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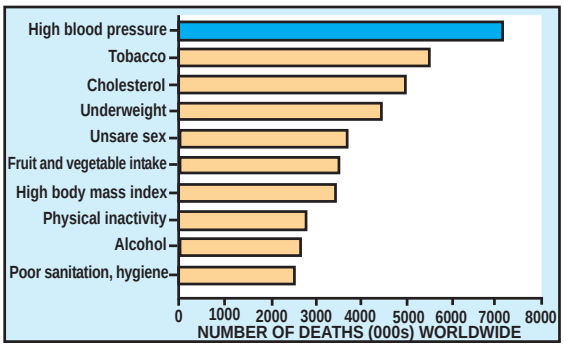
Editorial

Hypertension: A World-Wide Epidemic

Systemic hypertension is the most prevalent chronic medical condition worldwide. At the same time, it is not a benign condition, being touted as the number one attributable risk for death throughout the world as per a report of the World Health Organization in 2002. By that report, almost 7.1 million deaths per year was directly related to uncontrolled hypertension. The following table was published by WHO in 2002.

Table 1.



Hypertension is responsible for 69% of first MIs, 74% of cases of CHD, 77% of first strokes and 91% of cases of HF.

In a recent survey, worldwide obesity has doubled, while the western wealthy countries have reduced blood pressure and cholesterol levels. However, because of population growth and ageing, the actual number of people with uncontrolled hypertension rose from 600 million in 1980 to nearly 1 billion in 2008.

Blood pressure is getting controlled across the world but in India, it has risen : nearly 139 million Indians were suffering from high blood pressure at the end of 2008 which represents 14% of the global burden of uncontrolled hypertension.

As per WHO records, the average SBP went down by 2.7mm mercury among women globally, while increasing by 2.4 mm mercury in India while in men, it decreased by 2.3 mm mercury globally in the past three decades but in India it went up by 2.2 mm mercury.

In table 2, the important epidemiological surveys in India about the prevalence of hypertension has been depicted.

Table 2.

First Author	Year	Place	Age(yr.)	Sample Size	Prevalence (%)
Urban Populations					
Gupta R	1995	Jaipur	≥ 20	2212	30.9
Anand MP	2000	Mumbai	30-60	1662	34.0
Gupta R	2002	Jaipur	≥ 20	1123	33.4
Shanthirani CS	2003	Chennai	≥ 20	1262	21.1
Gupta PC	2004	Mumbai	≥ 35	88 653	47.9
Prabhakaran D	2005	Delhi	20-59	2935	30.0
Reddy KS	2006	National	20-69	19 973	27.2
Mohav N	2007	Chennai	≥ 20	2350	20.0

First Author	Year	Place	Age(yr.)	Sample Size	Prevalence (%)
Kaur P	2007	Chennai	18-69	2262	27.2
Yadav S	2008	Lucknow	≥ 30	1746	32.2
Rural Populations					
Gupta R	1994	Rajasthan	≥ 20	3148	16.9
Kusuma Y	2004	Andhra	≥ 20	1316	21.0
Hazarika NC	2004	Assam	≥ 30	3180	33.3
Krishnan A	2008	Haryana	15-64	2828	9.3
Todkar SS	2009	Maharashtra	≥ 20	1297	7.2
Bhardwaj R	2010	Himachal	≥ 18	1092	35.9
By Y	2010	Karnataka	≥ 18	1900	18.3
Kinra S	2010	National	20-69	1983	20.0

In table 3, the state of affairs regarding hypertension in the US has been shown.

Table 3.

National Health and Nutrition Examination Survey, Percent

	1999-2000	2009-2010
Awareness	70	81.9
Treatment	59	76.4
Control	34	53.3

In comparison, the Indian Heart Watch Study, the largest-ever risk factor study in India (2006-2010) found that awareness about hypertension among hypertensives was 57%, treatment received by 40% and control was achieved in only 25% cases.

Now let us check what the different countries are doing to control this menace. Health care spending in the U.S. dwarfs that found in any other industrialized country.In 2009, U.S. spending reached nearly $8,000 per capita. The other countries spent between one-third (Japan and New Zealand) and two-thirds (Switzerland and Norway) as much. US dedicated more than 17 percent of its gross domestic product (GDP) to health care compared with 12 percent or less in all other countries. But, still, for many U.S. households, health care has become increasingly unaffordable. In 2010, four of 10 adults went without care because of costs and the number of either uninsured or "underinsured" increased to more than 80 million. A 2007 survey in five states of US found that difficulty in paying medical bills contributed to 62 percent of all bankruptcies, up from 50 percent in 2001. In contrast, India's public spending on health as a proportion of GDP is among the lowest in the world as shown in table 4.

Table 4. Source: World Health Statistics, 2010, WHO

This leads to a vicious cycle of poor economies to incur the rich nations' disease and getting poorer in the process. Table 5 and table 6 depict this.

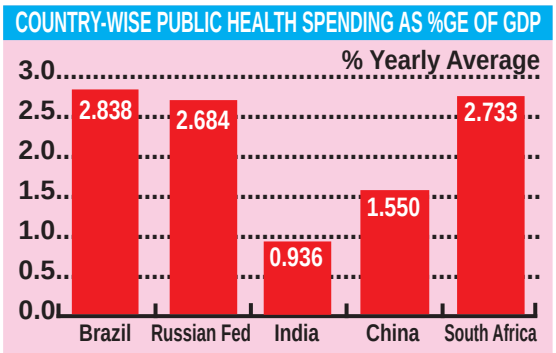


Table 5.

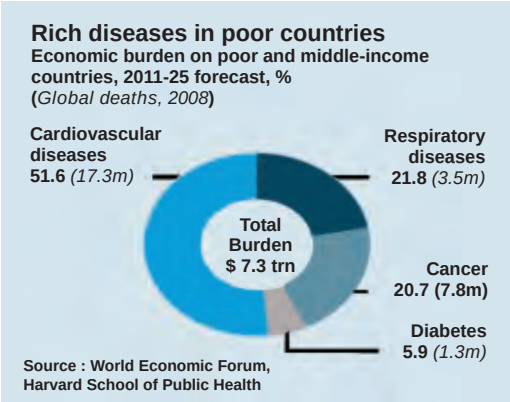
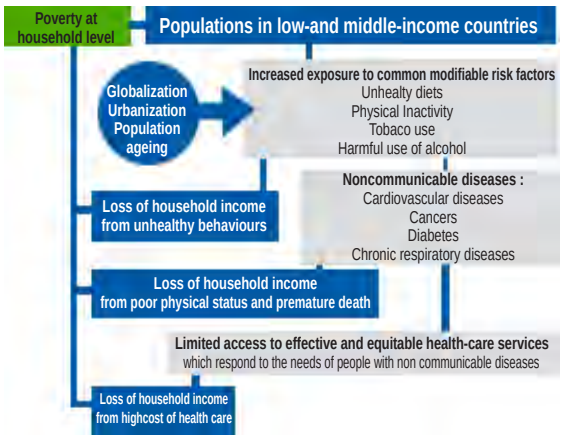


Table 6.



Public health approaches (e.g. reducing calories, saturated fat, and salt in processed foods and increasing community/school opportunities for physical activity) can achieve a downward shift in the distribution of a population's BP, thus potentially reducing morbidity, mortality, and the lifetime risk of an individual's becoming hypertensive.These public health approaches can provide an attractive opportunity to interrupt and prevent the continuing costly cycle of managing HTN and its complications, particularly in the poorer countries like India.

Further Reading:

1. National, regional, and global trends in systolic blood pressure since 1980: systematic analysis of health examination surveys and epidemiological studies with 786 country-years and 5.4 million participants. The Lancet, vol 377, p 568-577, Feb 2011.
2. Regional variations in cardiovascular risk factors in India: India heart watch. World J Cardiol, 2012 April 26, 4(4), 112-120.

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Importance of Meatoplasty in Open Cavity Mastoidectomy

Dr. Deepti Pandey[1], Dr. Soumitra Ghosh[2], Dr. Amitabha Roychoudhury[3], Dr. B. K. Roychaudhuri[4]

Abstract:

Objective: To highlight the importance of the size of meatoplasty in relation to the size of operative mastoid cavity in open cavity mastoidectomy.

Materials and methods: Prospective and retrospective observational study performed at the Dept of ENT & Head-Neck Surgery, Vivekananda Institute of Medical Sciences, Ramakrishna Mission Seva Pratishthan, Kolkata, over one year in 53 patients of unsafe otitis media undergoing open cavity mastoidectomy with conchomeatoplasty.

Results: All 53 patients underwent open cavity mastoidectomy with 11 patients undergoing small (<1.5cm), 29 patients, average (1.5cm) and 13 patients, large (>1.5cm) meatoplasty. All 29 patients undergoing an average meatoplasty and all 13 patients with large meatoplasty achieved dry ear by the second follow up at 6 weeks after surgery as against none of the 11 patients with small meatoplasty. All of the 53 patients had achieved a dry ear by 3 months of surgery.

Conclusion : Meatoplasty is an integral part of open cavity mastoidectomy. It's an important determinant of the ventilation, drainage, healing and persistently dry mastoid cavity. The size of meatoplasty should be as large as possible for a given size of post operative mastoid cavity.

Keywords:

Open cavity mastoidectomy, meatoplasty, conchomeatoplasty, unsafe chronic otitis media

Introduction:

The global burden of illness from chronic suppurative otitis media (CSOM) involves 65–330 million individuals with discharging ears[1].

CSOM accounts for 28000 deaths and a disease burden of over 2 million Disability Adjusted Life Years (DALYs). Over 90% of the burden is borne by countries in the South-east Asia and Western Pacific regions, Africa, and several ethnic minorities in the Pacific region. In India, the prevalence of CSOM is above 4%.

CSOM may be of safe and unsafe type. Unsafe CSOM requires mastoid surgey with or without tympanoplasty. Patients with extensive disease require canal wall down or open cavity or modified radical mastoidectomy (MRM), which involves the creation of a mastoid cavity with exteriorization of the cavity into the external auditory canal. The scutum or lateral wall of the epitympanum is removed along with removal of the malleus and incus. The posterior bony canal wall is lowered to the level of the facial nerve. An adequate meatoplasty is a prerequisite with the canal wall down mastoidectomy to facilitate egress of desqaumated epithelial debris and provide access to the mastoid bowl. It is an

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operative technique to widen the lateral cartilaginous part of the external auditory canal. Cartilage displaced anteriorly from the *cavum conchae* of the pinna, as well as bulky underlying soft tissue can cause narrowing of the lateral part of the ear canal. The aim of a meatoplasty is to enlarge the lumen of the entrance of the ear canal by removing the obstructing cartilage of *cavum conchae* as well as the underlying soft tissue. Larger the size of mastoid cavity, there should be a proportionately large and adequate meatoplasty.

The V/S ('V'- volume of air circulating through the cavity, 'S'- surface area of the cavity) ratio provides the relation between the size of mastoid cavity and that of meatoplasty[2]. A conchomeatoplasty should be so performed as to achieve adequate size of meatus in accordance with the size of mastoid cavity to provide an adequate surface volume ratio for aeration, epithelial stability and good post operative visualization of the cavity[3].

Aims & Objectives:

The aim of the following study was:

1) To evaluate the size of meatoplasty with their follow up results in patients of canal wall down mastoidectomy (CWD) and tympanoplasty.

2) To recommend the size of meatoplasty for adequate aeration and drainage of mastoid cavity.

Material & Methods:

The following study was conducted at the Department of ENT and Head-neck Surgery, Vivekananda Institute of Medical Sciences, Ramakrishna Mission Seva Pratishthan, Kolkata, for a period of 1 year. The course of change in meatoplasty size and length of time taken to achieve dry cavity were the key assessment measures of the outcome.

Fifty-three patients of unsafe CSOM, attending the Out Patient Department, meeting the eligibility criteria and subsequently, undergoing CWD mastoidectomy and tympanoplasty with meatoplasty, were included in the study. This study is a prospective and retrospective observational study. All the patients underwent detailed medical history and clinical examination. Each patient also underwent otoscopic examination, examination under microscope (EUM), pure tone audiometry, speech audiometry, tympanometry and X-ray of both mastoids. HRCT of temporal bones was done in patients with complicated CSOM and in revision cases. Apart from ear, the nose and throat were also examined in detail by cold spatula, anterior and posterior rhinoscopy, tongue depressor and indirect laryngoscopy to rule out any concomitant abnormality and source of infection.

Systemic examination of the patient was also performed with regards to the central nervous system, cardiovascular system, chest and abdomen to rule out systemic illnesses. Informed consent of all the patients was obtained. Only the patients willing to attend follow up visits as advised were included in the study.

Inclusion criteria:

All cases of unsafe CSOM undergoing canal wall down mastoidectomy, tympanoplasty and meatoplasty were included in the study.

Exclusion criteria:

Patients having intracranial complications and children below 12 years were excluded from the study.

Surgical Procedure:

CWD mastoidectomy with tympanoplasty along with adequate meatoplasty was performed through post aural approach under general anaesthesia in all the cases following standard surgical procedures. All the cases were operated by a single senior surgeon of the unit.

Type III tympanoplasty with temporalis fascia along with or without augmentation was performed to achieve a closed middle ear space after canal wall down mastoidectomy.

Conchomeatoplasty techniques include transposed skin flaps, removal of adequate conchal cartilage and cartilage from floor of the ear canal. Meatal skin flaps are sutured covering the cartilage remnants. In this study the conchomeatoplasty was fashioned following Koerner's technique[8]. In this method a tongue shaped laterally based flap of posterior half of canal wall skin was elevated at the beginning of procedure to preserve the canal skin. A wide strip of conchal cartilage was resected to widen the meatoplasty. The cartilage of helix and that of tragus were preserved. At the end of the surgery, the flap was sutured to the periosteum posteriorly (Fig. 1).

