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PRACTITIONERS’ COLUMN

LETTER TO EDITOR

NEWS & VIEWS
PAIN MANAGEMENT SHOULD BE GIVEN PRIORITY IN CANCER TREATMENT

Most oncologists seem to underestimate and undertreat pain. Unfortunately pain and terminal cancer are not synonymous. Pain can occur at all stages of the disease. As high as 50% of patients receiving anti-cancer therapy suffer from pain. Therefore systematic pain management should be instituted simultaneously even in early stage of cancer. As the disease progresses, more number of patients need pain relief (80% with advanced disease) and the treatment should be timely shifted from a curative one to pain and symptom relief.

A total pain relief is a holistic approach that should address not only physical pain but also social, psychological, emotional and spiritual elements of pain.

The experience of pain is unique to each individual. It is very complex and modified by the patients mood, morale and as it means to the sufferer. A thorough assessment and evaluation of the patient by the doctor assisted by the multidisciplinary team is required in the proper management of the patient. For pain is often undertreated and the cause is lack of knowledge, awareness of the significance of pain management and above all the fear of narcotics causing dreaded side effects. As the thrust of advanced cancer is very high in our part due to poor screening, a clinician either in an institution or a peripheral centre should also be aware of it and rightfully guide the patient to a pain clinic or a palliative centre for its management. A good pain control not only improves the quality of life but also fosters a realistic hope amongst patient.

Morphine by mouth is the universally accepted strong opioid of choice for cancer pain and as per W.H.O. guideline should be administered ‘round the clock’ with ‘rescue doses’.

A model for decision making in the care of patients with incurable cancer includes a decision of administration of curative and symptom control management versus palliation only, evaluating cost benefit ratio and most important is when to stop palliative chemotherapy.

Pain relief improves the qualitative life of the patient and therefore it is ethically imperative upon a clinician to provide such, where and when necessary.

Rekha Das
Editor
WHY SHOULD WE DIFFER, WHY NOT UNITED ??

We, the contemporary medical practitioners, always differ, not only in our approach towards a patient, diagnosis and plan of management, but also in accepting the professional opinion delivered by any of our colleagues in a different context.

Though the science of healing is ever changing with newer knowledge and concepts pouring in, guided by research on pathogenesis of a disease and invention of newer drugs and treatment modalities for intervention and at times the decisions differ basing on experience and expertise, approach to a particular illness, the probable diagnosis, plan of investigation and first line management are standardized in a treatment protocol and many are accepted as a drug policy all over a nation/state depending on available knowledge as a part of evidence based approach to human health care. Though books may vary minimally, the scientific opinion can never be many in such an exact science and the varied propositions/postulations/presumptions/theories only prove the incompleteness of our knowledge in the domain and prompts further studies.

On the other hand in this advanced era of information technology, in a priority matter like health, no body wants to take a chance and in most of healthcare instances our expert opinions and interventional plans are crosschecked with parallel consultants by clever parties.

Politicians, beurocrats, media and other intellectuals always try to take the advantage of this professional difference in technical as well as interpersonal matters and try to create more differences among us so that we can never be united to pose a threat to them socially and professionally rather suffer alone in our respective work fields.

Professional rivalry is highest amongst us which keeps us divided and to win the confidence of a stranger patient who never belongs to anybody, we start criticizing our own colleagues sacrificing our fellow feeling. This act of scissoring, axing, pulling our own friends down, creates an opportunity for others in society to take the advantage of this difference and to keep us suppressed at personal and professional front availing all services in time.

Better fellow feeling, sharing of information and knowledge and the attitude of working together for patients' sake not only will improve our professional growth but will establish a cohesion among the healer class of society, which will be productive for greater interest of both professionals as well as sufferers.

Jayanta K Panda, M.D.
S.C.B. Medical College, Cuttack
Osteoporosis is defined as a systemic skeletal disease characterized by low bone mass and micro-architectural deterioration of bone tissue with consequent increase in bone fragility and susceptibility to fracture.\textsuperscript{1} Bone strength reflects a combination of bone density and bone quality.\textsuperscript{2} Although bone density can be measured by reliable tools available, which forms the basis for diagnosis of osteoporosis, there are no reliable measures to quantify bone quality. However, a clinically applicable index of bone quality is a history of fragility fracture. Assessment of these two parameters forms the basis of prevention and treatment of osteoporosis.

The magnitude of the problem is gradually unfolding as a result of greater awareness and availability of better gadgets for precise measurement of bone density. The reported worldwide figures could still be an underestimate. The increase in average age of the world’s population has resulted in the increase in incidence and prevalence of osteoporosis. Approximately 200m women worldwide have osteoporosis. Male osteoporosis is equally important although it develops less.

Often due to higher peak bone mass. Osteoporotic hip fracture, a major complications, is expected to rise from 1.66m to 6.26m by the year 2050.\textsuperscript{3}

With the increase in longevity of the average Indian population, it is expected that osteoporosis and associated fractures will be a major cause of morbidity and mortality in the elderly. Although good epidemiological data on osteoporosis in India is lacking, some evidence suggests that osteoporosis is prevalent, fractures are more common in Indian males than females and it occurs 10 years earlier than in the West.

