGFs like Ranibizumab and Bevacizumab are the new treatment modalities for ROP.
- Surgical treatment – Surgery is advocated if Laser or Cryotherapy is unsuccessful in preventing progression to stage 4 or 5 ROP. For stage 4A or 4B, scleral buckling or lens sparing vitrectomy can be performed while for stage 5, lensectomy along with vitrectomy or open sky vitrectomy are the available options.

Statistics of ROP in BARC Hospital

A total of 11 patients were screened for ROP in the year 2018. 6 babies were diagnosed to be positive and treated for ROP. 4 required both Anti VEGF injection and Laser treatment whereas 2 babies were managed by Laser alone. 4 babies were found to be negative and 1 was lost to follow-up. All the treated babies have responded well to the treatment, avoiding the progression of the disease with chances of better visual outcome in future.

Conclusion

ROP is a serious blinding disease. The increase in incidence may be due to improving survival rate of premature babies, better diagnostic approaches and awareness about the disease. A two pronged approach is needed to tackle the emerging epidemic of blindness due to ROP in India. This requires close collaboration between neonatologists, pediatricians and ophthalmologists. All premature low birthweight babies receiving oxygen therapy should be closely and continuously monitored.

References:

1. Le crystal et al. Retinopathy of prematurity: Incidence, Prevalence, Risk factors and outcome, at a tertiary care centre in Telangana, JCOR.2016; 4: 119-122.
2. Text book of American Academy of Ophthalmology, Retina and vitreous.
3. ICROP Committee for the classification of late stages of Retinopathy of Prematurity, II. The classification of retinal detachment. Arch Ophthalmol. 1987; 105:906-912.
4. Section on Ophthalmology, American Academy of Paediatrics, American Academy of Ophthalmology, American Association for Pediatric Ophthalmology and Strabismus. Screening examination of premature infants for retinopathy of prematurity. Paediatrics 2006;117:572-576.

Review of probable contaminants isolated from blood cultures at BARC Hospital and significance of right technique for blood culture collection

Dr Sunayana M. Jangla (Microbiologist),
Dr Susan Cherian (Pathologist),
Dept. of Pathology

Introduction:

Blood culture is an important tool for detecting the presence of harmful living micro-organisms in the bloodstream. A positive blood culture may indicate a definitive diagnosis and prove life-saving. However, false positive results (contaminants) can limit the utility of this tool. Contaminated blood cultures are enormously costly (for patient as well as hospital/ laboratory), prolong hospitalisation, confuse the clinicians, can cause drug toxicity and lead to antibiotic resistance due to unnecessary use of antibiotics. These multidrug resistant organisms can spread to other patients, thus increasing the chances of hospital associated infections. Such is the devastating effect of contaminated blood cultures.

Tools that aid in interpreting the clinical significance of positive blood cultures:

hese help the health care workers to decide if the isolate is a pathogen or contaminant, namely identity of the organism, clinical features of the patient, number of blood culture sets that are positive, time for culture to be positive (time for growth to be detected) and number of blood culture vials within a set that show growth. Organisms which almost always are pathogenic are *Staphylococcus aureus*, *Streptococcus pneumoniae, E. coli* and other members of Enterobacteriaceae group, *Pseudomonas aeruginosa* and *Candida albicans*. But *Coagulasenegative Staphylococcus (CONS), Bacillus species* (except *anthracis*), *Micrococcus species*, *Corynebacterium species*,

Dr Sunayana M. Jangla

Streptococcus viridians and *Propionibacterium acnes* are presumed to be contaminants as they are normal flora of the skin especially if they grow in a single blood culture. Acceptable percentage of contaminated blood cultures is up-to 3% and target should be less than this. However, in the recent years an increase in the proportion of blood culture isolates representing contaminants has been noted as compared to the past. This may be due to newer continuously monitoring blood culture systems, media containing resin and the increased use of central venous catheters to obtain blood culture samples. *Coagulase negative staphylococcus* is known to grow in blood cultures of patients with infective endocarditis and those with prosthetic valves. But in recent years, this isolate is gaining significance as the etiologic agent of catheter-associated bacteraemia. Similarly, non-fermentative gram-negative bacteria are increasingly being isolated from blood cultures. These are not part of the normal flora of human skin and are ubiquitously distributed in

the hospital environment and associated with implantation of intravenous catheters or other foreign bodies in patients with underlying immunocompromised condition. But due to their low virulence, their significance in clinical specimens should be highly suspicious. These are often colonizers rather than infecting agents.