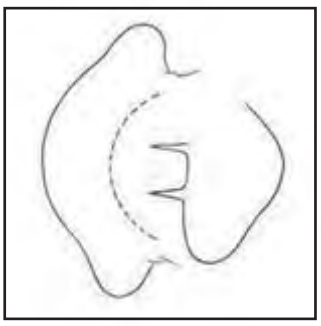

Fig. 1: Koerner's Flap

The size of meatoplasty was measured by a measuring gauze at the end of the surgery and afterwards, during the subsequent follow up visits. The diameter of meatoplasty was measured from the medial end of the tragal cartilage horizontally backwards touching the margin of conchal bowl.

The volume of the mastoid cavity was measured by instilling saline into the mastoid bowl from an insulin syringe and measuring the volume of saline required to fill the bowl at the end of the drill work during surgery and at subsequent follow up visits.

Post operative treatment and follow up:

Each patient received systemic antibiotics during the surgery and afterwards for up to 24 hrs and later a ten day course of oral antibiotics and a 2 week course of antihistaminics. Patients were discharged on the 7th post operative day, when the ear pack was removed along with removal of post-aural stitches. They were appointed for follow up at two weeks, one month, three months and six months after operation for dressing and reassessment.

At each follow up visit, detailed general and otologic examination was performed. Particular attention was focused on the period to achieve dry ear, infections, hearing ability, instances of residual cholesteatoma and mastoid cavity status etc. A colored photograph of the mastoid cavity and meatoplasty opening was taken with 4 mm otoendoscope and camera in each visit.

Statistical analysis:

One way ANOVA and students' paired 't' test was used for analysis of the data.

Observations & Results:

Fifty-three patients, 27 males and 26 females in the age range of 12 to 50 years were included in the study (Table 1).

Male	Female	Age range (years)
27	26	12-50

Table 1: Composition of the Study Group (n=53

Parameters	Size of Meatoplasty		
No of Patients (n=53)	Small (< 1.5 cm)	Average (1.5 cm)	Large (> 1.5 cm)
	11	29	13
Mean Time for Attaining Dry Ear (weeks)	7.5	5.2	4.5

Table 2: Surgical Outcome in Terms of the Size of the Meatoplasty

All 53 patients underwent canal wall down mastoidectomy with 11 patients undergoing small (<1.5cm), 29 patients, average (1.5cm) and 13 patients, large (>1.5cm) meatoplasty. All 13 patients with large meatoplsty achieved dry ear by 4.5 weeks after surgery. All 29 patients undergoing an average meatoplasty achieved dry ear by 5.2 weeks after surgery. All the 11 patients with small meatoplasty achieved dry ear by 7.5 weeks after surgery (Table 2).

Otoendoscopic pictures of the meatoplasty and the cavity at follow up visits were recorded.

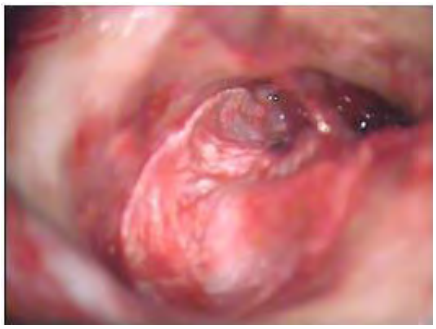

Fig. 2: Mastoid Cavity at the End of Surgery

Fig 2 Shows the mastoid cavity with temporalis fascia graft placement at the end of the surgery.

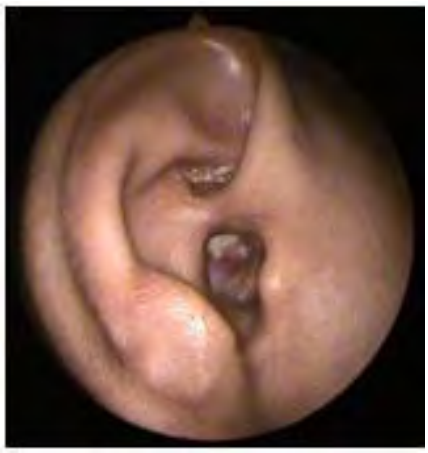

Fig. 3: Meatoplasty at 6 Week Post Operative Follow-up

Fig. 3 Shows the size of meatoplasty (1.6 cm) at the 6 week post operative follow up.

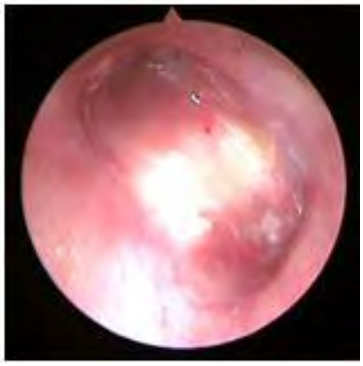

Fig. 4: Cavity at 6 Week Post Operative Follow-up

Fig. 4 Shows the cavity status at the 6 week post operative follow up. Graft is taken up well and the cavity is healthy.

Discussion:

CWD mastoidectomy with tympanoplasty is our choice of surgery in extensive unsafe variety of CSOM. The first three priorities in the surgery of unsafe type of CSOM are :

1. the elimination of progressive disease to produce a safe and dry ear
2. modification of the anatomy of the tympano-mastoid compartment to prevent recurrent disease
3. reconstruction of hearing mechanism

An adequate and proportionate conchomeatoplasty size in relation to the mastoid cavity size is essential to achieve a dry cavity in majority of the cases. The four most important factors which influence the healing of a mastoid cavity are:

i. incomplete exenteration of infected mastoid air cells
ii. inadequate lowering of the facial ridge
iii. an inadequate meatoplasty
iv. an open middle ear cavity

After meticulous canal wall down mastoidectomy, it is essential to fashion a sufficiently large conchomeatoplasty[2]. On the other hand, an extremely wide entrance of the outer ear canal is cosmetically unpleasant and air currents in the cavity can cause vertigo[7].

Before meatoplasty was developed by Stacke (1893) and Schwartze (1893), a postaural fistula was often created intentionally after mastoidectomy to facilitate management of the unsafe and diseased ear. This procedure is no more in practice, nowadays.

Portmann M & Portmann D[2] opined that, with time, the depths of the cavity become lined by squamous epithelium, which must be adequately aerated if it is to remain biologically stable, otherwise there is risk of recurrent cholesteatoma. This is the law of V/S ratio, where 'V' represents the volume of circulating air arising from outside, and 'S' represents the surface area of the cavity which is essential to be aerated. If 'S' is very large, then 'V' must also be large. The authors use three flap meatoconchoplasty (one conchal flap and two meatal flaps) with removal of cartilage to a greater or lesser extent. We, too, have followed the same philosophy except the 3 flap technique of conchomeatoplasty.

Parisier SC, Levenson MJ & Hanson MB[4] observed, that an adequately large meatus measures about 1.5 cm in diameter and should easily accommodate the surgeon's index finger. We have obtained the good result with size of the conchomeatoplasty of 1.5 cm or more.

Wormald PJ & van Hasselt CA[5] performed temporal bone dissections to design a surgical technique to minimize the known causes of a discharging cavity. They assessed the mean size of the cavity resulting from the new surgical

technique to be 2.6 ml. and the mean largest diameter of the meatus 10.1 mm. In our study, the mean size of the cavity after surgery was 4.37 ml. and mean largest diameter of the meatus at surgery was 15.96 mm.

Yetiser S, Kertmen M, Ozkaptan Y, et al[6] noted that the diameter of meatoplasty ranged from 1.7 to 2.6 cm in the early post operative period. They observed a decrease of 4 to 6 mm in the diameter within one year. In the present study, the diameter of meatoplasty ranged from 1.3 to 1.8 cm. We observed a decrease of 2 to 5 mm in the diameter within 3 months of the surgery.

Awad Z, Ranganathan B & Patel N[9] used digital photography to assess and record the meatoplasty opening. Digital photography was used to measure the surface area of widened meatus and comparison made with the preoperative and the contralateral meati. A measurable increase in meatal size was observed with repeatable results. We used otoendoscopy to record our observations regarding the size of conchomeatoplasty and the status of the operative mastoid cavity immediately after surgery and at each follow-up visit. The mean contraction in the size of meatoplasty was 3.1 mm (19.32%) and the mean reduction in the volume of mastoid cavity was 0.81 ml (18.64%). The mean time period for attainment of dry cavity was 5.73 weeks.

The study emphasizes the importance of an adequately performed conchomeatoplasty as the key factor in achieving a fast dry ear after a properly performed CWD mastoidectomy operation. The recommended size of concho-meatoplasty should be more than 1.5 cm on an average to achieve acceptable results.

Summary:

The retrospective and prospective observational study was conducted over one year at the Dept of ENT & Head and Neck Surgery, Vivekananda Institute of Medical Sciences, Ramakrishna Mission Seva Pratishthan, Kolkata.

Fifty-three patients of unsafe CSOM, in the age range of 12 to 50 years were included in the study.

Patients under the age of 12 years and those with intracranial complications were excluded from the study.

Informed consent of all the patients was obtained. All patients underwent CWD Mastoidectomy with tympanoplasty and conchomeatoplasty by Koerner's technique for extensive unsafe CSOM.

The parameters studied were

1. Size of conchomeatoplasty
2. Volume of mastoid cavity
3. Onset of dry cavity after surgery

The mean size of conchomeatoplasty in our study group was 15.96 mm. The mean decrease in the diameter of the conchomeatoplasty at the 3 month post operative follow up was 3.1 mm (19.32%). Mean volume of the mastoid cavity at the end of the surgery was 4.37 ml. The mean reduction in the volume at the end of 3 months after surgery was 0.81 ml (18.64%). The mean time period for attainment of dry cavity after surgery was 5.73 weeks. The mean time period for attainment of dry cavity after surgery for small conchomeatoplasty group was 7.5 weeks, for average conchomeatoplasty group 5.2 weeks and for large conchomeatoplasty was 4.5 weeks post surgery.

An enlarged external auditory meatus and a

shrinking mastoid cavity were clearly appreciated through the serial otoendoscopic pictures.

Conclusion:

Conchomeatoplasty is an essential part of the canal wall down mastoidectomy. It provides a channel for the epithelialization of the raw post operative mastoid cavity, drainage of the secretions, aeration of the entire cavity and post operative care. Evidently, it should be large enough to cater to the needs of the post operative cavity which has a much larger volume than the space that is normally exposed to the natural meatus.

The healing of the cavity in terms of reduction in the volume had no correlation with the anatomical outcome profile.

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Role of Systemic Hypertension in Ocular Disease

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Hypertension is a common systemic condition affecting approximately one billion, and ranked as the fourth largest mortality risk factor in the world. Hypertension confers cardiovascular risk by causing target-organ damage that includes retinopathy in addition to heart disease, stroke, renal insufficiency and peripheral vascular disease. As per previous[1] and current[2] reports of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC), retinopathy is considered as one of the several markers of target organ damage in hypertension. On the basis of the JNC criteria, the presence of retinopathy may be an indication for initiating antihypertensive treatment, even in people with stage 1 hypertension (blood pressure, 140 to 159/90 to 99 mm Hg) who have no other evidence of target-organ damage.

Studies have shown that the effects of systemic hypertension on the retinal, optic nerve head and choroidal circulation produce three distinct and independent manifestations: (i) Hypertensive retinopathy, (ii) Hypertensive optic neuropathy, and (iii) Hypertensive choroidopathy

Aetiology

Race: Afro-Caribbeans have a higher prevalence of hypertension and hypertensive retinopathy than Europeans.

Sex: The prevalence of hypertensive retinopathy is higher in women than in men.[3]

Smoking: Though the true association between smoking, hypertension and target-organ damage is difficult to assess due to the confounding effects of other lifestyle and socio-economic factors, studies focusing on malignant hypertension have shown a strong association between smoking and grade IV hypertensive retinopathy.[4]

Genetic factors: Certain specific genotypes are linked with an increased risk of hypertensive retinopathy. The D (deletion) allele of the angiotensin converting enzyme (ACE) gene is an independent risk factor for the development of end-organ damage in patients with essential hypertension and entails a 2.4-fold higher chance of retinopathy.[5] The hypertensive individuals who carry the apoepsilon 4 allele of apolipoprotein E gene or are homozygous carriers of a point mutation are at a significantly higher risk of retinopathy.[6,7]

Renal status: In patients with essential hypertension, persistent microalbuminuria is a marker of early end-organ damage including retinopathy.[8,9]

Cardiac status: Left ventricular hypertrophy and retinal vascular disease appear early in the course of blood pressure elevation and both changes develop in parallel.[10] The severity of hypertensive retinopathy and the renal involvement are more severe in patients with concentric rather than eccentric left ventricular hypertrophy.[11]

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Plasma leptin: A critical plasma leptin level for the development of retinopathy has been identified (10.2 ng. ml).[12] Leptin, an angiogenesis factor, was significantly higher in patients with grade 1 retinopathy even after correction for body mass index.[12] The elevated concentrations possibly relate to the damage of the vascular endothelium by high blood pressure.

Salt sensitivity: Hypertensive retinopathy is more common in salt-sensitive hypertension than in salt-resistant hypertension.[13] Secondary hypertension: Renal hypertension is associated with more severe retinopathy than essential hypertension. Atherosclerotic reno-vascular disease is associated with high-grade retinopathy.[14] Severe end-organ damage including grade III or IV retinopathy is common in hypertension secondary to pheochromocytoma.[15,16]

Refractory hypertension : Refractory hypertension is associated with advanced retinal involvement (grades II and III retinopathy).[17]

Pathophysiology

Hypertensive Retnopathy:

Retinal circulation undergoes a series of retinal micro vascular changes evident as clinical signs in hypertension, either directly by elevated blood pressure or indirectly via vasoactive substances (angiotensin II, endothelin-1 and decreased basal nitric oxide activity). The underlying pathophysiology of these signs can be divided into stages.[18]

1. Vasoconstrictive stage : there is vasospasm and an increase in retinal arteriolar tone owing to local autoregulatory mechanisms clinically evident as generalized narrowing of the retinal arterioles.

2. Sclerotic stage: persistently elevated blood pressure leads to intimal thickening, hyperplasia of the media wall, and hyaline degeneration in the subsequent, sclerotic stage. This stage corresponds to more severe generalized and focal areas of arteriolar narrowing, changes in the arteriolar and venular junctions (i.e., arteriovenous nicking or nipping [Figure A]), and alterations in the arteriolar light reflex due to opacification (i.e., widening and accentuation of the central light reflex described as “silver wiring” or “copper wiring” [Figure B]).