A comprehensive management plan for osteoporosis includes evaluation of those at highest risk, exclusion of secondary causes of low BMD (bone mineral density) and selection of appropriate treatment. It needs to be first established that osteoporosis is primary or secondary. Primary osteoporosis is the more common form and related to age related bone loss. Post menopausal women constitute an important group within the primary disease. Secondary osteoporosis results from several diseases and use of drugs that predispose to bone loss.
Bone densitometry is the gold standard for the diagnosis of osteoporosis, and dual energy x-ray absorptiometry (DEXA) is the preferred technology for measurement. Quantitative computed tomography (QCT) and peripheral measurements are useful screening tests for assessing fracture risk but they are not validated for use as diagnostic tools for the purpose of management.

The DEXA results are interpreted based on WHO criteria as indicated in Table 1.

There are published guidelines for the use of bone densitometry and the population under high risk have been identified as -(i) post-menopausal women aged 65 years and older (ii) post menopausal women younger than 65 years with risk factors (personal history of fracture, family history of fracture, use of glucocorticoids for more than 3 months, co-morbid conditions that predispose to secondary osteoporosis), (iii) patients with fragility fracture (iv) patients with medical conditions or those taking medications associated with bone loss should be evaluated by densitometry. Recommendations for those who need treatment have been proposed by international organization based on criteria as presented in Table - 1A.

But there are several caveats that needs to be taken into account while interpreting data and planning medical treatment of osteoporosis. A substantial percentage of osteoporotic fractures occurs in women who have T score above 2.5. There is significant discrepancy between the spine and hip T scores. Osteoporosis therapy can reduce the risk of fracture by as much as 50 %, but some women have fractures despite treatment (7). Since treatment of osteoporosis is for a prolonged period of time, the cost factor, compliance and safety needs to be assessed before initiating treatment.

Therapy for postmenopausal osteoporosis is considered to be primary prevention when it is prescribed for those at risk without a T score below -2.5 or a history of fragility fracture, and is considered to be treatment for those with established disease, including previous osteoporotic fracture, markedly reduced BMD or both.

The treatment of osteoporosis involves management of osteoporosis associated fractures, universal prevention measures and medical therapy.

Non-pharmacological measures:
Calcium and Vitamin D:

Calcium and Vitamin D have been the adjunctive therapy for osteoporosis for amelioration of bone loss. Meta-analysis of 15 trials involving calcium intervention in healthy and post menopausal women with osteoporosis demonstrated an increase of nearly 2 % of BMD in spine after 2 years but the risk of vertebral and non-vertebral fracture was not reduced to a statistically significant level. A total of 1200-1500 mg per day through diet supplement is recommended for all post menopausal women. Vitamin D is essential for calcium absorption and maintenance of muscle strength which prevents fall in elderly. Supplementation of Vitamin D has been shown to reduce the risk of hip fracture and non-vertebral fracture in elderly. The daily dose recommended by the NIH is 400 to 800IU.
In a recent large study of the Women’s Health Initiative group (WHI), calcium with Vitamin D supplement did not reduce overall fracture rates except in a small subgroup of cases, but there was an increased incidence of nephrolithiasis.\textsuperscript{12}

Physical activity:

A recent Cochrane metaanalysis found that muscle strengthening, balance training and withdrawal of psychotropic medications protect against falls which results in fractures in elderly.\textsuperscript{13} Regular physical activity including aerobic, weight bearing and resistance exercise is effective in increasing BMD of the spine. But no large trials have established that these interventions reduce fracture risk.\textsuperscript{14}

Pharmacological Intervention:

There is a large body of evidence which indicates that aggressive medical therapy in osteoporotic individuals reduce risk of fracture and improve quality of life. Several pharmacological options are available and they are classified according to their mechanism of action (Table 2). The two main classes of drugs are antiresorptive agents that block bone resorption by inhibiting activity of osteoclasts and the anabolic agents that stimulate bone formation by acting primarily on the osteoblasts.

Table 3 lists agents indicating their ability to reduce fractures at different sites and recommendations for treatment.

Antiresorptive agents:

These agents suppress osteoclast activity and slows remodeling cycle enabling mineralisation of bone matrix and stabilizing trabecular architecture.

Bisphosphonates:

These agents are widely used and considered the first line therapy for treatment of post menopausal osteoporosis. They are also first line therapy in male osteoporosis and glucocorticoid induced secondary osteoporosis. These agents suppress resorption by inhibiting the attachment of osteoclasts to bone matrix and enhancing programmed cell death. Alendronate and risedronate are the two second generation nitrogen containing bisphosphonate, shown to increase BMD and reduce incidence of hip, vertebral and non-vertebral fracture by 50\% in the first year of treatment.\textsuperscript{15} The antifracture activity of biphosphonate is unrelated primarily to increase of BMD.\textsuperscript{16} The most common adverse effect is gastro-intestinal upset (20-40\%) which is dose related. Alendronate is given in doses of either 10mg daily or 70 mg weekly, Risedronate 5mg daily or 35 mg weekly. Iblandronate has been approved by the US FDA in 2005 for both prevention and treatment of osteoporosis. Dosage - 2.5 mg daily or 150mg monthly has been shown to reduce incidence of vertebral and non-vertebral fracture in women with osteoporosis.