Data from BARC Hospital: 2,527blood culture samples for aerobic culture were received in microbiology laboratory of BARC hospital between January 2018 and December 2018 from different wards, intensive care unit and OPD. Tools like identity of organism, clinical features, time for culture to be positive and presence of vascular catheter were used to analyse if the isolate was a true pathogen or probable contaminant. 298 isolates of non-fermentative Gram-negative organisms and Gram-positive organisms *other than Staphylococcus aureus* were obtained. Of these, isolates were more from males as compared to females and highest number were from adults followed by geriatric and paediatric age group. Maximum number of these were from ward 4A (Female medical) followed by ward 4B (Male medical), 2B (Paediatric), OPD and ICU. Commonest symptom among patients from whom these isolates were obtained was fever followed by cough with breathlessness, loose motions with vomiting, cough with cold and urinary complaints. Details of organism, time for culture to be positive, presence of vascular catheter and presence of immunocompromised condition (diabetes mellitus, chronic kidney disease on dialysis, malignancy, HIV positive, steroid intake in COPD and Bronchial asthma) is given below in Table-1.

Most of these blood cultures were positive after 48 hours of incubation where-as pathogens are obtained within 24 hours in automated blood culture systems. All were received as single blood cultures except 4 samples which were obtained as two pairs, each pair from two different patients. The number of patients with vascular catheter like central line was very low and immunocompromised state was present in some patients only. 4 patients had vascular catheter along with immunocompromised state. "At risk" patients (immuno compromised and presence of catheters) were 84. Probable contaminants were 214 which indicates prevalence of blood culture contamination rate to be 8.5%. Hence, focus should be on decreasing the rate of contaminants and encouraging the growth of pathogens to prevent treatment failure in the patient.

Measures to decrease the number of contaminated blood cultures: Measures can be taken at various steps of blood culture collection technique focussing mainly on the following steps:

1. Site: Blood cultures should be obtained from venipuncture rather than from an intravascular catheter.
2. Standard precautions: Hand washing with soap and water should be done and gloves should be worn before the procedure.
3. Skin disinfection: Use of Chlorhexidinegluconate and allowing it to dry before collection can aid in doing so.
4. Number of blood culture sets and vials per set: This should be according to current standard guidelines.
5. Training: Regular training about blood culture collection technique can reduce contamination rates.

References:

1. Forbes BA, Sahm DF, Weissfield AS. Bailey and Scott's diagnostic microbiology. 12th edn. Missouri: Mosby Elsevier 2007:334-362
2. Weinstein P M. Blood Culture Contamination: Persisting problems and Partial Progress. J Clin Microbiol 2003; 41(6):2275-2278.

Table 1:

Organism (Number)	<24 hours	24-48 hours	>48 hours	No. of patients having vascular catheter	No. of patients having immuno compromised condition
Gram positive					
Micrococcus species (144)	01	69	74	01	32
Coagulase Negative Staphylococcus(CONS)(60)	02	27	31	03	19
Corynebacterium species(27)	0	10	17	0	14
Bacillus species (other than *anthracis*) (11)	01	02	08	0	02
Kocuria species (1)	0	0	01	0	0
Gram negative (Non-fermentative organisms)					
Sphingomonaspaucimobilis (13)	0	04	09	0	03
Achromobacterxyloso xidans(9)	0	02	07	0	0
Ochrobacter Anthropii(8)	0	01	07	0	01
Acinetobacterbaumannii(6)	0	06	0	02	02
Stenotrophomonasm altophila(5)	0	02	03	01	02
Achromobacterdenitrificans (3)	0	0	03	0	02
Acinetobacterlwoffii(2)	0	02	0	0	01
Brevundimonasdiminuta(2)	0	01	01	0	0
Pseudomonas putida(2)	0	0	02	0	01
Brevundimonasvesicularis(1)	0	0	01	0	01
Burkholderia cepacia(1)	0	0	01	0	0
Burkholderiapseudomallei(1)	0	0	01	0	01
Pseudomonas stuttzerii(1)	0	01	0	0	0
Rhizobium radiobacter(1)	0	0	01	0	0
Total (298)	**04**	**127**	**167**	**07**	**81**

3. Fairfax R M, Salimnia H. Beware of Unusual Organisms Masquerading as Skin Contaminants Intech Open, DOI:10.5772/50300.