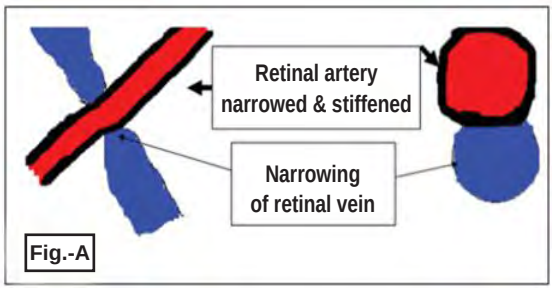

Fig.-A

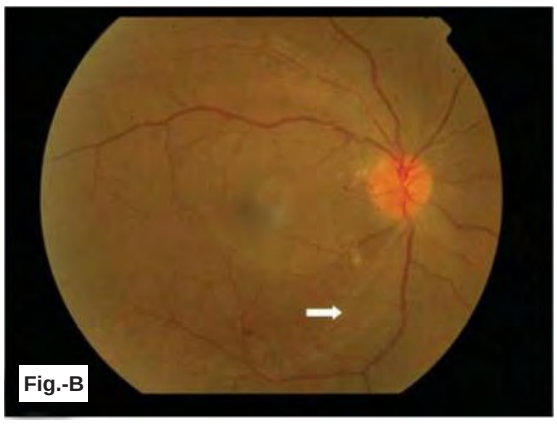
Fig.-B

3. Exudative stage : in which there is disruption of the blood–retina barrier, necrosis of the smooth muscles and endothelial cells, exudation of blood (haemorrhages), lipids (hard exudates), and retinal ischemia (cotton-wool spots). (Figure C).

Fig.-C

Hypertensive Optic Neuropathy:

When hypertension becomes very severe, intracranial pressure may become elevated with subsequent effects on the optic nerve manifested as optic nerve ischemia and optic disc swelling (papilledema) referred to as malignant hypertension or hypertensive optic neuropathy (Figure D).

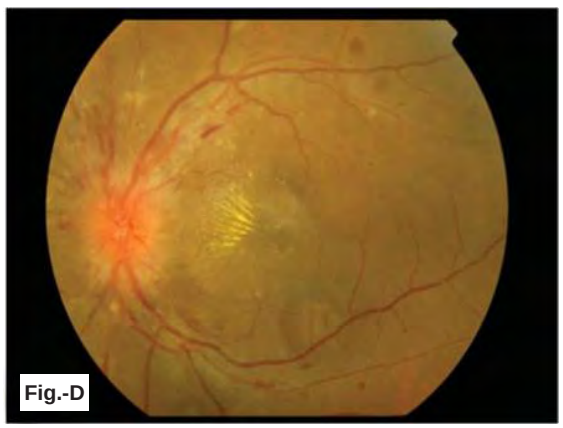

Fig.-D

Hypertensive choroidopathy:

The choroidal arterioles undergo fibrinoid necrosis in severe hypertension leading to the infarction of segments of the choriocapillaris, wherein patches of overlying retinal pigment epithelium (RPE) may appear yellow (Elschnig's spots) (Figure E), which may become hyper pigmented with a margin of hypo pigmentation as they resolve.

Less commonly, linear RPE hyperplasia may develop (Figure F) over infarcted choroidal arterioles (Siegrist's streaks) and localized bullous neurosensory or RPE detachments may be observed. Generalised retinal–arteriolar narrowing and arteriovenous nipping are markers of vascular damage from chronic hypertension. In contrast, other signs (focal arteriolar narrowing, retinal hemorrhages, micro-aneurysms, and cotton-wool spots) were related to current but not previous blood-pressure levels and may therefore be more indicative of the severity of recent hypertension.[18]

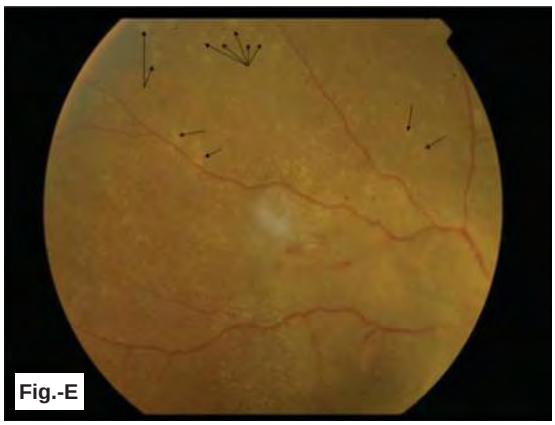

Fig.-E

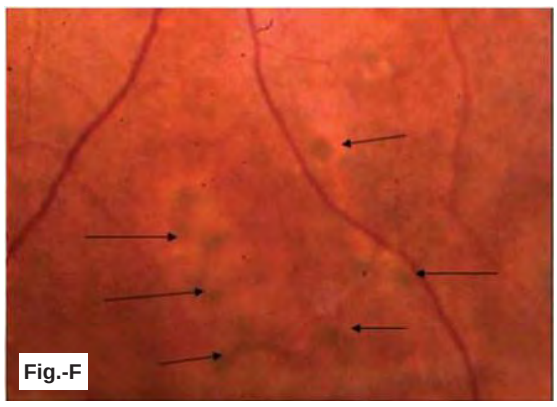

Fig.-F

Clinical Manifestations

Hypertensive Retinopathy:

The fundus features commonly described in hypertensive retinopathy include focal and generalised arteriolar narrowing, microaneurysms, intra-retinal haemorrhages, cotton-wool spots, hard exudates and optic disc swelling. Changes of secondary arteriolosclerosis are arteriovenous nipping, changes in the arteriolar light reflex and arteriolar sheathing and occlusion. Abnormal vascular permeability produces flame-shaped haemorrhages, retinal oedema and lipid exudates. The deeper blot haemorrhages appear with progression of hypertension and indicate worsening ischaemia. Deposition of lipid around the fovea may lead to the formation of a macular star. Complications of severe untreated hypertension include haemorrhagic detachment of the internal limiting membrane of the retina, subhyaloid and vitreous haemorrhage. Arteriovenous crossing changes include banking of the venule distal to the crossing (Bonnet's sign), nipping of the blood column (Gunn's sign) and displacement of the venule at right angles to the arteriole (Salus's sign). The increased arteriolar light reflex correlates with hyalinisation of the arteriolar wall with copper wire or silver wire appearance of the light reflex.

Clinical classifications:

- Keith, Wagener and Barker hypertensive retinopathy classification (Talble 1)
- Scheie's classification (Table 2)
- Wong and Mitchel three grade classification (Table 3)

Table 1

Keith, Wagener and Barker hypertensive retinopathy classification	
Grade	**Features**
I	Mild generalized retinal arteriolar narrowing
II	Definite focal narrowing, arterivenous nipping and irregular arteriole
III	1 & 2 along with retinal haemorrhage, hard exudaate, cotton wool spots with or without macular star. Early disc swelling starting nasally
IV	Severe grade 3 with papilledema

Table 2

Scheie's Classification	
Grade	**Features**
0	No changes
1	Barely detectable arteriolar narrowing
2	Obvious arteriolar narrowing with focal irregularities plus light reflex changes
3	Grade 2 plus copper wiring & retinal haemorrhages/exudates
4	Grade 3 plus silver wiring & papilledema

Table 3

Wong and Mitchell three grade classification		
Grade	**Retinal Changes**	**Systemic Association**
Mild	Generalized or focal arteriolar narrowing, arterivenous nicking, opacification (copper wiring) of arteriolar wall or a combination of these sign	Modest association with risk of clinical stroke, subclinical stroke, cardiovasucular mortality
Moderate	Haemorrhages (blot, dot or flame shaped), cotton wool spots, hard exudates, micro aneurysms or a combination of these signs	Strong association with risk of clinical stroke, subclinical stroke, cognitive decline and mortality from cardiovascular causes
Malignant	Signs of moderate retinopathy plus swelling of optic disc	Strong association with renal failure and mortality

Hypertensive optic neuropathy:

Papilloedema or bilateral disc swelling represents grade IV hypertensive retinopathy in the Keith Wagener-Barker classification.[19] It was considered an essential criterion of malignant hypertension.[19] Traditionally papilloedema in hypertension has been considered to be a poor prognostic sign for survival.[19] The WHO criterion for malignant hypertension is severe hypertension with bilateral retinal haemorrhages and exudates.[20] Conversely, it has been suggested that in the presence of elevated systemic blood pressure, isolated papilloedema without retinopathy could represent a variant of malignant hypertension.[21] Other causes of papilloedema like space-occupying lesions and benign intracranial hypertension, however, need to be excluded.[21] The pathogenesis of papilloedema secondary to systemic hypertension is controversial and the several theories include:[22] (i) ischaemia, (ii) raised intracranial pressure, and (iii) as a part of hypertensive retinopathy encephalopathy. Papilloedema secondary to hypertension usually resolved following good control of blood pressure although some developed disc pallor.[23] Longstanding chronic hypertension may result in retinal nerve fibre loss[24] and experimental animal studies[25,26] found ischaemia to be the possible underlying cause.

Hypertensive Choroidopathy:

Choroidal lesions secondary to elevated blood pressure are less well recognised than retinopathy in the current literature. The underlying mechanism relates to choroidal ischaemia and its effects on the retinal pigment epithelium and retina.[27]

The more commonly described features of hypertensive choroidopathy are choroidal vascular sclerosis, Elschnig spots representing focal areas of degenerative retinal pigment epithelium and the diffuse patchy atrophic retinal pigment epithelial degeneration of chronic hypertensions.[28,29] Siegrist's streaks, linear retinal pigment epithelial changes, are the

sequelae of acute hypertensive choroidopathy and generally indicative of a poor prognosis.[30] Serous retinal detachment, a prominent feature in the animal model, is less common in clinical settings.[31,32]

Management

Based on Wong and Mitchell Classification[18]

- Mild hypertensive retinopathy: Routine care, closer blood pressure monitoring, better control of hypertension.
- Moderate hypertensive retinopathy: May need physician referral, diabetes, hyperlipidemia should be excluded, possible indication for hypertensive treatment & other cardiovascular risk factors.
- Severe hypertensive retinopathy: Urgent step wise control of BP as sudden lowering may lead to stroke.With adequate treatment resolution of signs may occur over a period of one year.

Ocular Diseases Secondary to Systemic Hypertension

Systemic hypertension has been associated with a large number of ophthalmic conditions. They are described briefly in the context of the current literature.

Retinal Vein Occlusion:

Retinal vein occlusions occur most commonly at the level of the lamina cribrosa (central or hemispheric retinal vein occlusion) or at an arteriovenous crossing (branch retinal vein occlusion). They commonly present with sudden painless visual loss or a field defect. The signs in the acute phase may include engorged tortuous retinal veins, superficial flame retinal haemorrhages, retinal oedema, cotton-wool spots and disc swelling[33]. Central retinal vein occlusions are clinically divided into nonischaemic and ischaemic.[33] In the presence of persisting retinal nonperfusion, neovascularisation may develop on the disc, retina or iris. Treatment with laser photocoa-gulation is indicated in these cases.[33,34] If untreated, sight-threatening complications such as vitreous haemorrhage and secondary rubeotic glaucoma may develop.[33] Several studies have demonstrated that systemic hypertension is associated with an increased risk of developing central, branch and hemi-central retinal vein occlusions.[35,36,37] Systemic hypertension was also found to be associated with branch macular vein occlusions in younger patients.[38] A recent study[38] found a significantly higher prevalence of arterial hypertension in branch retinal vein occlusion compared with central and hemi-central retinal vein occlusion. Arterial hypertension was also more likely to be present in ischaemic rather than nonischaemic central retinal vein occlusion.[39]

Retinal Arterial Macroaneurysm:

Retinal arterial macroaneurysms are acquired focal aneurysmal dilatations of the retinal arterioles, usually occurring in the first three orders of the arteriolar tree. They are commonly seen in hypertensive retinopathy giving rise to star-shaped exudation and sometimes complicated by pre-retinal or intravitreal haemorrhage.[40,41] Spontaneous resolution with thrombosis within the macroaneurysm may occur[40]. In the presence of active leakage laser photocoagulation may be applied directly to the macroaneurysm.[40]

Non-Arteritic Anterior Ischaemic Optic Neuropathy:

Essential arterial hypertension is significantly associated with non-arteritic anterior ischaemic

optic neuropathy.[42] Clinically, this presents with uni-lateral painless disc swelling followed by disc pallor and irreversible visual loss.[43] Symptoms may include a classical altitudinal field defect or a central scotoma.[43] The exact mechanism may be chronic hypoperfusion of the small end-arterial optic nerve head vessels caused by over-treated hypertension or abnormal vascular autoregulation.[44] Recent studies have suggested that ocular or optic nerve head ischaemic disorders may be due to a combination of systemic arterial hypertension and hypotension.[45,46,47]

Cranial Nerve Palsies:

Cranial nerve palsies, including third, fourth, sixth and seventh cranial nerve palsies, are commonly found secondary to systemic hypertension.[48,49,50,51] They are often isolated events causing acute symptomatic diplopia.[52] They usually resolve spontaneously within three months with strict BP control.[52] Neuroimaging may not be indicated in the absence of other neurological signs or pupillary abnormalities.[52]

Diabetic Retinopathy:

Diabetic retinopathy is a microvascular disorder in which endothelial cell malfunction and impaired regulation of retinal perfusion occur owing to chronic glucotoxicity.[53]

UKPDS 50 demonstrated that the incidence of diabetic retinopathy was strongly associated with higher blood pressure.[54] UKPDS, EUCLID and other studies have shown retardation in the progression of diabetic retinopathy with improved control of blood pressure.[55] The HOPE study results showed that it was both safe and beneficial to lower BP already within the 'normal' range with angiotensin converting enzyme inhibitors in patients with known vascular risk factors including left ventricular dysfunction, hypertension or diabetic microalbuminuria.