Selective Oestrogen Receptor Modulator:

These agents inhibits bone resorption by blocking cytokine signaling to osteoclasts as do oestrogens. The commonly used agent isRaloxifene in the dose of 60 mg daily and it increases spine bone mineral density to a small degree but decrease the risk of vertebral fracture by
40% in women with osteoporosis but has no effect on the risk of nonvertebral fracture. New selective oestrogen receptor modulators are currently in phase 2 and 3 clinical trials.

Hormone replacement therapy:

This was the primary therapy for post menopausal women with osteoporosis. It has been shown to substantially increase BMD and reduce vertebral, nonvertebral and hip fractures. Presently its routine use is not recommended due to problems arising out of nonskeletal risks like DVT, cardiovascular mortality and breast cancer, and availability of safe and potent drugs.

Calcitonin:

It is an endogenous peptide that partially inhibits osteoclasts. Nasal and subcutaneous calcitonin is approved for post menopausal osteoporosis. Nasal calcitonin in a dose of 200 IU per day has been shown to reduce the incidence of vertebral fracture. It is also useful in reducing pain associated with acute vertebral fractures.

Teriparatide:

Recombinant human parathyroid hormone (1-34) is the first anabolic agent approved for the parenteral treatment of osteoporosis associated with the risk of fracture. It stimulates bone remodeling by stimulating bone formation. It reduces apoptosis of osteoblasts. It has been shown to increase BMD in lumbar spine and hip bone and reduce incidence of vertebral and nonvertebral fractures in daily dose of 20 microgram per day subcutaneously. This could be the choice of therapy in patients who cannot tolerate bisphosphonates and continue to have multiple fractures. Combination therapy:

There is currently no data to support the contention that combination therapy is superior to monotherapy in reducing the fracture rate in post menopausal women.

Strontium ranelate:

This drug has been newly introduced into the therapeutic armamentarium for treatment of osteoporosis. It acts orally and stimulates calcium uptake in bone while inhibiting bone resorption. Daily administration of 2g orally for 3 years reduces risk of vertebral fracture by over 40% in post menopausal women with osteoporosis.

Glucocorticoid induced osteoporosis:

Corticoids by any route increase bone loss. Daily prednisolone doses of 7.5 mg result in significant osteoporosis. Calcium, vitamin D supplements and weight bearing exercise programmes that maintain muscle mass are suitable first line therapies.

Bisphosphonates have produced positive effects on bone density in a number of studies. There is evidence of fracture prevention with alendronate.

Future areas of study:

Despite consensus on many issues with regard to treatment based on clinical evidence there are still areas of uncertainty. The optimal timing and type of preventive therapy are still not clearly defined as in case of post menopausal women with T scores between -1 and -2.5 with no risk factors. Also uncertain is the appropriate treatment patients who continue to have fractures despite aggressive pharmacological intervention. Much research is currently being done to develop new pharmaceuticals for the treatment of osteoporosis, particularly those with anabolic effect on the osteoblast like statins and osteoprotegerin.
### TABLE 1

**World Health Organisation Criteria for Diagnosis of Osteoporosis Based on Bone Mineral Density.**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Bone mineral Density</th>
<th>T Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Within 1 SD of reference mean*</td>
<td>-1 or above</td>
</tr>
<tr>
<td>Osteopenia</td>
<td>&gt;1 but &lt;2.5 SD below reference mean</td>
<td>Below -1 but above -2.5</td>
</tr>
<tr>
<td>Osteoporosis</td>
<td>2.5 SD below reference mean</td>
<td>-2.5 or below</td>
</tr>
<tr>
<td>Severe osteoporosis</td>
<td>2.5 SD below reference mean plus atleast 1 fragility fracture</td>
<td>-2.5 or below</td>
</tr>
</tbody>
</table>

* Reference mean based on normal values for a young adult. Data from World health Organ Tech Rep Ser 1994; 843:1-129.