4. Rattanaumpawan P, Ussavasodhi P, Kiratisin P, Aswapokee N. Epidemiology of bacteraemia caused by uncommon non-fermentative gram-negative bacteria. BMC Infectious Diseases 2013;13:167.

5. M47-A: Principles and Procedures for blood culture; Approved Guideline-CLSI 6. Pathology users Handbook uploaded on Pathology department site on LAN.

Dental Care

Dr Julli Bajaj
Department of Dentistry
BARC Hospital

We all know that health is wealth and it starts right at the oral cavity. To maintain good oral hygiene, millions of people use toothbrush every day. Due to variations in practice and availability of variety of products in the market, a dentist faces lots of queries from patients, regarding brushing of teeth. Some of the frequently asked questions are answered in this article.

• Which is the best toothbrush?

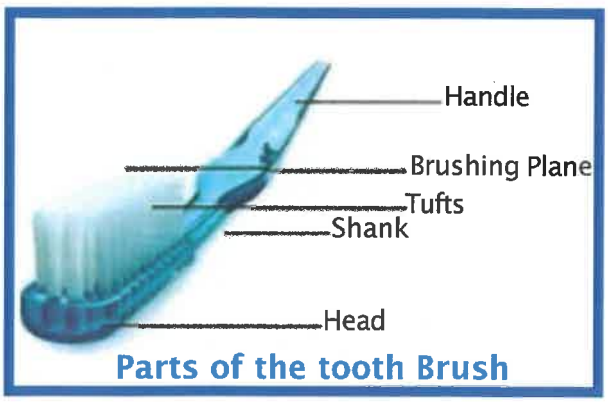
According to American Dental Association (ADA), Ideal characteristics of manual toothbrush:

1. It should confirm to individual patient requirement in shape, size, and texture.
2. It should be designed for utility, efficiency and cleanliness.
3. Toothbrush should have a long, wide and slightly bent handle (for a firm grasp).
4. Soft nylon bristles with rounded ends so that it does not harm the gums.
5. Toothbrush head should be small (1"by1/2") for easy access to all areas of mouth, teeth and gums.

Brushing surface should be concave with following measurements:

1. 1-1.5 inches in length (25.4 to 31.6 mm long).
2. 5/16 to 3/8 inches in width (7.5 to 9.5 mm wide).
3. 2 to 4 rows of bristles.
4. 5 to 12 tufts per row.

Bristle hardness is proportional to square of the diameter and inversely proportional to the square of bristle length. Toothbrushes are characterized as soft, medium and hard according to bristle diameter.



Parts of the tooth Brush

1. Soft bristles – 0.2 mm (0.007inch)
2. Medium bristles – 0.3 mm (0.012 inch)
3. Hard bristles – 0.4mm (0.014 inch)

It is always recommended to use soft toothbrush with double angulation of head and neck.

- **How frequently the brush should be changed?**

It should be changed periodically after every 3 months.

- **How long should one brush the teeth?**

It is an imperative that one should brush the teeth the right way, using the right tools and technique. If the technique is wrong, it won't matter how long one spends brushing. The recommended time by ADA is two minutes twice a day.

- **What is the right technique of brushing?**

The toothbrush should be held at a 45- degree angle to the gums and making up-and-down motion. One should brush outer and inner tooth surfaces, back molars, and your tongue.

- **What type of toothpaste to use?**

Toothpaste with the ADA seal of approval which means that adequate evidence of safety and efficacy have been demonstrated in controlled, clinical trials, preferably with added fluoride should be used.

If the teeth are hypersensitive to hot and cold sensitive toothpaste designed for sensitive teeth should be used. These desensitizing toothpaste, contains Strontium Chloride or Potassium Nitrate, protect exposed dentine by blocking the dentinal tubes connected to pulp tissue. Desensitizing pastes must be used for at least one month before any therapeutic effects are felt.