Retinal arterial occlusion, Age related mascular degeneration,Glaucoma are other diseases found to be associated with systemic hypertension.

Conclusion

Profound, often silent, multisystemic effects of hypertension makes it a major health issue worldwide. Hypertensive microvasculature changes in eye are of predictive and prognostic value in the management of systemic complications secondary to hypertension, including diabetes, cardiovascular, cerebro-vascular and other systemic vascular diseases. Although current recommendations for management of hypertension do not mandate routine ophthalmoscopic examination, newer screening tools such as digital imaging and computer analysis may permit an early and consistent detection that may be important in identifying and managing modifiable risk factors and provide prognostic information for cardiovascular risk and disease progression. However in hypertensive emergency retinal examination may be of diagnostic and therapeutic help.

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Diet in Hypertension

Mrs. Soma Kundu

Hypertension is a common public health problem in developed countries. Untreated hypertension leads to many diseases like cardiac diseases, renal disease. It is often called a "silent killer" because people with hypertension can be asymptomatic for years. Prevention and management decrease the incidence of hypertension. Lifestyle modifications has a prominent role for prevention and management of hypertension.

Dietary Factors:

Changing four modifiable factors has documented efficacy in the primary prevention and control of hypertension. These four factors are overweight, excessive salt consumption, physical inactivity and alchohol consumption.

Excess Body Weight:

Body weight is a determinant of blood pressure in most ethnic groups at all ages. The risk of developing elevated blood pressure is two to six times higher in overweight than in normal weight persons. Clinical trials have documented that weight loss lower blood pressure, one meta analysis suggested that mean systolic and diastolic blood pressure reductions from an average weight loss of 5.1 kg were 4.4 and 3.6 mm hg respectively[1]. Life style intervention trials have uniformly achived short term weight loss, primarily through a reduction in total calorie intake. In several instances substantial weight loss have been sustained over greater than equals to 3 years. Weight reduction, ideally attaining a BMI less than 25 kg/m^2 is an effective approach to prevent and treat hypertension.[2]

Excessive Consumption of Salt Intake:

Epidemiological studies, clinical trials and meta-analysis, all suggest that high dietary salt intake rises blood pressure. The results of DASH - sodium trials suggested that decreasing sodium intake by 40 mmol/day caused a greater lowering of blood pressure[3]. The Inter Salt Study also showed systolic blood pressure was significantly related to dietary sodium intake[4]. So a reduction in salt intake not more than 6g daily (2400 mg na) is recommended to prevent hypertension.

Alcohol Consumption:

Observation studies and clinical trails have documentation a direct close dependent relationship between alcohol intake and blood pressure, particularly if the intake of alcohol increases above 2 drinks/day[5]. Moderate alcohol intake is an effective approach to lower blood pressure.

Other Dietary Factor:

Potassium:

High potassium intake is associated with reduced blood pressure. Meta - analysis have documented a significant inverse relationship between potassium and blood pressure in nonhypertensive and hypertensive individuals[6]. A high potassium intake can be achived through diet rather than supplement because accompanied by a variety of other nutrients. In the DASH trial the increased fruits and vegetables consumption may help to reduce elevated or high blood pressure[3]. High potassium intake from foods poses no risk in healthy population with normal kidney function.

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Lifestyle Modifications:

Life style medications are definitive therapy for all persons with hypertension. These should be tried before drug therapy to be initiated. If life style modifications fail to correct the blood pressure still it will help to increase the efficacy of pharmacologic agents.

Weight Management:

The effectiveness of weight reduction has been well documented in hypertensive patients who are over weight (BMI greater than 29.9) should need weight reduction through hypocaloric diet and exercise. The energy intake should be 20-25 kcal/kg of ideal body weight/day. This diet leads to loss of body weight from 0.5 to 1 kg/week. This modest loss will not only lower blood pressure but also normalizes blood lipids and glucose. Maintanance of body weight is very critical. So regular exercise and use of a food dairy are effective for weight maintenance.

Salt Restriction:

Modarate salt restriction (6 gm salt or 2400 mg Na) is recommended for treatment of hypertension. This can be achieved by cooking with as little salt as possible, avoiding highly salted and processed foods.

Alcohol:

Alcohol intake should be limited to less than two drinks /day (24 oz beer, 10 oz wine or 2 oz 100-proof whiskey) in men and one drinks/day in women.

Exercise:

Exercise is strongly associated with success in weight reduction and weight maintenance. Moderate physical activity that is 30 to 45 minutes of brisk walking on most days of the week is recommended as an adjunct therapy in hypertension.

Diet Chart of A Hypertensive Patient:

Diet chart is individualized. It varies from person to person according to his/her age, weight, height, sex and activity. Following diet chart is just a sample menu of a hypertensive patient and this is not applicable to all.

Sample Menu:

Early Morning: Tea 1 cup with milk (20 ml) and without sugar, Marie biscuits-2 pcs (10 gm)

Break Fast: Brown Bread - 2 Slices(50 gm) or Corn Flex - 3 Table Spoon (50 gm) + Milk (double toned) - 200 ml.

Mid Morning: Mixed fruit salad -1 plate (250 gm) (from water melon, ripe papaya, guava and cucumber).

Lunch: Rice -2 cups, (from 50 gm of uncooked weight) or Chapatti -2 pcs (from 50gm of atta), Pulses-1 cup (from 25 gms of uncooked weight), Fish – 1 pcs (50 gms) or Curd -1 bowl (from 200 ml of double toned milk), Plain Salad- 1 plate.

Tea Time: Tea 1 cup with out sugar + Milk-20 ml.

Snacks: Apple-one (150 gms) + Cucumber-one.

Dinner: Chapaties - 2 pcs(from 50gms of atta), Mixed Vegetables -2 servings, Fish-1 pc / Chicken-1 pc), Plain Salad -1 plate,

N.B:

1. Daily 4 teaspoon of oil to be used for cooking.
2. Daily 1 teaspoon of salt to be used for whole day's cooking

This diet provides approximately Energy-1400 kcal/day and sodium 2000mg/day.

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Assessment of Endothelial Dysfunction

Dr. D. Maji

Endothelium is the monolayer of endothelilal cells lining the lumen of blood vessels. The endothelium has many vital and diverse (depending on the particular vascular bed) physiological roles, such as regulation of blood vessel tone, permeability, metabolism and haemostasis. Impairment of endothelial function manifests clinically as oedema, hypertension, abnormal vasoconstriction and hypercoagulability. Indeed, it is a widely held view that impaired endothelial function is also the initial step in atherogenesis, which is largely responsible for ischaemic heart disease and thrombotic strokes,Understanding endothelial function is likely to be a key to modifying risk factors of cardiovascular disorders and their sequelae[1].

Nevertheless, the ideal method(s) of assessing endothelial physiology (and, therefore, pathology) remains uncertain. Various indices have been used to assess endothelial activation, dysfunction and damage: the ideal index would not only be specific to the endothelium but would also be stable and easily measurable — the 'gold standard' remains uncertain, as available indices quantify different aspects of endothelial physiology. In addition, words such as damage, injury, dysfunction and activation are currently freely used in the study of endothelial cell biology without a clear definition, or even a consensus, of their meaning[2].

Various stimuli will cause the endothelium to secrete or release biologically active molecules such as nitric oxide, endothelin, tissue factor and tissue plasminogen activator. The loss of appropriate control of the expression and/or release of these products may result in pathological changes. These changes include leukocyte adherence to and infiltration of the vessel wall (due to increased expression of adhesion molecules, hypertension, peripheral vasoconstriction, and abnormal vascular compliance from imbalance of nitric oxide, endothelin and prostaglandin synthesis) and oedema from loss of correct permeability functioning. Furthermore, thrombosis may arise from the loss of the normally anticoagulantp nature of the endothelium. The anticoagulant nature is maintained by synthesis of prostacyclin, nitric oxide and tissue plasminogen activator, as well as protein C activation by the thrombin/thrombomodulin complex.

Endothelial dysfunction and damage can be detected by various methods: a measure of its ability to respond appropriately to simulated increased shear force (i.e. flow-mediated dilatation) or the concentrations of various molecules that it produces may each give an indication of abnormality.

Plasma Markers Associated with Endothelial Damage/Dysfunction:

von Willebrand Factor:

von Willebrand Factor (vWF) is a multimeric glycoprotein that is synthesized exclusively in endothelial cells and megakaryocytes. High levels of vWF are a (poor) prognostic indicator for myocardial infarction, re-infarction and

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mortality. vWF is also a prognostic indicator of other cardiovascular events such as stroke and the requirement for arterial surgery in patients with hypertension, intermittent claudication, angina and ischaemic heart disease. In addition, high vWF predicts the development of thrombo-embolic events and poor prognosis in patients with rheumatoid arthritis and systemic sclerosis[3].

In unstable coronary artery disease, an early increase of vWF in 48h is an independent predictor of adverse clinical outcome at 14 and 30 days. In the setting of left ventricular dysfunction, levels of vWF have been shown to be abnormal, with the highest level associated with left ventricular aneurysms. Levels of vWF are also positively correlated with New York Heart Association class in chronic heart failure.

Soluble Thrombomodulin (sTM):

As previously mentioned, the endothelium is usually in a resting state and constitutes an anticoagulant surface. Under these conditions, the endothelium synthesizes thrombomodulin and secretes prostacyclin (PGI_2), nitric oxide (NO) and tissue type plasminogen activator (t-PA). With endothelial damage, the endothelium becomes activated and provides pro-coagulant activities at the surface, expressing tissue factor, adhesive molecules and binding sites for factors IX and X, and increasing secretion of plasminogen activator inhibitor (PAI-1).

Thrombomodulin is a transmembrane proteoglycan with a molecular mass of 75 kDa, located on the vascular and lymphatic endothelium surfaces, that functions as an anticoagulant. It has a high affinity for thrombin, forming a 1:1 thrombin-thrombomodulin complex that inhibits fibrin formation, platelet activation, and protein S inactivation by thrombin. The complex also activates protein C, which will inactivate factors Va and VIIIa of the intrinsic pathway. Moreover, formation of this complex directly inhibits the capacity of thrombin to clot fibrinogen and activate platelets.

Levels of sTM are elevated in diabetes mellitus and atheromatous arterial disease, and are higher with increased vascular complications[4]. Some argue that it is a marker of microvascular rather than macrovascular complications, as its levels are not affected by the presence of peripheral vascular disease in diabetics. Levels of sTM may also be altered by treatment with ACE inhibitors, which reduces albuminuria in diabetics, as well as preventing nephropathy, independently of blood pressure control. However, in the ARIC (Atherosclerosis Risk in Communities) study, low sTM was a predictor of future ischaemic heart disease at 6 years follow-up.

E-selectin:

E-selectin (CD62E) is a cell-surface-bound leukocyte adhesion molecule specific to endothelial cells. It mediates the interaction between leukocytes, platelets, and the endothelium. Increased surface expression of E-selectin is probably a reflection of endothelial activation rather than damage. It is not expressed by normal resting endothelial cells.

The soluble form of E-selectin can be detected in healthy controls, and is raised in patients with cancer, haematological disorders (myelodys-plastic syndromes and thalassaemia), ischaemic heart disease, atherosclerosis, hypertension, diabetes and septic shock. Although both soluble E-selectin and vWF are elevated in hypertension, controlling blood pressure reduces vWF but not E-selectin. In addition, vWF, but not E-selectin,

is elevated in hypercholestrolaemia. All three markers, (vWF, E-selectin and sTM) are increased in ischaemic heart disease[5].

Nitric Oxide ('endothelial-derived relaxing factor')

NO contributes to the control of basal and stimulated regional blood flow in man. Intra-arterial infusion of N-monomethyl-L-arginine (L-NMMA), a specific NOS inhibitor, into arteries of healthy controls results in a significant fall in basal blood flow and attenuates the dilator response to infused acetylcholine. The ability of blood vessels to vasodilate in response to increased shear force (i.e. the force exerted on the blood vessel wall as a result of laminar blood flow) also requires an intact endothelium. NO is a highly unstable molecule with a half-life of <6 s *in vivo*, being rapidly oxidized to nitrite, and subsequently nitrate. It is synthesized from L-arginine by NOS, and we now recognize that cardiac myocytes express two types of NO synthases, endothelial NO synthase (eNOS) and inducible NO synthase (iNOS). The production of NO is stimulated by shear stress via the eNOS, and by inflammatory cytokines such as TNF-α via the pro-inflammatory iNOS. TNF-α downregulates eNOS expression while at the same time inducing iNOS.

Basal production of NO is increased in CHF due to stimulation of iNOS. Experiments suggest that NO, while counteracting the systemic vasoconstriction in CHF, is potentially lethal to myocytes in high concentrations, in addition to being a negative inotrope[6].

Endothelin:

Endothelin, is an endogenous 21-amino acid peptide and a powerful vasoconstrictor produced not only by vascular endothelial cells but also by other cell types, including adrenal cortex, myocardium, vascular smooth muscle cells, renal tubular epithelial cells, glomerular mesangial cells, glial cells, macrophages, mast cells and pituitary cells. Indeed, various studies have shown that ET-1 level is an excellent prognostic marker in CHF, leading to the current interest in endothelin antagonists in the management of heart failure. While it has shown some promise in that it affects the haemo-dynamics favourably, it is not known whether these agents can reduce mortality in CHF.

Endothelin is synthesized as an approximately 200-amino acid pre-pro-hormone. Post-translational cleavage yields a 38–39 amino-acid pro-endothelin, which undergoes further cleavage to yield the final 21 amino-acid product. Four isoforms of endothelin have been identified to date and designated ET-1, ET-2, ET-3 and ET-4, alongside two receptors for endothelin, termed ET_A and ET_B, which are expressed on endothelial cells, vascular smooth muscle cells, cardiac myocytes and fibroblasts.

Endothelin has positive inotropic, and positive chronotropic, mitogenic and pro-inflammatory properties, as well as its vasocontrictor effects. Its release is stimulated by many factors, such as shear stress, hypoxia, epinephrine, angiotensin II, cortisol, thrombin, pro-inflammatory cytokines (TNF-α, IL-1 and IL-2) and transforming growth factor β.