### TABLE 1A

**Guidelines for treatment of postmenopausal osteoporosis**

<table>
<thead>
<tr>
<th>Organisation</th>
<th>Whom to treat</th>
</tr>
</thead>
<tbody>
<tr>
<td>National Osteoporosis Foundation</td>
<td>T score below -2.0 with no risk factor</td>
</tr>
<tr>
<td></td>
<td>T score below -1.5 with one or more risk factors</td>
</tr>
<tr>
<td></td>
<td>Any spine or hip fracture.</td>
</tr>
<tr>
<td>American Association of Clinical Endocrinology</td>
<td>T score below -2.5</td>
</tr>
<tr>
<td></td>
<td>T score below -1.5 with fractures.</td>
</tr>
</tbody>
</table>

### TABLE 2

**Drug Therapy for Osteoporosis**

- **Antiresorptives**
  1. HRT
  2. SRM
    - Raloxifene
    - Lasofoxifene
  3. Bisphosphonates
    - Alendronate
    - Risedronate
    - Ebandronate
    - Zoledronic acid
  4. Calcitonin

- **Anabolic agents:**
  1. Fluoride
  2. Synthetic parathyroid hormone (Teriparatide)

- **Agents with dual mode of action**
  1. Strontium ranelate
<table>
<thead>
<tr>
<th>Drug</th>
<th>Method of Administration &amp; Dose</th>
<th>Reduction In Risk of Fracture</th>
<th>FDA Approval</th>
</tr>
</thead>
<tbody>
<tr>
<td>Biphosphonates</td>
<td>Oral</td>
<td>Vertebral, non-vertebral &amp; hip fracture</td>
<td>For Trt &amp; prevention,</td>
</tr>
<tr>
<td>Alendronate</td>
<td>35-70 mg/wk, 5-10mg/day</td>
<td>Vertebral, non-vertebral &amp; hip fracture</td>
<td></td>
</tr>
<tr>
<td>Risendronate</td>
<td>30-35 mg/wk, 5mg/day</td>
<td>Vertebral, non-vertebral &amp; hip fracture</td>
<td></td>
</tr>
<tr>
<td>Ibandronate</td>
<td>150mg/mth,2.5mg/day</td>
<td>Vertebral fracture</td>
<td></td>
</tr>
<tr>
<td>SERM</td>
<td>Oral</td>
<td></td>
<td>For Trt &amp; prevention</td>
</tr>
<tr>
<td>Raloxifene</td>
<td>60mg/day</td>
<td>Vertebral fracture only</td>
<td></td>
</tr>
<tr>
<td>Anabolic agents</td>
<td>Subcutaneous, daily</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTH (1-34)</td>
<td>20ug</td>
<td>Vertebral and non vertebral Fracture</td>
<td>For Trt only; generally used for severe osteoporosis</td>
</tr>
<tr>
<td>(teriparatide)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcitonin</td>
<td>Subcutaneous or nasal 100-200 IU</td>
<td>Vertebral fracture only</td>
<td>For treatment only</td>
</tr>
<tr>
<td>Oestrogens</td>
<td>Oral or transdermal</td>
<td>Vertebral, non-vertebral &amp; hip fracture</td>
<td>For prevention only</td>
</tr>
</tbody>
</table>


REFERENCES :


DIABETIC CYSTOPTHAY - A MISUNDERSTOOD ENTITY

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INTRODUCTION

Diabetic Cystopathy is a common complication of long standing Diabetes Mellitus. Often this complication is not kept in mind and misunderstood. The clinical presentation and assessment of diabetic cystopathy is described along with treatment and prognosis especially in Prostatic Surgery.

REVIEW OF LITERATURE

Three decades ago ketoacidosis was the major frequent complication of diabetes mellitus. With increasingly better healthcare today long term degeneration complications are more prevalent. Diabetics patients show several kinds of complication like cardiovascular disease, neuropathy, retinopathy, nephropathy. It also affects the bladder-called diabetic cystopathy.

Cystopathy is most often mis-interpreted and younger people are sent with an ultrasound for treatment of prostatic disease.

Diabetic cystopathy is a frequent complication of DM. (45%) the prevalence rate is not related to age and sex. The prevalences increases with the duration, 25% in patient with 10 yrs of D.M. more than 50% in patients of 45 years duration.1

Studies in humans as well as animals released that diabetic cystopathy is induced by polyneuropathy that affects sensory and autonomic nerve fibres. Although the cause of neuropathy is not well understood. some of the proposed pathogenesis include altered metabolism of glucose, ischaemia, superoxide induced free radical formation and impaired axonal transport.2 Recent evidence shows one of the major mechanism that induces diabetic neuropathy is a change in the availability of neurotrophic factor such as Nerve growth factor (NGE) that is produced by target organs.

Sensory and autonomic nerve dysfunction leads to impaired sensation of bladder fullness, therefore increase bladder capacity and decreased bladder contractibility.3 Residual urine is the logical sequele. Diabetic cystopathy occurs silently and insidiously. The symptoms do not appear until the disease is in an advanced stage. Therefore secondary complications occur. The most important is urinary tract infection. Post void residue and retention leads to infection which at times is
refractory to treatment. Other complications are vesicoureteral reflux, hydroureronephrosis caused by prolonged retention, pyelonephritis, stones. At times septicaemia may occurs. Finally CRF may develop, because of high post void residue and upper tract and kidney changes in addition. In elderly male the symptoms are further complicated by prostatic disease and CVA.

Diagnosis should be made by history of neurological symptoms, neurological examination and bladder evaluation.