- **Which toothbrush is better electric or manual?**

Electric brushes are both rechargeable and non-rechargeable. Electric brushes have features such as a timer that turns the toothbrush off after two minutes and pressure sensors that regulate the speed of the toothbrush. This ensures that one does not use too much pressure while brushing, make them more efficient than manual toothbrushes.

The head of these brushes work by four technologies (table-1). The frequency of oscillation is 40Hz.

Electric and manual toothbrushes are effective brushing tools; features that equate to benefits are more a matter of personal preference. Most people use the electric gadgets incorrectly which thins the enamel. Electric toothbrushes are recommended for targeted population who has limited manual dexterity, such as a disabled or elderly person, patient facing shoulder or arm problem, arthritis, bed ridden and those who wear braces etc.

Major drawback with electric toothbrushes is though they are programmed the efficiency of electric toothbrushes depend on batteries, you will either have to charge your toothbrush or

Table: 1

Technology	Circular	Counter oscillation	Sonic	Oscillating/ rotating
Description of motion.	The brush head rotate in one direction.	Different tufts on brush head rotate in different direction.	Side to side motion.	The brush head spins a quarter-turn in one direction, then a quarter-turn in the other.

replace its batteries timely. Traveling with an electric toothbrush is hassle; they are bulky and require a charger. Dropping electric toothbrush can be fatal. They don't have flexible neck which helps you to brush inaccessible areas of the mouth and are costly.

Conclusion:

There have been a few studies that prove electric toothbrushes remove statistically significant more plaque compared to manual toothbrushes and are better at fighting gum disease. Despite the studies, oral health remains healthy as long as one consistently takes care of the teeth. No matter which type of toothbrush is used, it is vital to use right technique.

References:

1. Carranza's Clinical Periodontology- 10 edition.
2. Essentials of Preventive and Community Dentistry- 4 edition
3. Ida Marini, et al Combined effects of repeated oral hygiene motivation and type of toothbrush on orthodontic patients: A blind randomized clinical trial. Journal the Angle Orthodontist: September 2014, Vol. 84, No. 5, pp. 896-901.
4. C.Deery et al The effectiveness of manual versus powered toothbrushes for dental health: A Systematic review Journal of Dentistry. March 2004, Vol.32, issue 3, pp. 197-211.

Corrigendum

PULSE, April 2017 Volume 18, page 8. - "New anti-cancer medicines developed"
The article erroneously mentions that 'Scientists of Radiation Biology and Health Sciences at BARC, have developed two anti-cancer medicines from the fruit extract of the Rampatri plant.'

The corrected sentence should read as 'Scientists of Bio-Organic Division at BARC, have developed two anti-cancer medicines from the fruit extract of the Rampatri plant'. The error is regretted.

Editor, Pulse

NEWER SERVICES

Fertility Clinic at BARC Hospital

The Department of Obstetrics and Gynaecology has started a new facility of Fertility Clinic in Room No. B-23, Ground floor at BARC Hospital. The facility was inaugurated by Smt Debarati Basu on July 14, 2017.

The clinic has been approved for all Assisted Reproductive Technology (ART) procedures under PCPNDT act by the Government of Maharashtra. At present, Intrauterine Insemination (IUI) procedures are performed at this clinic. The infrastructure consists of a collection room, andrology laboratory and a consultation room. The collection room is in the vicinity of andrology lab and provides privacy for semen collection. Andrology lab has facility for refrigeration, storage of injections and media. It has a workstation with laminar air flow hood, phase contrast microscope, Mackler chamber, test tube warmer and centrifuge. Semen wash and preparation is performed here prior to the procedure of IUI.

The consultation room has facility for couple counselling, in-house sonography for follicular studies and to perform procedure of IUI. The team at fertility clinic consists of a part time infertility specialist, an embryologist and a technologist. The data till December 2018 shows that 269 patients were registered at fertility clinic, 1350 follicular studies were performed and 110 women underwent IUI with success rate of 20.9%. The clinic also plans to provide sperm cryopreservation facilities in near future which will allow couples to store semen samples for future IUI cycles.