Increased levels of plasma ET-1 have been observed in systemic hypertension, type 2 diabetes mellitus, dyslipidaemia, angina, cardiogenic shock, myocardial infarction[7], Raynaud's phenomenon, cerebral vasospasm, atherosclerosis and heart failure. It has been suggested that elevated endothelin levels may reflect endothelial dysfunction and damage.

Using vWF as a marker of endothelial dysfunction, ET-1 levels have been observed to be increased along with vWF in patients with type 2 diabetes mellitus and dyslipidaemia; synthesis of vWF and ET-1 are also increased following exposure of cultured endothelial cells exposed to tri-iodothyronine. ET-1 and vWF levels are also directly correlated in both congestive heart failure and after heart transplantation in idiopathic dilated cardio-myopathy. However, in contrast to flow-mediated dilatation (see discussion below) which improves following heart transplantation for end-stage heart failure, there is evidence that ET-1 levels increase further, perhaps as a consequence of immunosuppressive therapy. There is also experimental evidence that ET-1 inhibits synthesis of NO in smooth muscle cells via ET_A receptors. As ET-1 levels are elevated in CHF, this observation is entirely consistent with a concomitant decrease in NO.

Flow-Mediated Dilatation/Endothelium-Dependent Dilatation:

Flow-mediated dilatation (FMD) is now increasingly used as a research tool for the assessment of endothelial function, as it has been shown to be accurate and reproducible. By FMD, we mean the high-frequency ultrasound assessment of arterial diameter rather than plethysmography; where plethysmography is used, it has been qualified.

Celermajer *et al*. first described FMD to detect endothelial dysfunction in children and adults at risk of atherosclerosis in 1992, in which the underlying mechanism was nitric oxide (NO) release[8]. They measured FMD in the superficial femoral and brachial arteries by comparing the diameter of the aforementioned arteries using high frequency ultrasound at rest, and comparing with measurements during reactive hyperaemia induced by an inflated pneumatic cuff at a pressure of 300mmHg for 4.5 min, and after sublingual glyceryl trinitrate. Reactive hyperaemia results in increased shear force (i.e. the force exerted by laminar blood flow on the vessel wall), which is already known to stimulate NO release. Sublingual glyceryl trinitrate, on the other hand, causes endothelial-independent vasodilation.

Others have modified and refined the method of ultrasound assessment of FMD. For example, the use of brachial artery vs. radial artery, and the location of the blood pressure cuff on the upper arm or forearm, FMD of the brachial artery was significantly higher after upper-arm occlusion compared to forearm occlusion, it seemed likely that local ischaemia plays a part. FMD (measured as percentage change in diameter) of the radial artery is greater than brachial artery. FMD has also been shown to be dependent upon the anatomical vessel size but independent of body mass index. The effect of age on FMD is unclear, as some studies have shown a correlation, whereas others have not. That FMD is greater in smaller arteries may be explained by greater hyperaemic wall shear stress in response to the same stimulus. Reduced FMD has been demonstrated in children with familial hypercholesterolaemia and adult smokers at risk of atherosclerosis or with established coronary artery disease.Others have demonstrated impaired FMD in patients with hypertension, and shown it to be a marker of future cardiovascular events in patients with essential hypertension. Moreover, endothelium-dependent vasodilatation by plethysmography has also been shown to be impaired in type 2 diabetics, as well as in human and animal models

of congestive heart failure. This could perhaps be explained by the fact that stimulated release of NO is impaired, an end result of endothelial dysfunction[9,10].

Long-term therapy with angiotensin converting enzyme (ACE) inhibitors improves FMD in congestive heart failure (CHF). The proposed mechanism by which ACE inhibitors improve FMD is interesting. ACE itself is virtually identical to kininase II, which degrades bradykinin *in vivo*. By inhibiting this enzyme, ACE inhibitors increase the availability of bradykinin. FMD is a measure of endothelial function/dysfunction, and the method indirectly measures NO release in response to shear stress due to laminar blood flow. The method requires specialized and expensive equipment, as well as highly trained technicians, to produce valid, reproducible data. Its attraction is that it is non-invasive and allows repeated measurements.

Circulating Endothelial Cells:

Perhaps the best proof of endothelial damage would be to observe desquamated, but not apoptotic endothelial cells in circulating blood. A method to capture these cells has been developed, and used to prove that endothelial injury occurs in acute myocardial infarction and unstable angina (but not stable angina), confirming a separate pathogenic mechanism. Mutin *et al*. used an immunomagnetic separation assay based on S-Endo 1 monoclonal antibody directed against the endothelial antigen CD146 to capture circulating endothelial cells in myocardial infarction and unstable angina[11].

Circulating endothelial cells were not present in blood obtained from healthy controls and patients with stable angina, but were present in the blood of patients with myocardial infarction and unstable angina[12]. This is consistent with the currently held theory that the pathogenesis of myocardial infarction and unstable angina involves atheromatous plaque rupture as the initial event, whereas a 'fixed' stenosis results in stable angina. The presence of circulating endothelial cells is therefore direct evidence of endothelial injury.

Conclusion:

The assessment of endothelial function is essential in cardiovascular disease. Modulation of endothelial function would have implications for the thrombus-related complications (including myocardial infarction, stroke and thrombo-embolism) that commonly occur in heart failure. Many Endothelial markers are not mere bystanders in the disease process. Apart from FMD, which is an indirect measure of NO production, vWF, sTM and NO are all components involved in the complex process of thrombogenesis. Furthermore, the immune system may be more closely linked to the endothelial function than previously thought.

Nevertheless, the ideal method(s) of assessing endothelial physiology (and, therefore, pathology) remains uncertain. Various indices have been used to assess endothelial activation, dysfunction and damage: the ideal index would not only be specific to the endothelium but would also be stable and easily measurable—the 'gold standard' remains uncertain, as available indices quantify different aspects of endothelial physiology.

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Role of MRI in Staging Urological Malignancies

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In the modern era management of cancer, pre-treatment staging is very important for guiding treatment protocol based on scientific terms. Advances have occurred in practically all aspects of MR imaging of the genitourinary tract. Continued improvements in MR hardware, such as the advent of higher-magnetic-field-strength MR scanners and higher-resolution functional imaging techniques may aid in improving staging accuracy further. Having this knowledge should help in decision making, particularly in patients contemplating different treatment modality.

Renal Mass:

MRI of the kidney is often utilized in the evaluation of renal masses in patients who have had an indeterminate CT or US study or in patients who are allergic to iodinated contrast or have renal insufficiency. MRI has proved to be one of the most accurate means of staging RCC patients and has got staging accuracy upto 95%. Although MRI appears slightly more sensitive than CT, it is equally non-specific for distinguishing between Robson's Stage I and II disease (T1-T2 vs. T3a). Detection of peri-nephric invasion, or its absence, can be improved by using fat-suppressed contrast-enhanced images, with enhancement of previously low signal intensity areas in the perinephric tissue indicating extrarenal tumor extension.

The most important role of MRI is in assessing venous involvement.Imaging in the sagittal and coronal planes is particularly helpful for assessing the superior extent of caval tumor thrombus in relation to the diaphragm, hepatic veins and right atrium. Conventional spine-echo sequences have a very high accuracy for detecting venous invasion. However, limited flip-angle GRE (gradient echo sequence) techniques facilitate the imaging of vascular structures and their use in RCC has allowed accuracies of 100% in assessing vena caval invasion, 88% for renal vein tumor thrombus and 80% for atrial invasion. Intravenous administration of gadopentatedimeglumine may improve the accuracy further, particularly when combined with 3D magnetic resonance venography (MRV). MRI has been shown to be superior to CT for delineating the upper extent of the tumor thrombus in the IVC. Detection of invasion of the IVC wall necessitates resection of the affected segment and subsequent vascular reconstruction. To date, wall invasion has not been reliably detected by imaging but MRI does appear able to make this diagnosis. The most reliable signs of vessel wall invasion are a tumor signal on each side of the vessel wall and vessel wall enhancement. In fact properly performed MRI is equal to or better than inferior venacavagraphy in assessment of RV and IVC tumor thrombus .

The sensitivity and specificity of MRI for N-staging in RCC, however, do not exceed those of CT. A disadvantage of MRI relates to the absence of a universally acceptable bowel contrast agent, which in thin patients particularly can result in confusion between bowel and lymph

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node masses. However, MRI has the advantage that it can distinguish between large collateral vessels and lymph nodes when the CT findings are uncertain. The accuracy of assessing visceral invasion of RCC with MRI has been reported to be between 97% and 98% because the excellent contrast resolution of MRI allows assessment of the spread of Stage IV tumors to neighboring organs. Despite its accuracy in staging RCC, MRI does have several disadvantages for the detection and staging of the disease. Small renal cancers (<1 cm) or calcification within a mass may go undetected.

In recent years, interest in nephron-sparing surgery has been growing. The presence of a pseudocapsule around a renal tumor is a sign of lack of perinephric fat invasion and therefore a favorable sign for partial nephrectomy. The sensitivity of CT in depicting a pseudocapsule is low (10–26%), whereas MRI shows a moderate to high sensitivity in depicting the pseudocapsule (54–93%). On MRI, a pseudocapsule presents as a hypointense rim around the tumor on both T1-weighted and T2-weighted images, but can be best seen on the T2-weighted images and sometimes on gadolinium-enhanced GRE images.

Adrenal Mass:

The MRI, in particular, shows significant utility in distinguishing adrenocortical carcinoma from nonfunctional adenomas and pheochromocytomas. The relatively higher fat content of adrenal adenomas compared with ACs has also been used in the new chemical shift imaging (CSI) MRI to further enhance the distinguishing capacity of these studies. MRI is not considered superior to adrenal CT for indeterminate adrenal nodules although it may sometimes be helpful for example if only non IV contrast CT is possible or a pheochromocytoma is being considered. Chemical shift imaging is the most accurate MRI method for distinguishing between adenomas and malignancies. MRI is generally not used to characterize small (<1 cm) masses because of its lower resolution compared to CT.

Neoplasm of Collecting System, Pelvis and Ureter:

Although information is limited, MRI at present has little to offer in diagnosis and staging. MRI has an advantage over CT in its ability to display ureter in direct coronal, saggital and oblique planes and can also define the extent of the tumor. However, MRI does play a role if CT and endoscopy are not feasible as in case of poor renal function and ureteric obstruction. MRI can help to differentiate the post operative fibrosis near the ureteric stump from ureteric stump carcinoma (after nephro-ureterctomy), therefore overstaging of stump carcinoma can be avoided.

Bladder Cancer:

Beside its multi-planar anatomical imaging utility, MRI has the potential to offer functional information through pulse sequences such as Diffusion Weighted MRI (DW MRI) and Dynamic Contrast Enhanced MRI (DCE MRI). MR Cystoscopy (MRC) can be a useful adjunct to conventional cystoscopy to localize difficult-to-see lesions for instance, those that require retroflexing of the scope. MRC has several advantages over conventional cystoscopy such as being relatively impervious to hematuria, urethral strictures and anterior bladder wall lesions (which can be difficult to reach via cystoscopy). Importantly, it is not possible to perform a biopsy with MRC. Accuracy of MR imaging on a stage-by-stage basis has been reported to be between 64% and 85%.

Staging accuracy of MRI in differentiating

superficially versus deeply muscle-invasive tumors, and organ-confined versus non–organ-confined tumors is between 85% and 95%. Superficial tumors may be differentiated from muscle-invasive tumors on contrast-enhanced images. MR is not accurate in distinguishing superficial tumors involving only the mucosa (T1) from early muscle invasion (T2a). MR may be able to distinguish superficial muscle invasion (T2a) from deep muscle-invasion (T2b) in many cases. Invasion into the perivesical fat can be identified when there is macroscopic invasion (T3b) but microscopic invasion (T3a) cannot be reliably identified. MRI is accurate in staging more advanced disease, such as invasion of adjacent organs, indicating T4 disease. Based on MR imaging features, it is not possible to differentiate between different histologic cell types of bladder tumors, but few clues may lead to the differentiation of transitional from non–transitional-cell carcinomas. Non–transitional-cell carcinomas tend to cause marked bladder wall thickening and tend to be larger than TCC.

The diagnosis of nodal metastatic disease relies on size criteria but sensitivity is low, as normal sized nodes may contain metastatic deposits. Pitfalls for interpretation include : detection of small tumors less than 1cm; tumors with minimal elevation from the bladder surface; differentiation of bladder mucosal inflammation and tumour, which may result in overcalling T3 disease; inability to detect microscopic invasion of perivesical fat, resulting in understaging; underdistension or overdistension of the bladder, which may result in difficult interpretation of bladder muscle invasion. On MRI, differentiation of acute edema or hyperaemia, present during the first week after TURBT; and tumor is difficult. Therefore tumor staging after TUR will be less accurate. Because MRI is the best imaging modality for invasive and metastatic tumor, patients with stage higher than T_2 or stage T_1 grade 3 disease should be considered for further staging with MRI.

Prostrate Cancer:

With functional multi-modality MR imaging, it is possible to detect and exactly localize the tumor in the prostrate with more than 90% accuracy. An important role of morphologic T2-weighted MRI is the assessment of local extracapsular extension and invasion of the seminal vesicle in a patient with no documented distant metastases. Signs of extracapsular spread include irregular bulging of the prostatic outline, breach of the capsule with infiltration of the periprostatic fat, asymmetry of the neurovascular bundles, and loss of the rectoprostatic angle. Seminal vesicle invasion may be suspected in the presence of an abnormally low signal intensity within the lumen of the seminal vesicle or by focal thickening of the seminal vesicle walls. The reported sensitivities and specificities for local staging range from 14-100% and from 67-100%, respectively. Because MRI cannot detect microscopic invasion, low sensitivity values are not unexpected. The main indication for local MRI staging, however, is the assessment of capsular and vesicular integrity in a patient clinically staged as T1c or T2c. Such patients obviously should not be inappropriately upstaged by MRI and therefore a conservative approach is adopted in which only unequivocal capsular or vesicular extensions are assigned a T3 status. This implies high specificity reading (no false positives) at the expense of a lower sensitivity.