The bladder dysfunction is characterised by loss or impairment of bladder sensation. At time this is recognised after infection of urines. Most of ten signs of peripheral neuropathy is present (75-100%).

Functional evaluation of bladder is made by urodynamic studies like, uroflowmetry, cystometry and urethral pressure profile electromyography of sphincter muscles. Urodynamic study is essentially the pressure and flow study of bladder normally. During filling phase the pressure slowly rises and when sensation of fullness occurs there is a sharp rise of vesicle pressure; the sphincter pressure which was high during filling falls down and urine flow occurs.

In diabetic cystopathy, since the sensation is lacking the curve is flat, long and the low detrusor pressure with low peak flow, prolonged duration in uroflow meter. The residual urine is increased.

When this condition is complicated by prostatic enlargement or CVA, there may be detrusor hyperreflexia, elevated detrusor pressure but low flow of urine.

It may be emphasized here that diabetic patients taken for prostatic surgery must be assessed well for presence of lack of sensation of fullness and poor contractility of detrusor. The prognosis in this after surgery may not be that good and preoperatively the patient must be explained.

TREATMENT:

Controlling the blood glucose level is the first step. However it also must be kept in mind that control of blood glucose level does not necessarily prevent diabetic cystopathy. The prevalence rate of diabetic cystopathy in patient with oral hypoglycemic agents is 25%.

The next step is to prevent or eliminate residual urine. This can be achieved by scheduled time voiding. Sometimes cholinergic receptor agonist may help.

In severe cases intermittent catheterisation may be needed.

In patient having prostatic obstruction alfa blockers or surgery may be helpful but full understanding must be given to the patient regarding the outcome.

In diabetics it is worthwhile to have careful surveillance of voiding symptoms and elevated residual urine to prevent long term complications that are secondary to diabetic cystopathy.

REFERENCES:

"An ISO 9001-2000 Certified Hospital"

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  ★ 19" Flat Panel H.D. Monitor
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- Diagnostic & Therapeutic ERCP for treatment of
  ★ CBD/Pancreatic stones
  ★ CBD strictures / Obstructive Jaundice
- Lap. Gynecology
- Lap. Urology
- Uretero-renoscopy for Ureteric Stones/Tumors
- PCNL - for Key Hole Surgery for Kidney Stones.
- Endoscopic Urology : TURP for Prostate
  ★ TURT for Bladder tumors
  ★ OIU for Stricture Urethra
  ★ Lithotripsy for Bladder stones
- Advanced Orthopaedic & Trauma Surgery
- Spinal Surgery - Key Hole / Endoscopic Spinal Surgeries for Spondylolisthesis
- Piles : Non surgical treatment with IRC

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OSTEOPOROSIS IN POSTMENOPAUSAL WOMEN

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ABSTRACT:

Eighty menopausal women in the age range(45-73) were exposed to the study in between December 2002 to June 2003. Their Bone Mineral Density was assessed by Achill's Express Bone Ultra Densitometer. Their average BMD was calculated according to T-score by using WHO guidelines for classification into Normal, Osteopenic and Osteoporotic. Findings were statistically analysed and the results were compared with the work done by other authors. It was observed that the incident of osteopenia and osteoporosis was high in post menopausal women as compared to normal. Even though menopause is the leading cause of osteoporosis in this age group, it can be improved by slight modification of the diet and lifestyle.

Key words: Osteoporosis, BMD, Menopause, Ultrasound Bone Densitometry.

INTRODUCTION

In 1990 a panel of experts arrived at a conclusive definition of osteoporosis “As a systemic disease characterized by low bone mass and micro-architectural deterioration of the skeleton, leading to enhanced bone fragility and increased risk of fracture”¹. This definition gives two important skeletal changes in osteoporosis, namely bone mass and diminished bone quality.

Bone mineral density is the ash weight, the residual after complete dehydration represents mineral content and is proportionate to bone strength². When Photon or X-rays are passed through bone, they are attenuated by bone and the degree of attenuation is directly proportional to the ash weight density of the bone. Gradually with the advance techniques, the accuracy of measurements improved. It largely depends on the soft tissues surrounding the bone. Since less soft tissue surrounds the peripheral skeleton, the peripheral measurements are more accurate. The more accurate the device, the lower the diagnostic variability as defined by the standard deviation. Measurement done specially for peripheral include Radiographic Densitometry (for metacarpals and Calcaneus) and Ultrasound Bone densitometry.

Ultrasound Bone densitometry is a non-invasive, non-hazardous method, in which ultrasound waves when passed through calcaneus get modified. Ultrasound waves are also attenuated when transmitted through tissues. Bone mineral density is expressed in terms of Gm/ cm sq.

BMD Measurements: The speed of sound is calculated in the inbuilt computer of the machine, which gives the stiffness index of the bone. The Standard Deviation (T-score) reflects the Bone mineral density.

Osteoporosis of postmenopausal variety is defined as a skeletal disorder in
which the absolute amount of bone is decreased relative to that of younger or menstruating individuals although the remaining bone is normal in chemical composition.