Dental & Radiology Units at Kharghar Dispensary

A full-fledged Dental section at Kharghar dispensary was made operational by Dr Mala Sankav, Head, Dental Unit on all weekdays with effect from August 2018 for the benefit of Kharghar dispensary CHSS beneficiaries with the help of dental technical staff & MO I/C Kharghar dispensary, Dr Harry Ralte, under the motivation & guidance of Head Medical Division along with the help of TSD / Civil / Computer Division.

Radiology Unit at Kharghar was made operational by Dr Ajay Chaubey (Head, Dept. of Radiology) in April 2018 and functions on every Monday, Wednesday and Friday.

PUBLICATIONS

Department of Anaesthesia

1) Contributions of academic institution in high income countries to anesthesia and surgical care in low income countries: Are they providing what is really needed? Chellam S, Ganbold L, Gadgil A, Orgol S, Lonnee H, Roy N, Gelb AW. Can J Anaesth. 2018.

Semen Examination using Phase Contrast Microscope

Andrology Workstation with Laminar Flow

Spermfuge: Centrifuge with temperature control

Department of Gynaecology

1) Placenta percreta: A successful conservative outcome. Dr. Mishra, Dr. Desai, Dr Prabhu, Dr. Jadhav. International Journal of Reproduction, Contraception, Obstetrics and Gynaecology, vol.7, issue 8, August 2018.
2) "Masik Pali Aani Takrari" Dr. Santoshi Prabhu, 'Amhi Marathi' by BARC Marathi Bhasha Parishad, March 6, 2018.

Department of Pathology / Microbiology

1) Study of Bacteriological profile and antibiotic sensitivity pattern in samples received from patients attending a tertiary care hospital in Mumbai. Dr. Sunayana M. Jangla, Dr. Raji Pillai, Journal of Evolution of Medical and Dental Sciences, 2018, 7(3), 284-290.
2) Speciation and antifungal sensitivity testing of Candida isolates in various clinical samples in a tertiary care hospital in Mumbai. Dr. Sunayana. M. Jangla, Dr. Raji Pillai, Mrs. Sofia C. Patel. International Journal of Biomedical Research, 2018, 9(3), 106-111.
3) Role of GeneXpert MTB/Rif in detection of tuberculosis from clinical samples: A report from a tertiary care hospital in Mumbai. Dr. Sunayana M. Jangla, Dr. Susan Cherian, Dr. Raji Pillai. International Journal of Microbiology Research, 2018, 5 (1), 143-146.
4) Post-operative wound site infection caused by Nocardia species. Dr. Sunayana M. Jangla, Mr. Bhupesh S. Machhi. Journal of Krishna Institute of Medical Sciences, 2018,7(2),99-101.
5) Human Salmonellosis caused by rare Salmonella serotypes -Report of two cases. Dr.Sunayana M. Jangla, Dr. Susan Cherian, Mrs. Sofia C. Patel. Journal of Krishna Institute of Medical Sciences, 2018,7(4), 113-116.
6) Hydroxychavicol, a key ingredient of Piper betle induces bacterial cell death by DNA damage and inhibition of cell division. Deepti Singha, Shwetha Narayanamoorthy, Sunita Gamre, Ananda Guha Majumdar, Manish Goswami, Umesh Gami, Susan Cherian, Mahesh Subramanian. Free Radical Biology and Medicine, 2018,120, 62 – 71.
7) Comparison of the Automated Blood Culture System Vs Conventional Methods For Culture of Body Fluids. UmeshGami, Sofia Patel, Raji Pillai, Uma Chaturvedi, Prachi Gaddam and Susan Cherian. International Journal of Recent Scientific Research. Oct 2017,Vol 8,Issue 10, 20437 - 20441.

Department of Surgery

1. Kalbhande J, Budhkar A, Kuldeep V, Pimpalkar P. Pernicious Constipation in Adult Secondary to B12 Deficiency- A case report. Indian Journal of Applied Research [S. I.],v.8, n.2,apr.2018.
2. Budhkar A, Kalbhande J. Emergency meshplasty in obstructed inguinal hernia with septic shock and renal failure: A Case Report. Indian Journal of Applied Research [S. I.], v.8, n.7, July 2018.
3. Kalbhande J, Kuldeep V, Budhkar A, Aher N. Acute gastric dilatation because of binge eating in a mentally retarded child. JMSCR Jan 2018.
4. Kalbhande J, Budhkar A. A Rare Case of Spontaneous Reduction of Adult Appendicular Intussusception with Acute Appendicitis, 2019, JMSCR, 655-58.