Magnetic resonance spectroscopy provides information about the relative concentration of cellular metabolites in the prostrate, such as citrate and choline. Citrate is a marker of normal prostatic tissue, while an increased concentration of choline is suggestive of a tumor lesion. The complementary changes of both metabolites are used to predict the presence or absence of prostrate cancer. When used in combination with T2-weighted images, sensitivities and specificities ranging from 59-94% and 80-95%, respectively, have been reported. A useful correlation between the choline-to-citrate ratio and tumor aggressiveness (Gleason score) has also been demonstrated, and a particularly high negative predictive value was found in ruling out high-grade prostrate cancer (ie, Gleason 4+3 or higher grade) in men presenting with an increased prostate-specific antigen (PSA) value.

Endorectal MRI utilizes a magnetic coil placed in the rectum to better visualize the zonal anatomy of the prostate and better delineate tumor location, volume, and extent (stage). On T2-weighted images, zonal anatomy is evident : Prostrate cancer is characterized by low T2 signal intensity in the normally high-signal-intensity peripheral zone. Accurate localization of cancer within the prostrate gland is of increasing value, given the widespread interest in more focal forms of therapy, whether with radiation or percutaneous sources (ie, cryotherapy or high-intensity focused ultrasound). The ability of endo MRI/MRSI to identify cancer is dependent on tumor size and grade. Cancers less than 0.5 cm in diameter may be missed with endoMRI/MRSI, whereas those larger than 0.5 cm are identified with reasonable accuracy, although volumetric measurements may be imprecise. Similarly, cancers of higher grade (Gleason score 8–10) are better imaged than those of lower grade. Although the inability of endoMRI/MRSI to consistently identify low-volume or low-grade cancers may be seen as a limitation of the technique, such cancers are likely to have a long natural history even without treatment, so failure to detect and treat them may have little impact on cancer-specific morbidity and/or mortality. Identifying the presence or absence of ECE and SVI before treatment is of considerable value not only in selecting but also in applying the treatment. For instance, patients with suspected ECE who select radical prostratectomy may require a wide surgical excision. Similarly, those who select radiation may benefit from a combined modality treatment (eg, androgen deprivation and radiation, combined external beam radiation and brachytherapy). ECE is seen as a focal irregular capsular bulge, asymmetry, or invasion of the neurovascular bundles and/or obliteration of the rectoprostatic angle. Endorectal MRI has been reported to have a sensitivity of 13% to 91% and specificity of 57% to 97% for the assessment of ECE. The sensitivity and specificity for assessing SVI vary between 20% and 80% and between 92% and 98%, respectively, in several reported series. Recent reports from Memorial Sloan-Kettering Cancer Center found that endoMRI/MRSI added incremental value to the use of clinical variables alone for assessing prostrate cancer. The incremental value was greatest for intermediate-and high-risk groups of patients. Evidence is now emerging that endoMRI/MRSI may be helpful in assessing response to treatment, most commonly with radiation and/or androgen deprivation.

In MR lymphangiography (MRL), Combidex consists of iron-oxide containing nanoparticles is used. With MRL, it is possible to examine the

entire abdomen instead of only a restricted area surrounding a few pelvic blood vessels, as is the case with PLN dissection. The sensitivity, specificity, negative and positive predictive values are respectively 82%, 93%, 96%, and 69%. The high negative predictive value (>96%) of MRL means that after a negative result on MRL, PLN dissection does not have to be performed. Due to the latter, obtaining a diagnosis with MRL is economically cheaper and results in fewer complications than with the current invasive diagnostic technique of MDCT + PLND. In addition, in at least in 30% of patients, thanks to MRL, nodes are detected which are not found by the routine PLN dissection, as they are located in the internal and common iliac, perirectal and para-aortic regions. MRI has shown great promise as a tool for the noninvasive assessment of prostate cancer. Although functional MRI has still several limitations, it is hoped that advances in 3.0 T multiparametric MRI as well as advances in molecular imaging will further improve patient care by enabling even better treatment selection, planning, and outcomes.

Testicular Cancer:

Current indications for the use of MRI include the detection of brain metastases and the diagnosis of suspected meningeal disease or spinal cord involvement. A MRI can reduce the incidence of diagnostic surgical explorations for scrotal pathology. The technique has been proven highly accurate in the differentiation of extratesticular from intratesticular disease, providing good results in the distinction between benign and malignant testicular masses and allowing the accurate evaluation of the local extent of the disease in cases of testicular carcinoma. MRI is particularly recommended when sonographic findings are inconclusive or inconsistent with the clinical findings. Several studies have showed that MRI findings could be closely correlated with the histologic characteristics of testicular neoplasms, providing a preoperative classification of the histologic type of testicular tumors. Further more, MRI offers no advantage over CT for imaging and staging of the retroperitoneum in patients with testicular cancer.

Urethral Cancer and Penile Cancer:

MR imaging advantage over other imaging modalities due to its multiplanar imaging and superior contrast resolution in assessing the periurethral spread of the tumor. Tumor extension into the tunica albuginea or septa of corpus cavernosum can be readily demonstrated on T2W images. MR imaging has been reported to be accurate for evaluating local urethral tumors in 90% of patients. MRI of pelvis has benefit of showing contiguous or regional metastasis from urethra and perineum and it may be preferred when clinical evidence suggests pelvic vascular involvement. MRI is useful for local cancer staging and for assessing the inguinal lymph nodes in penile cancer. With regards to the primary tumor, the initial assessment should be made by physical examination. It has been shown that in experienced hands, its correlation with the histopathologic examination after surgery is superior to that which can be derived from magnetic resonance imaging (MRI). These modalities would be reserved for lesions in which an adequate exam could not be performed, such as in the morbidly obese patient. However, the use of an intracavernosal injection of prostaglandin E1 as an adjunct prior to MRI scan has shown promise in some series by improving its accuracy in assessing the clinical stage of the primary tumor. The sensitivities and specificities, respectively, for this modality in

correctly assessing clinical T1 tumors are 85% and 83%, for T2 tumors 75% and 89%, and for T3 tumors 88% and 98%. Equally problematic is the staging of nodal disease. Here again, the initial assessment is made by physical examination, through palpation of the bilateral inguinal region. If the nodes are non-palpable after an adequate physical exam, there is generally no indication for imaging. A technique to identify lymph node metastases using MRI following the intravenous injection of ferromagnetic particles has shown a high degree of sensitivity (sensitivity of 100%, a specificity of 97% and a positive predictive value of 81.2% in the ability to detect micro-metastatic disease).

MRI in Staging of Skeletal Metastasis:

^{99m}Tc bone scintiscan findings are nonspecific in determining the cause of increased uptake, particularly in solitary lesions. The major advantages of WB-DWI include the fact that no ionizing radiation is administered and no injection of isotopes or any contrast medium is necessary. Importantly, whole-body examinations are possible in reasonably short data acquisition times. WB-DWI excels at lesion detection in the bone marrow, being better than CT scans and bone scans for detecting bony disease. MRI depicts early hematogenous dissemination of the tumor to the bone marrow before reactions in adjacent bone are detectable on ^{99m}Tcscintiscans. MRI is particularly well suitedto detect spinal metastases, and most authoritiesagree that it is superior to planar scintigraphy for this purpose. Magnetic resonance imaging is potentially the technique of choice in evaluating prostate bone metastases as it is sensitive to early changes in bone marrow that precede the osteoblastic response in the bone matrix. In particular, vertebral bodies have a large medullary cavity, and hence the cortical involvement leading to positive bone scintigraphy occurs late. Furthermore, tumour cells may reside between trabeculae in which they may be recognised on MR but not on bone scintigraphy or plain film.

Given the vast domain of applicability of MRI in urological malignancies diagnosis and staging and the information provided thereof, it appears as the era has come when it would replace, if not substitute, all other imaging modalities available As for now, use of MRI is traditionally in its infancy, partly in due of practicing urologist preference for CT which may be related to better acquaintance and understanding of CT images as compared to MRI. Magnetic resonance imaging technology will continue to pose challenges with the evolution of advanced techniques. Understanding and mastering the challenges of MRI imaging today is crucial and will ultimately result in superior spatial resolution, speed, and consistent image quality when compared with present day systems. In today's diagnostics environment, some consider cost to be the only true barrier to the widespread adoption of MRI for clinical imaging. Only time will tell if today's hurdles to MRI can be overcome. Regardless of the future of MRI specifically, MRI modalities will continue to evolve and definitely will fulfil new needs of urologist.

Epidemiology of Hypertension

Prof. Soumitra Kumar

Hypertension is an important risk factor for death, stroke, and CVD, and is a major cause of end-stage renal disease. WHO has recognized hypertension as the leading global risk for mortality in the world. Kearney et al[1] estimated that the prevalence of hypertension in 2000 was 26% of the adult population globally, and that in 2025, the prevalence would increase by 24% in developed countries and 80% in developing countries. According to a recent review on 'the global burden of hypertension", the estimated prevalence of hypertension (in people aged 20 years and older) in India in 2000 was 20.6% among males and 20.9%[1] among females and is projected to increase 22.9% and 23.6%, respectively, in 2025. The estimated total number of people with hypertension in India in 2000 was 60.4 million males and 57.8 million[2] females and is projected to increase to 107.3 million and 106.2 million, respectively, in 2025. There appears to be a steady increase in hypertension prevalence over the last 5 years, more in urban than in rural areas. It is well recognized that hypertension is now a major health problem in India, and this calls for large, nationwide, multicentric, prospective, and supervised epidemiological studies[3]. CVDs caused 2.3 million deaths in India in the year 1990; this is projected to double by the year 2020. Hypertension is directly responsible for 57% of all stroke deaths and 24% of all coronary heart disease-related deaths in India. This fact is important because hypertension is a controllable disease and a 2 mmHg population-wide decrease in blood pressure can prevent 151,000 stroke and 153,000 coronary heart diseae-related deaths in India[4]. Thus, there is a need to increase awareness, detection, and adequate control of blood pressure[3].

2003 witnessed 7th report of the Joint National Committee (JNC7) on Prevention, Detection, Evaluation and Treatment of High Blood Pressure[5] and the first version of the joint European Society of Hypertension (ESH)/ European Society of Cardiology (ESC). Guidelines for the management of arterial hypertension. The next version of ESH/ESC Guidelines was produced in 2007[7] and very recently, in June 2013, the latest version of the ESH/ESC Guidelines for management of arterial hypertension has been produced. In contrast, the 8th report of the JNC is still awaited even after a gap of ten years and it is being dubbed as "Late" JNC report. Some salient features of the ESH/ESC Guidelines 2013, which is currently the most important current overview to consider the totality of hypertension treatment, are being discussed below:

The 2013 Task Force reviewed all relevant data since the last revision (in 2007), with 18 specific diagnostic and therapeutic areas identified as containing significant change.

A major development is the decision to recommend a single systolic blood pressure target of 140 mmHg for almost all patients. This contrasts with the 2007 version which

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recommended a 140/90 mmHg target for moderate to low risk patients, and 130/80 mmHg target for high risk patients. "There was not enough evidence to justify two targets" said Professor Robert Fagard (Leuven, Belgium).

Other Changes Include:

- An increasing role for home blood pressure monitoring, alongside ambulatory blood pressure monitoring.
- A greater emphasis on assessing the totality of risk factors for cardiovascular and other diseases. For example, most people with hypertension also have additional risk factors such as organ damage, diabetes, and other cardiovascular risk factors. These need to be considered together before initiating treatment, and during the follow-up.
- Special emphasis on specific groups e.g. diabetics, the young, the elderly, and drug treatment of the over 80s. Women are also considered separately, e.g. during pregnancy. Special consideration is given to new treatments such as renal denervation for resistant hypertension – which is described as "promising", although more trials are called for.
- New guidance on how and when to take anti-hypertensive drugs. The report indicated no treatment for high normal blood pressure, no specific preference for single drug therapy, and an updated protocol for drugs taken in combination. The guidance takes a liberal attitude to choice of first step drugs, noting the evidence that the beneficial effect of hypertension depends largely on blood pressure lowering. Rather than presenting a hierarchy of drugs (a genetic 1st, 2nd, 3rd choice and so on), the approach taken promotes individualized treatment, i.e. to help physicians to decide which drugs to give in which clinical/demographic condition.

  The Guidelines were developed over an 18 month period. Task Force members from both societies met several times to finalise the content, which was reviewed twice by a group of 40 European reviewers in addition to the internal reviewers. Both the ESC and the ESH appointed Task Force Chairs, who worked together to coordinate the Guideline development. For the first time, the Guidelines have graded the evidence according to class of the recommendation and the level of evidence.

Several therapeutic issues are still open to question and would be answered only from further investigations. Some of these are:

(i) Benefit of drug treatment for grade 1 hypertension at low to moderate CV risk or in elderly patients with SBP between 140 to 160 mmHg or for subjects with "White Coat Hypertension".

(ii) Are strategies based on control of out-of-office BP over strategies based on conventional (office) BP control advantageous?

(iii) Does central BP add to CV event prediction in untreated and treated hypertensive patients?

(iv) Do invasive procedures (eg. renal sympathetic denervation) for treatment of resistant hypertension compare favourably with the best drug treatment and provide long-term BP control and reduction of morbid and fatal events.

(v) Benefit of treatment of high normal BP in high-risk individuals.

It appears reasonable that over next few years, randomized clinical trials (RCTs) would address these issues and their impact on reduction of morbidity and fatal events will be keenly awaited.

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Silent Rupture of Myomectomy Scar During the Third Trimester of Pregnancy in a Primigravida : A Case Report

Dr. Santosh K Verma[1], Dr. Archana Singh[2], Dr. Sibani Sengupta[3]

Introduction:

Rupture of the uterus in pregnancy is a rare and often catastrophic complication responsible for high maternal as well as fetal morbidity and mortality[1]. In a scholarly article Nahum et al[1] have described 25 peer reviewed publications and it was found that the incidence of uterine rupture was 1 in 1,146 pregnancies (0.07%). The present case is one such rare occurrence where there were nonspecific symptoms which to some extent delayed the diagnosis and definitive therapy; the maternal mortality could be avoided due to prompt action taken after the diagnosis was made.