Bone mass decreases as a result of bone loss which occurs normally after 35 years of age in both sexes. The rate of bone loss in men is 3% to 5% per decade. In women the bone loss before menopause is small and parallels that of men. Acceleration of bone loss occurs around menopause and averages 2% per year over the next decade. WHO (1994) created a diagnostic category of low bone mass to alert clinicians about postmenopausal ladies not receiving HRT.

More than 200 million women worldwide have osteoporosis. In India, according to the available data more than 61 million people have osteoporosis. Of these, 80% are women. There are 500,000 cases of osteoporotic fractures each year. Considering the female population, more than eighty million are above the age of 45 years; nearing the age of menopause. Of these an estimated 35% of postmenopausal women are osteoporotic. So the nation is exposed to a population who need urgent preventive strategies as visualized by Joshi et al. The life span of average Indians has increased and this also contributes to the increased incidence of osteoporosis. Recent data indicates that Indians have lower bone density than their North American & European counterparts.

In Orissa no established data is available in Osteoporosis till now. So it was worthwhile to conduct the study on menopausal women.

MATERIAL & METHODS:

Eighty menopausal women within age range 45 to 73 years were exposed to the study. The study was conducted in different urban areas of Cuttack city. Forty young women of age range between 19 to 35 years were selected as controls. Subjects with history of endocrine disorder were not included in the study.

The instrument to measure BMD was Achillis Express bone Ultradensitometer. The foot was cleaned and electrolyte gel was applied to both sides of the calcaneus before the person rested the right heel in the space provided on the platform of the instrument. Care was taken to see that the heel did not move while the ultrasound was passed and the machine gave a signal at the end of the test. After the required calculations were done inside the machine, a printed chart

![Image of BMD measurement](image-url)
came out. This instrument used transducers at fixed position to minimize errors due to contact pressure, change of position and variable heel width.

The speed of ultrasound waves gets modified when passed through calcaneus and is calculated in the inbuilt computer of the machine, which gives the stiffness index of the bone. The standard deviation of stiffness index (T-score) is reflective of the bone mineral density.

The readings found in the study group of eighty menopausal women were tabulated along with the findings of forty young women taken as controls. Comparison between the younger and the older persons was also done in the study group to evaluate the effects of age.

Our results were compared with that of studies done by different authors.

**OBSERVATIONS:**

**Table – I:**
Distribution of age in the study group

<table>
<thead>
<tr>
<th></th>
<th>Age Range</th>
<th>Total</th>
</tr>
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<tbody>
<tr>
<td>NORMAL</td>
<td>19 – 35 yrs</td>
<td>40</td>
</tr>
<tr>
<td>MENOPAUSAL</td>
<td>45 – 55 yrs</td>
<td>48</td>
</tr>
<tr>
<td>MENOPAUSAL</td>
<td>56 – 73 yrs</td>
<td>32</td>
</tr>
</tbody>
</table>

**Table – II:**
Comparison of BMD of menopausal with control group

<table>
<thead>
<tr>
<th></th>
<th>Age Range</th>
<th>T-Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>STUDY GROUP (80)</td>
<td>45 to 73 yrs</td>
<td>-1.7605 MEAN</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.4441 SD</td>
</tr>
<tr>
<td>YOUNG FEMALES (40)</td>
<td>19 to 35 yrs</td>
<td>-1.0295 MEAN</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.0002 SD</td>
</tr>
</tbody>
</table>

**Table – III:**
Comparison of BMD of early menopausal Women (45-54 yrs) with late menopausal women (55-73 yrs)

<table>
<thead>
<tr>
<th></th>
<th>Age Range</th>
<th>T-Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>EARLY MENOPAUSAL WOMEN (48)</td>
<td>45 to 54 yrs</td>
<td>-1.3295 MEAN</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.2871 SD</td>
</tr>
<tr>
<td>LATE MENOPAUSAL WOMEN (32)</td>
<td>55 to 73 yrs</td>
<td>-2.4068 MEAN</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1.4437 SD</td>
</tr>
</tbody>
</table>
Table-IV: Distribution of osteopenia and osteoporosis in premenopausal and postmenopausal groups.

<table>
<thead>
<tr>
<th></th>
<th>Premenopausal</th>
<th>Postmenopausal</th>
</tr>
</thead>
<tbody>
<tr>
<td>NORMAL</td>
<td>10 (25 %)</td>
<td>14 (17.5 %)</td>
</tr>
<tr>
<td>OSTEOPENIA</td>
<td>23 (57.5 %)</td>
<td>31 (38.75 %)</td>
</tr>
<tr>
<td>OSTEOPOROSIS</td>
<td>7 (17.5 %)</td>
<td>21 (26.25 %)</td>
</tr>
</tbody>
</table>