STUDENTS OF BARC HOSPITAL PASSING FINAL DNB EXAM IN YEAR 2018

Dr Prina Bhavsar
Dept. of Ophthalmology

Dr Md Sehal Khan Abid
Dept. of Orthopaedics

Dr Anu Karthika
Dept. of Psychiatry

Dr Neelam Gote
Dept. of Paediatrics

Dr J. Shiva,
Dept. of Paediatrics

Dr Neha Singh
Dept. of Paediatrics

Dr Kowsalya P
Dept. of Anaesthesia

Dr Tista Ganguly
Dept. of Anaesthesia

Dr Yagyesh Shah
Dept. of Pathology

Dr Vengababu J.
Dept. of
Gen. Medicine

Dr Ankur Chaurasia
Dept. of ENT

Dr Pradip B.
Dept. of
Gen. Medicine

ACHIEVEMENTS

National Board of Examination recognized the outstanding contribution of Dr Alpa S. Amin to DNB Teaching Program in the specialty of Paediatrics and conferred her with the title of "ADJUNCT PROFESSOR" for a period of three years from 21/09/2018.

Dr Niranjan G. Nagpur, visiting Dermatologist, BARC Hospital, received "Teacher's Par Excellence Award" during Dermacon International 2019 by IADVL.

Dr Jyotsna Galinde, visiting Maxillofacial Surgeon, BARC Hospital, was awarded the "Lifetime Achievement Award" for her contribution to the field of Oral and Maxillofacial Surgery on the occasion of 6th MSC AOMSI organized by oral and maxillofacial surgery of Nashik on 1st February, 2019.

Shri Kiran Mahegoankar, UDC, posted at Dombivali Dispensary received the "DAE Meritorious Service Award" on 30th October 2018 (Founder's Day) for the year 2017 for his contribution to "Administration".

Dr Rohan Jadhav (Medicine Unit), Dr Yogesh Shejul (Medicine Unit), Dr Santosh Kumar (Paediatrics Unit) participated in the medical camp arranged on 8th Dec, 2018 for local populations of SMF Project area (Village Nayakanhatty, Challakere, Chitradurga, Karnataka).

COLS (Compression Only Life Support) program: Cardio-Pulmonary Resuscitation performed by lay-persons outside hospital setting.

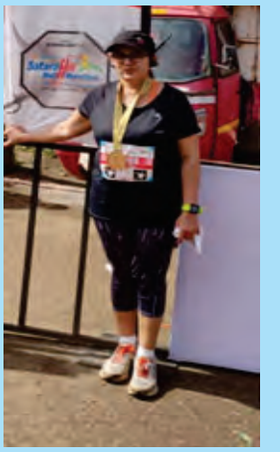
COLS program: All the consultants from department of anaesthesia and resident doctors Dr. Rashmi, Dr. Hemesh Shewale, Dr. Soujanya, Dr. Poorva Magarkar, technicians Mr. Pankaj Yadav, Mr. Krapit Shreni, Mr. Shashank Rai, Mr. Rajesh Kumar along with staff nurses Ms. Pratima Rodrigues, Ms. Sharayu Mahesh, Ms. Priya Palshikar and Ms. Reshma Malankar conducted this program in small teams during December 2018 - January 2019 and trained 884 students from secondary divisions of BARC school nos. 1 to 6.

BARC Hospital experts and Kharghar dispensary conducted a Breast Health Check-up Camp for CHSS beneficiaries on October 30th, 2018.

October being the Breast Cancer Awareness month, BARC hospital experts and Kharghar dispensary conducted a Breast Health Check-up Camp for CHSS beneficiaries on October 30, 2018. 104 patients attended the screening. The team comprised of Dr. Anita Gadgil, Dr. Santoshi Prabhu, Dr. Kanchan Bantwal, Dr. Anuradha Chakraborty, Dr. Debjani Pal, Dr. Vaishali Wadhe, Dr. Pratima Pimpalkar; Sisters Shobhana Anthony, Priya Palshikar, Shaila Khakse and Vaidehi Ugale. The team elaborated on different types of Breast lesions, benign or malignant, etiology, clinical features and treatment options. Stress was laid upon Self Breast Examination which was demonstrated and taught to the participants.