Report of the Case:

Booking history and records: Mrs. R. D. was a 31yr. old primigravida married for 10yrs. with history of myomectomy in the year 2011. She visited the OPD at RKMSP on the 4th Sept. 2012 with a 15 weeks intrauterine gestation. Her LMP-21/5/12 and EDD-28/2/13. (confirmed by USG).

Her baseline investigation reports which included blood group and Rh type, Hb%, pcv, thyroid profile, blood sugar, routine urine and stool were all within normal limits.

From her previous surgical records, it was found that she actually had a myomectomy which started with laparoscopy and ultimately ended up with a laparotomy as the cavity was opened during the procedure and the wound repaired after laparotomy.

Second Trimester:

During second trimester she had post prandial blood sugar (2 hr after 75gm glucose) 138mg/dl for which she received medical nutrition therapy. Her obstetric examination was found to be within normal limits, when examined clinically. Heranomaly scan was also within normal limits.

Third trimester:

At the beginning of the third trimester, she was examined at the OPD as usual and then was admitted on 18th Jan, 2013 at 34 weeks 1 day. This was because of two factors:-

1. She had a history of myomectomy
2. Her USG findings were suggestive of mild IUGR GA- 32 weeks +2 days (GA by LMP 34 weeks +1 day) AFI – 4.5cm

   COLOUR DOPPLER- normal study

Her USG was repeated after 1 week (25.1.13) @ 35 + weeks-it reconfirmed our finding; so, our decision was to deliver her on completion of 36 weeks, that is, on or after 31st January, or earlier in the event of any emergency.

On 26th January, Saturday, at 12 noon a repeat USG was done in our departmental day care and all the findings were as before-this was done as there were two consecutive holidays.

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[3]Asso. Prof., Dept. of G&O, RKMSP-VIMS

Description of the Emergency:

On 26th January, at about 8 pm, the patient complained of epigastric distress – she gave history of taking some junk food in the afternoon. The on-duty labor room officer examined her immediately.

All the vitals were checked and found to be normal including fetal heart rate. She was given a Ranitidine tablet and said that she felt better. About an hour later, she was rechecked and everything was found to be normal.

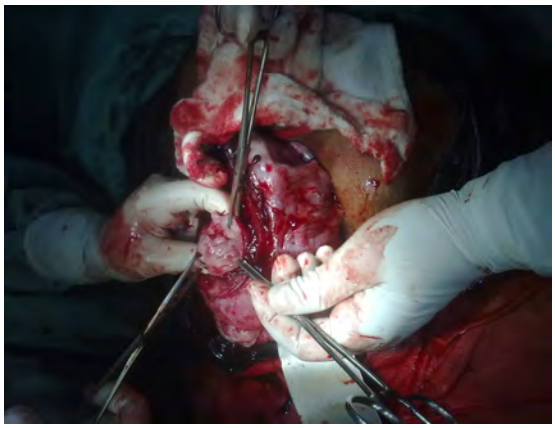
At about 10 pm, regular checkup by on duty staff nurse using Doppler failed to localize the fetal heart. Immediately, she was taken to the day care unit (whichis adjacent to the antenatal ward). Fetal heartsound could not be localized by the Registrar on duty-there were some retro placental clot raising suspicion of abruption or uterine rupture.

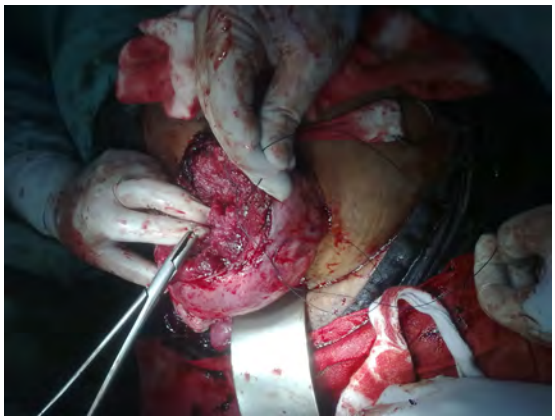
At that time, for the first time, the patient complained of mild dizziness and on checking, her pulse was found to be feeble, rate 100/min. BP-80/60 mmHg. Uterine contour clinically appeared to be normal.

The senior registrar was called from Radiology department, he confirmed the findings of absent FHS and also diagnosed Hemoperitoneum, suggesting uterine rupture.

Meanwhile, as her general condition was deteriorating, fluid transfusion was started and the patient was taken to the operation theatre. Blood was made available immediately.

Patient's general condition, however, improved after transfusion of crystalloids as well as volume expanders.





Repair Details:

Under general anesthesia,a midline infra-umbilical longitudinalincision was made. At laparatomy the peritoneal cavity was found to be full of dark blood and an intact Amniotic membrane which had to be opened to deliver (by breech) a freshly dead baby floating in a *clear* liquor. The placenta was loosely adherent to the rupturesite. After delivering the dead fetus and after births, the uterus was delivered through the incision.The fundus was open along the sagittal plain. There was surprisingly little amount of active bleeding and there was a submucus fibroid of about 2.5 cm × 2.5 cm in

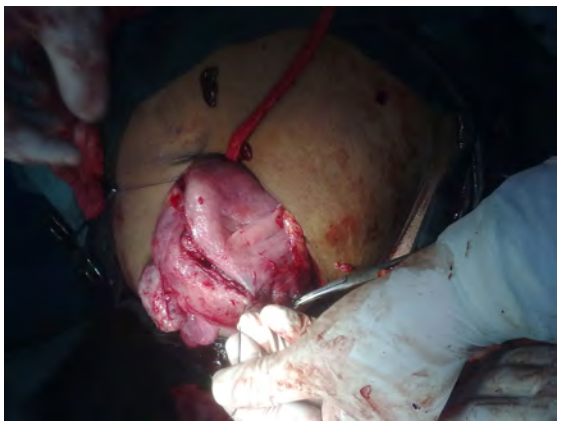
diameter over the anterior uterine wall. It was quickly enucleated and the bed sutured.

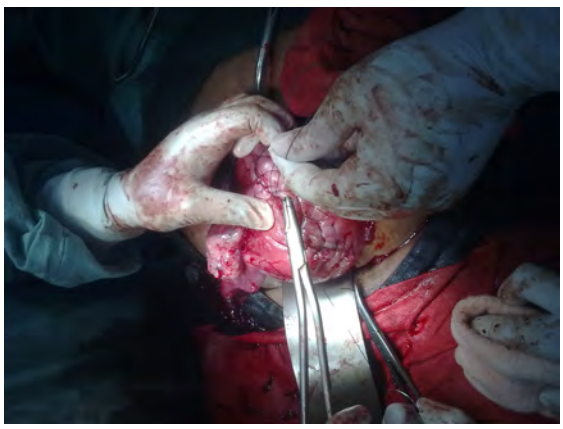
Considering the poor obstetric outcome and the then stabilized condition of the patient, clear cut margins of rupture wound, a decision of repair was taken.

The uterus was repaired in two layers using continuous 1-0 vicryl over a round body needle. The patient was transfused with one unit of whole blood during operation.

Post-Operative Period:

Patient was monitored closely and another unit of whole blood was transfused. Patient was stable and recovered satisfactorily.





Follow up:

The patient came for follow up after 6 weeks and she had no complaints at that time. She was advised to use barrier contraceptives till the return of her periods.

Sumary:

- The patient was a primigravida who had undergone Myomectomy by laparoscopy, but since the cavity was opened inadvertently, the repair was done by laparotomy.
- Interval between the above repair and conception was more than 1 year.
- The scar ruptured silently at 3rd trimester (35 weeks), while she was posted for Cesarean section at 36 weeks.
- Complete repair was possible.

The Fetus could not be saved as the rupture was extremely deceptive in its presentation, thus delaying diagnosis.

Discussion:

Uterine rupture occurs when a full-thickness disruption of the uterine wall that also involves the overlying visceral peritoneum (uterine serosa) is present. By definition, it is associated with the following:

- Abdominal pain
- Clinically significant uterine bleeding
- loss of uterine contour
- Fetal distress and fetal death,
- Protrusion or expulsion of the fetus and/or placenta into the abdominal cavity
- Shock[1]

These necessitate prompt laparotomy and delivery, followed by Uterine repair or hysterectomy as the situation permits.

In the present case, none of these symptoms / signs were present till late. The fetal heart was initially normal (at about 8 pm) and as she complained of epigastric distress, which was attributed to some junk food taken in the afternoon, and she was clinically stable, the risks of rupture did not cross the mind of the on-duty Registrar. She was found to be in Shock only when she complained of mild dizziness while the USG was being done by the Registrar of Radiology to confirm fetal demise and to find whether she was having Accidental hemorrhage or a Rupture. This may be compared to a case mentioned in Munro Kerr's book of Operative Obstetrics where a woman climbed and alighted several flights of stairs repeatedly as she was feeling some uneasiness and feeling like voiding and defecating, before being diagnosed with a rupture[1a].

Nearly all uterine ruptures that involve uteri with myomectomy scars have occurred during the third trimester of pregnancy or during labor. Only 1 case of a spontaneous uterine rupture has been reported before 20 weeks of gestation[2]. In the present case, the rupture occurred in the 3rd trimester.

Brown et al reported that among 120 term infants delivered after previous Trans abdominal myomectomy, no uterine ruptures occurred, and 80% of the infants were delivered vaginally[3]. In contrast, Garnet identified 3 uterine ruptures among 83 women (4%) who had scars from a previous myomectomy and who underwent elective cesarean delivery because of previous myomectomy[4].

Such reports do not clearly identify the factors that are important for assessing the risk of subsequent uterine rupture (e.g., number, size, and locations of leiomyomata; number and locations of uterine incisions; entry of the uterine cavity; type of closure technique). Further studies to investigate these issues are needed.

Dubuisson et al reported 100 patients who underwent laparoscopic myomectomy and found 3 uterine ruptures during subsequent pregnancies[5]. Only 1 rupture occurred at the site of the previous myomectomy scar, resulting in the conclusion that the risk of pregnancy-related uterine rupture attributable to laparoscopic myomectomy is 1% (95% CI, 0-5.5%). However, the rarity of spontaneous uterine rupture raises the question that whether the 2 uterine ruptures at sites that were not coincident with previous myomectomy scars were attributable to the previous myomectomies. If so, a markedly higher (3%) uterine rupture rate is associated with previous laparoscopic myomectomy.

In the present case , there was a solitary myoma which was removed by Laparoscopy but was repaired by laparotomy. The rupture site was close to but not exactly along the line of the scar.

Different authors reported no pregnancy-related uterine ruptures in 4 studies of 320 pregnancies in women who previously underwent laparoscopic myomectomy[6,7,8,9]. However, in all 4 studies, the number of patients who were allowed to labor was low, and a high percentage of deliveries were by scheduled cesarean delivery (80%, 79%, 75%, and 65%, respectively).

In a prospective study from Japan, there were no uterine ruptures among 59 patients with a successful vaginal delivery after a prior laparoscopic myomectomy[10]. In a multicenter study in Italy with 386 patients who achieved pregnancy after laparoscopic myomectomy, there was 1 recorded spontaneous uterine rupture at 33 weeks' gestation (rupture rate 0.26%)[11].

Uterine rupture has been reported to occur as late as 8 years after laparoscopic myomectomy[12]. This finding suggests that additional investigations with long-term follow-up are needed.

Landon et al reported a perinatal death rate from uterine rupture of 2% (2 of 124) among 19 academic centers in the United States. These studies indicate that the incidence of perinatal death associated with uterine rupture is decreasing in the modern era[13].

According to Nahum et al[1] Surgical intervention after uterine rupture in less than 10-37 minutes is essential to minimize the risk of permanent perinatal injury to the fetus. However, delivery within this time cannot always prevent severe hypoxia and metabolic acidosis in the fetus or serious neonatal consequences.

The most consistent early indicator of uterine rupture is the onset of a prolonged, persistent, and profound fetal bradycardia. Other signs and symptoms of uterine rupture, such as abdominal pain, abnormal progress in labor, and vaginal bleeding, are less consistent and less valuable than bradycardia in establishing the appropriate diagnosis.

The general guideline that labor-and-delivery suites should be able to start cesarean delivery within 20-30 minutes of a diagnosis of fetal distress is of minimal utility with respect to uterine rupture. In the case of fetal or placental extrusion through the uterine wall, irreversible fetal damage can be expected before that time; therefore, such a recommendation is of limited value in preventing major fetal and neonatal complications. However, action within this time may aid in preventing maternal exsanguination and maternal death, as long as proper supportive and resuscitation methods are available before definitive surgical intervention can be successfully initiated[1].

Our journey will be complete if we can find that the Mother has been able to give birth to alive baby in near Future.

Acknowledgement:

The authors acknowledge Dr. Radhakanta Paul MD (VIMS) for his active support.

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An Unusual Case of Joint Pain and Panuveitis in a 9 Year Old Child

Dr. Merina Mandal[1], Dr. D. K. Mukherjee[2], Dr. S. R. Pal[3], Dr. Suparna Guha[4], Dr. Sumita Basu[5], Dr. P. K. Saraf[6], Dr. P. Sahu[7], Dr. P. K. Pal[8]

Abstract:

Non-granulomatous anterior uveitis is a common entity in oligoarticular juvenile idiopathic arthritis (JIA). Presence of panuveitis in juvenile idiopathic arthritis is not so common. Presence of panuveitis with juvenile arthritis goes more in favour of one of the following - early onset sarcoidosis, Blau syndrome and infective etiologies (tuberculosis, syphilis, toxoplasma). All are uncommon differentials.

A case of a juvenile arthritis and panuveitis in an early onset sarcoidosis is being reported here.

Case Report:

A 9 years old female child was referred to our hospital with the chief complaints of itching, redness, watering from both the eyes and intermittent joint pain for the last 2 years. Two years back there was a sudden appearance of polymorphous rash all over the body which persisted for 1 month and disappeared later with some medication. It was followed by recurrent episodes of multiple joint pain involving both large & small joints like elbows, wrists, knees, ankles and joints of fingers and toes. It was associated with fever. She had ocular symptoms of itching, mild redness and watering from both the eyes, but she had no pain, diminution of vision or floaters. She had no past history of any ocular ailments, tuberculosis or jaundice.