Table-V

Distribution of young premenopausal women according to BMI and BMD

<table>
<thead>
<tr>
<th>BMI</th>
<th>Normal</th>
<th>Osteopenia</th>
<th>Osteoporosis</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;18.5</td>
<td>1</td>
<td>5</td>
<td>nil</td>
<td>6</td>
</tr>
<tr>
<td>18.5-25</td>
<td>3</td>
<td>10</td>
<td>1 (7.14%)</td>
<td>14</td>
</tr>
<tr>
<td>&gt;25</td>
<td>6</td>
<td>8</td>
<td>6 (30%)</td>
<td>20</td>
</tr>
</tbody>
</table>

Table-VI

Distribution of early postmenopausal women according to BMI and BMD

<table>
<thead>
<tr>
<th>BMI</th>
<th>Normal</th>
<th>Osteopenia</th>
<th>Osteoporosis</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;18.5</td>
<td>nil</td>
<td>2</td>
<td>nil</td>
<td>2</td>
</tr>
<tr>
<td>18.5-25</td>
<td>4</td>
<td>10</td>
<td>4</td>
<td>18</td>
</tr>
<tr>
<td>&gt;25</td>
<td>4</td>
<td>17</td>
<td>7</td>
<td>28</td>
</tr>
</tbody>
</table>

Table-VII

Distribution of late postmenopausal women according to BMI and BMD

<table>
<thead>
<tr>
<th>BMI</th>
<th>Normal</th>
<th>Osteopenia</th>
<th>Osteoporosis</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;18.5</td>
<td>nil</td>
<td>nil</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>18.5-25</td>
<td>1</td>
<td>1</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>&gt;25</td>
<td>5</td>
<td>11</td>
<td>9</td>
<td>25</td>
</tr>
</tbody>
</table>
Table-VIII
Comparison with observations of other authors:

<table>
<thead>
<tr>
<th></th>
<th>Normal &gt; -1.0</th>
<th>Osteopenic &lt;=-1.0 And &gt;-2.5</th>
<th>Osteoporotic &lt;= -2.5</th>
</tr>
</thead>
<tbody>
<tr>
<td>STUDY GROUP</td>
<td>14 (17.5%)</td>
<td>41 (51.25%)</td>
<td>25 (31.25%)</td>
</tr>
<tr>
<td>RAO 2002 MAHARASTRA</td>
<td></td>
<td>(18.75%)</td>
<td>(33.75%)</td>
</tr>
<tr>
<td>80 CASES</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PANDE 2001 NAGPUR</td>
<td></td>
<td>(49.54%)</td>
<td>(42.2%)</td>
</tr>
<tr>
<td>109 CASES</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

DISCUSSION:

From Table – II the Standard Error (SE) of the difference between these two groups was 0.251 and z-value was –2.912. The p-value was found to be less than 0.01 which was highly significant.

Table-III compared the mean T-score between early menopausal women and late menopausal women. It was statistically analysed by applying z-test. The standard error of difference (SE) between these two groups was 0.3156 and z-value was 3.4134. The p-value was less than 0.01 which was highly significant. A peculiarity in the distribution of severe osteoporosis was evident in the study group. Severe osteoporosis cases (T-score <=-2.6) were seen more in (60-73)yrs than in age group (45-59)yrs.

The distribution of osteopenia and osteoporosis in premenopausal women shows that women suffered more from osteopenia (57.5%) as compared to postmenopausal group (38.75%) as seen in Table IV. But in postmenopausal group more women were osteoporotic (26.25%) as compared to the premenopausal group (17.5%). So decline in the level of the hormone oestrogen has definite role to play in the causation of osteoporosis after menopause.

When we tried to find out the distribution of cases according to body mass index and T-score, it was evident that women suffered more from osteopenia in premenopausal as well as postmenopausal group as evident from Table-V. Osteopenia was more marked in premenopausal group (57.5%) than postmenopausal group (51.25%).

As seen in Table-VI cases of osteoporosis was more in postmenopausal group (31.25%) than the premenopausal group (17.5%). In early postmenopausal group, the nutritional status showed that the percent of osteoporosis was high in higher body mass index and not so in late postmenopausal group.

From Table-V, Table-VI and Table-VII it was evident that incidence of osteopenia was high in all the groups of B.M.I. In early postmenopausal group, the nutritional status showed that the percent
of osteoporosis was high in higher BMI which was more marked in the late postmenopausal group. This may be related to decreased physical activities in elderly women. The elderly postmenopausal women can be advised to increase the level of physical activities if possible. Physical activities may have a two fold contribution in reducing risk of fracture. First it may enhance bone strength by optimizing BMD and improving bone quality by promoting adaptive changes in bone architecture. Secondly it has the potential to reduce the risk of accidental fall.

When findings of study group was compared with similar work done by other Indian authors incidence of osteopenia (51.25%) was more as compared to RAO of MAHARAstra (33.75%) and incidence of osteoporosis much less (31.25%) as compared to RAO (47.5%).\(^8\) When compared with the work of PANDE of NAGPur their subjects were more normal (49.54%) and less osteoporotic (8.26%).\(^9\) This was clearly evident from Table-VIII. Further study involving more number of people is required to evaluate the distribution of osteoporosis cases those who at risk of developing fracture in future.