EXTRA-CURRICULAR ACHIEVEMENTS

Shri Akashdeep Huriwal (Dept. of Pharmacy) completed The Navy Half Marathon (21km) in November 2018 at Bandra Kurla Complex



Dr Pratibha Toal, Head Anaesthesia Unit, successfully completed "BNP Endurathon"25km Marathon, at Sanjay Gandhi National Park, Borivli (July 2018) and Satara Ultra Hill Marathon 21km (Sept. 2018

Dr Nalini Bhat stood second in the 10km veteran category of the Anushaktinagar Monsoon Marathon held in 2018

Shri Achyutananda Palei (Dept. of Pharmacy) completed Diploma in Yogic Education (Yoga Teachers Training Course) from Yoga Vidya Niketan Vashi in April 2018 and conducted class for male participants on International Yoga Day-2018, which was celebrated at BARC Hospital

Dr Urmila Peshoton from OYC Dispensary was the Official pacer (2.05 hours) at Tata Mumbai Marathon in January 2018. She got 3rd rank in the Navi Mumbai half Marathon in February 2018, got 2nd rank in Kanakia Monsoon challenge run (10km) at BKC in July 2018 & she also completed 21km in 2.07 hours at TMM in 2019

Dr Satish Mishra
Dept. of Surgery
BARC Hospital

SEGREGATION OF WASTE GENERATED IN HOSPITAL

CONTAMINATED WASTE (Infectious) *20% all hospital waste*

NON-CONTAMINATED WASTE (Non- infectious) 80% of all hospital waste

RED BIN

Recyclable biomedical waste. Autoclaved, shredded, recycled

ALL PLASTIC AND RUBBER; includes:

Gloves, IV bottles, IV sets, syringes without needles, vacutainers (with needles cut), drains, drain bags, catheters, urinary bags

Exceptions:

blood bags (along with blood transfusion sets) and antimicrobial culture plates

YELLOW BIN

Non-recyclable biomedical waste. Undergoes Incineration / Plasma Pyrolysis

ALL NON-PLASTIC, NON-RUBBER; includes:

Body parts / fluids like placenta, pus, debrided tissues, amputated parts.

Gauge/cotton plaster -cast.

Caps, face masks, disposable gowns

Discarded linen, mattress, beddings

Expired or discarded medicines

Empty bottles / ampules of cytotoxic (chemotherapy) drugs and vaccines

Chemicals- used or discarded disinfectants, formalin, and laboratory chemicals (ideally separate collection system leading to effluent treatment system)

Blood bags with residual or discarded blood and blood components (along with blood transfusion sets), culture plates, microbiological wastes & culture plates

CARDBOARD BOX WITH BLUE COLORED MARKING

Metallic body implants

Glassware- intact/broken; includes

Empty medicine vials and ampules

Exception:

Empty bottles & ampules of cytotoxic (chemotherapy) drugs – to be put in yellow bin

PUNTURE PROOF HARD PLASTIC BOX

ALL SHARPS; includes:

Needles, syringe with fixed needle Scalpel, blade, suture needle, K wires

Change the box when 2/3 full

BLACK BIN

Non-contaminated waste (kitchen waste, food waste, stationary)- goes to sanitary landfills

New rules:

Green bin – wet waste

Black bin- dry waste

In a survey at BARC Hospital, the most common confusion was regarding items to be disposed in red bin and yellow bin. Many healthcare professionals are of the knowledge that segregation between Red and Yellow bin depends on the nature of contamination, i. e. whether the waste is smeared with blood or pus.

Segregation does not depend on nature of contamination (pus/blood etc); rather it depends upon whether the waste is recyclable or not.

Chief Editor

Dr. Shrividya Chellam

Dept. of Anaesthesia & MOIC Casualty Unit, BARC Hospital
Anushaktinagar, Mumbai - 400 094.

Computer Design, Graphics & Layout by

Khan Shahid J.A.

SIRD, BARC, Trombay, Mumbai - 400 085.

Published by

Scientific Information Resource Division
Bhabha Atomic Research Centre, Trombay, Mumbai - 400 085.