There was no similar history in the siblings or other family members.

Her immunisation status was complete as per the U.I.P. schedule.

On examination, patient was afebrile. No lymph node was enlarged. There was no anaemia, cyanosis, jaundice, clubbing or edema. BCG vaccination scar was present.

Her visual acuity was 6/6 both eyes. Pupils were sluggishly reacting. There was muddy sclera, mild conjunctival hyperaemia and papillary reaction in palpebral conjunctiva bilaterally consistent with the features of allergic conjunctivitis. There was also bilateral symmetrical anterior non-granulomatous uveitis characterised by cells, flare, small keratic precipitates (kps) on the endothelium and central posterior synechiae. Simultaneously there was posterior uveitis which was also bilateral and symmetrical characterised by perivascular sheathing and candle wax drippings in inferior quadrants of retina. There was trace vitreous cells. No band shaped keratopathy, cataract or glaucoma was noted. Intra ocular pressure (IOP) was 14 mm Hg in both eyes.

Fig. 1: Muddy Sclera and Conjunctival

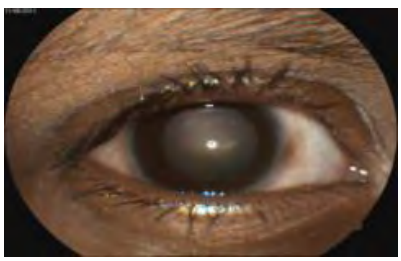

Fig. 2: Posterior Synechia of Right Eye

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Hyperaemia - Features of Allergic Conjunctivitis

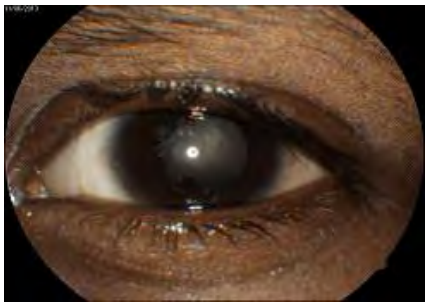

Fig. 3: Posterior Synechia of Left Eye

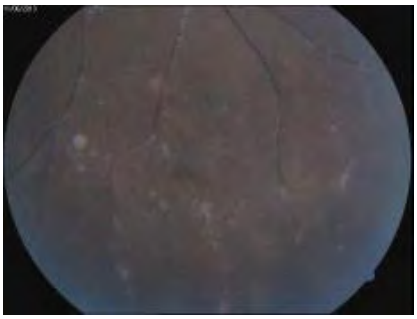

Fig. 4: Perivascular Sheathing and Candle Wax Drippings in Right Eye

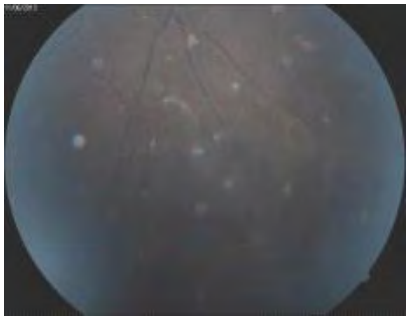

Fig. 5: Perivascular Sheathing and Candle Wax Drippings in Left Eye

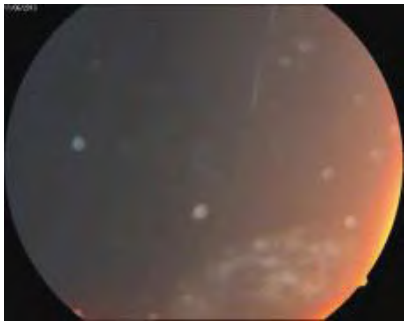

Fig. 6: Condensation Band in Vitreous

Fig. 7: Elbow Joints - Normal

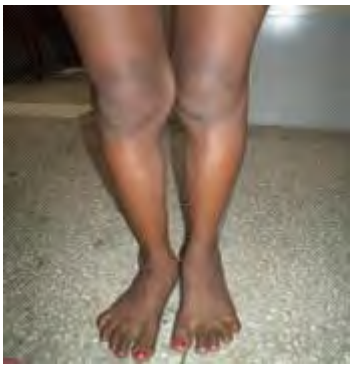

Fig. 8: Knee Joints - Normal

On systemic examination, joints of the extremities were non-tender and non- swollen at the time of presntation. Other systems were within normal limits.

From these findings differentials are - early onset sarcoidosis, Blau syndrome, juvenile idiopathic arthritis with panuveitis and infective etiologies like tuberculosis, syphilis and toxoplasma.

Complete blood count was normal. ESR was 57 mm, CRP was 12 mg/L (semiquantitative method), platelet count was 1,80,000/cmm. Though ANA was positive (50 U done by ELISA method), but Rheumatoid factor (by latex agglutination) and HLA B-27 were negative. Serum angiotensin converting enzyme (ACE) (by spectrophotometry - 55 U/L), serum calcium (10.1 mg/dl) and urinary calcium (93.1 mg/24 hrs) were normal. VDRL was non-reactive. Anti-toxoplasma IgG (<4 IU/mL) and anti-toxoplasma IgM (0.04 IU/mL) were negative. Routine and microscopic examination of urine was normal. Mantoux test was negative (done with 5 TU). Chest X-ray (PA view) did not show any hilar lymphadenopathy or parenchymal changes. X-ray knee and elbow joints were normal without any erosive changes. Ultrasound of whole abdomen showed only mild hepatomegaly but no nodular lesions suggestive of granulomatous deposits in any viscera. Liver and renal function test was normal (Serum urea - 23 mg%, serum creatinine - 0.5 mg% and SGPT - 35 U/L) excluding involvement of these organs and clearance for immunomodulator therapy. Anti-HIV I & II was done (0.06 - non-reactive) before starting methotrexate.

Patient was started on topical steriod for anterior and short course of oral steroid for posterior uveitis for 4 wks. But posterior uveitis was still persisting. Then subcutaneous inj methotrexate 10 mg (12.5 mg/M^2 body surface area) once wkly for last 2 weeks was given. Along with this tab calcium 500 mg and tab folic acid 5 mg were also prescribed as patient was on methotrexate.

Discussion:

In this case the onset of disease was at 7 years of age with rash which disappeared subsequently. It was followed by intermittent polyarticular arthralgia. It was ANA positive but Rheumatoid factor negative. There was also a panuveitis characterised by non-granulomatous anterior uveitis and posteriorly perivascular sheathing with candle wax drippings. There was no positive family history.

Juvenile idiopathic arthritis (JIA) is the most common chronic arthropathy during childhood. It is characterized by inflammation of joints and involvement of other organs. Uveitis and ANA positivity are most common in oligoarticular variety than polyarticular. Usually there is chronic non granulomatous anterior uveitis[1] and rarely panuveitis[2]. Posterior uveitis rarely complicates rheumatic diseases in children and its presence suggests the diagnosis of sarcoidosis rather than juvenile idiopathic arthritis (JIA)[3]. Joint changes in JIA are erosive[4]. Thus JIA was excluded and other causes of panuveitis were investigated.

Tuberculosis was excluded as Mantoux test was non-reactive and chest X-ray showed no parenchymal lesions. There was no history of tuberculosis in the past and no contact history of it in the family.

Syphilis was excluded as VDRL was non-reactive.

Toxoplasma was excluded as anti-toxoplasma Ig G and Ig M were negative.

Among paediatric cases, a special subtype of sarcoidosis, called the early-onset sarcoidosis (EOS) is seen with a distinct triad of skin, joint and eye disorders without pulmonary involvement[5]. A disease with same phenotypic features but autosomal dominant inheritance is known as Blau syndrome[6]. Now these two are thought to be in the same disease spectrum[3]. Blau in 1985 first reported this disease in a large 4-generation family in which 11 members had a variable constellation of granulomatous arthritis, iritis, and skin rash[7]. Usually these are seen in less than 4 years of age but may be seen later.

Originally identified as the susceptibility gene for Crohn's disease, NOD2 / CARD15 gene mutation (located on chromosome 16; 16q12) is also proved to be responsible for Blau syndrome and sporadic EOS cases[8,9]. However, it is unclear how over activation of the NOD2 protein causes the specific pattern of inflammation affecting the joints, eyes and skin that is characteristic of these disorders[10].

A granulomatous dermatitis is usually the earliest sign of EOS characterized by papulomacular rash that may persist or spontaneously disappear. Arthritis is another common feature characterised by remarkably proliferative synovitis described as ‘‘boggy’’ or cyst-like. Preservation of range of motion and absence of erosive radiographic changes are characteristic. Panuveitis (less frequently anterior uveitis) is commonly bilateral and granulomatous[3]. But it may be non-granulomatous[11,12]. Patients are generally negative for antinuclear antibodies, rheumatoid factors, and HLA-B27. Pathologic investigation of organs reveals granulomatous inflammation[13]. Mild anemia, elevated sedimentation rate, variable elevation of angiotensin-converting enzyme (ACE) levels are characteristic. Chest radiographs lack hilar lymphadenopathy and joint films show para-articular osteopenia but rarely erosions or narrowing[14].

In this case though preliminary work up for sarcoidosis including serum ACE, serum and urinary calcium was negative, but clinically presence of skin rashes, poyarthralgia and panuveitis suggested the diagnosis of early onset sarcoidosis / Blau syndrome disease spectrum. X-ray chest did not show any hilar lymphadeno-pathy. There was preservation of joint movement and X-ray knee and elbow joints suggested no erosive changes. The pattern of posterior uveitis, segmental periphlebitis and candle wax drippings, indicated sarcoidosis[15]. Uveitis is commonly granulomatous in sarcoidosis but it may be non-granulomatous type[11,12].

The diagnosis can be supported by the demonstration of noncaseating granulomas within skin, synovial or conjunctival biopsies, and the presence of CARD15/NOD2 mutations[11,16]. Biopsy can not be done in this case as rash spontaneously disappeared, there was no active inflammation of the joints during presentation in this hospital and conjunctiva was uninvolved. Gene testing was not feasible for single patient.

Sarcoid uveitis is uncommon in the Asian Indian population and its diagnosis often remains clinical[17], especially in the absence of biopsy testing.

Systemic corticosteroids are the mainstay of therapy; however, given the chronicity of this disease and the young age of onset, steroid-sparing agents such as methotrexate are often used for control of arthritis, eye disease, other systemic involvement and / or abnormal calcium metabolism[18]. Case reports of the use of TNF-

α inhibitors have thus far yielded mixed results[19]. Uveitis can be treated topically but often requires systemic therapy and surgery to treat the complication[14].

With proper treatment, the sequelae of chronic arthritis and uveitis can be lessened, but as with many chronic conditions, the side effects of long-term systemic therapies may prove as disabling as the condition itself[20]. Eye involvement represents the most severe aspect of the disorder and can appear early in childhood or in adulthood. Blindness, camptodactyly (flexion contracture of fingers and toes) are seen as late sequelae, and occurs in 30%[21].

So arthritis in juvenile age group may be associated with anterior and posterior uveitis. Examination of fundus is important to detect posterior uveitis which may suggest other differential diagnosis as discussed. Though JIA is most common, other causes including early onset sarcoidosis and Blau syndrome should be kept in mind. History & thorough systemic examination is mandatory.

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Institute News

Academic & Other Achievements

l A four day's Workshop on ENT and Head & Neck Surgery was held from 20th to 23rd March 2013, where a good number of participants from this institute and outside attended.

l A whole day rural diabetes camp was organized by the Ramakrishna Mission Seva Pratishthan in collaboration with Calcutta Diabetes and Endocrine Foundation, Kolkata on 24th March, 2013 at Kotulpur Ramakrishna Ashrama, Kotulpur, Bankura. The primary target was to see the diabetic care and management amongst the villagers of the Bankura district, surrounding Kotulpur Ramakrishna Ashrama.

Monks & Doctors at the Kotulpur Diabetes Camp

The team included 18 members of which 3 diabetologist one physician and two ophthalmologists two dietitians and ten diabetes care givers who helped in educating the patients and doing blood glucose, biothesiometry, urine test for the presence of Microalbumin and HbA1C testing. Fundoscopy was done also to screen the diabetic retinopathy. A total of 146 patients were provided with medical service during the time.

Doctors Treating the Patients

l MRCOG Part-I and II examination held here in the month of March 2013. A live hernia workshop was organized by the department of surgery on 6th & 7th April 2013, with a great number of participation from other institute.

l This year assessment has been done by the Medical Council of India regarding the recognition of the permitted post graduate degree seats on four disciplines of our institute namely Gynae & Obstetrics, Pathology, General Medicine and Anaesthesiology. The assessment was conducted along with the post graduate degree practical examination in concerned discipline in our institute, in the month of May 2013. The assessors did not make any adverse comment during the inspection process and we hope we will get the recgonition soon.

l Prof. Debashis Maji, Head, Department of Medicine got nominated as member of Research

Area Panel on Diabetes, Indian Council of Medical Research (ICMR) by the Ministry of Health and Family Welfare, Govt. of India. The first meeting of said panel was held on 20th May, 2013, Chaired by Prof. A. K. Das, Additional Directorate General of Health Services & Director, Prof. of Medicine, JIPMER, Pondichery. A decision of preparing a Registry for type 1 Diabetes patients on India was made. Prof. Uday Chaudhuri and Dr. Abhay Dey Psychiatry Unit, Dept of Medicine have attended the Annual Conference of American Psychiatry Association at Sanfransisco in May 2013. Prof. Chaudhuri has been nominated for Life Fellow by the said Association from the coming year.

l Uttarakhand Relief

A team of doctors from Ramakrishna Mission Seva Pratishthan were sent to Uttarakhand to extend voluntary services in the medical relief camp organised by Ramakrishna Mission, Belur Math. Medical services were extended to those affected by the unprecedented natural disaster in form of flash floods and landslides in the month of June 2013.

Trecking on the Way to Medical Relief Camp

Distribution of Medical Kits

Doctors Treating the Patients

Close Examination of the Patient