**SUMMARY:**

In India only isolated reports of BMD is available and no centralized data is available. 61 million Indians have Osteoporosis, of them 80% are females. There is inverse relationship between BMD and risk of fractures. The complications are preventable by early detection and correction of the deficient levels of BMD by appropriate therapy and avoidance of risk factors.

Menopausal women have significantly lower bone mineral density.

**CONCLUSION:**

Bone mineral density measurement is recommended as the best approach to screen individuals for risk of developing Osteoporosis. Ultrasound bone densitometry is a non-invasive, non-hazardous method for measurement of Bone mineral Density.

**REFERENCES:**


5. Das B.K.: Osteoporosis- a silent epidemic In editorials OMJ 2003;22(2): 44


ORIGINAL ARTICLE

RECONSTRUCTIONS IN HEAD AND NECK MALIGNANCIES, A REVIEW OF 99 PATIENTS

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Anderson Cancer Center, Houston, Texas, USA

ABSTRACT:

The authors presented an observational work on the reconstruction of head and neck cancer after their curative excision, mostly oro-mandibular defects of various sizes ranging from 6cm to 12cm. The principal objective was evaluation of the various methods of such reconstruction done in order to achieve the best cosmetic and functional outcome in terms of adequate mouth opening, oral competence, deglutition of semisolid to solid foods, speech intelligibility minimum donor site morbidity and complication. The authors reported 24 reconstructions including 10 bony reconstructions. Free flap was preferred to pedicled flap for the reason of better aesthetic and functional outcome. Free fibula was mostly preferred due to its characters akin to native mandible. 75 patients were also observed in the outpatient clinic and evaluated the follow up period for aesthetic and functional aspect, donor site morbidity and long term complication. Free fibula was found to be the best option.

Key Words: Head and neck malignancy; Oromandibular cancer; Mandibular and soft tissue defects; Mandibular reconstruction; Oral lining reconstruction; Soft tissue and skin cover; Vascularized bone graft; Micro vascular free flap

INTRODUCTION

Reconstruction of head and neck defects in general and oro-mandibular defects in particular, represents a challenge to the head and neck reconstructive surgeon. The most common indication of oro-mandibular reconstruction remains ablative surgery for neoplastic disease of oral cavity and oropharynx. Other causes of oro-mandibular defects include osteoradionecrosis, trauma and congenital deformities. After mandibular resection particularly following complex radical resection for advanced oropharyngeal carcinomas invading the mandible, the restoration of form and function is paramount for rehabilitation of such patients.

This review will focus mainly on reconstruction of patients segmental mandibular defects with or without loss of inner lining, soft tissue of the neck and the outer skin in various permutations and combinations of tissue volume.

The aesthetic deformity and functional losses that occur with mandibular defects depend on the size and location of the segmental mandibular defect. In general, mandibular defect in the posterior body or ramus are better tolerated. As the defect extends to involve the symphysis or the anterior body of the mandible significant
deformity and loss of function occurs. Mastication and deglutition is compromised as structural support for the tongue and larynx is lost. Airway compromise as a result of tongue prolapsed can occur and necessitate tracheostomy. Even small defects in the posterior body or ramus of the mandible can lead to malocclusion over a period of time as the mandible shifts to the affected side\(^1\). Mandibular reconstruction is undertaken to address these significant functional and esthetic deficits the first aim in mandibular reconstruction is accurate classification of the defect and understanding the likely resultant functional deficits. Urken et al (1991) described a classification scheme for the bony mandibular defect as well as a scheme to classify the soft tissue defects that are often encountered in conjunction with mandibular defects.

Classification details as below- C-condyle, R-ramus, B-body, \(S^H\)-symphysis half, so also for the other half.\(^2\)

![Urken classification of mandibular defects](image)

Urken et al\(^2\) also emphasize accurate classification of associated soft tissue defects of the oral cavity and the oropharynx. Defects of the buccal mucosa, labial mucosa, soft palate, floor of mouth, tongue and pharynx are classified. A thorough understanding of these defects helps the reconstructive surgeon plan the soft tissue reconstruction that is often required along with bony mandibular reconstruction. Once bony and soft tissue defect is classified, the aim is to now restore the form and function. The ideal reconstruction restores the bony contour of the native mandible to minimize the aesthetic deformity. Functional considerations include restoration of mastication deglutition, articulation and maintenance of the adequate airway.

**MATERIALS AND METHODS**

The first author from Acharya Harihar Regional Cancer Center, Cuttack, India, in the department of Surgical Oncology, visited the MD Anderson Cancer Center, Houston, Texas, USA in its department of Plastic Surgery, under UICC-ICRETT fellowship and under the supervision of Professor Geoffrey L. Robb, The Chairman of Plastic Surgery made some important observations regarding various options of reconstructions in head and neck malignancies after their curative resections.

During the period of one month in the whole month of May 2006, 24 number of reconstruction of defects after curative resections for various head and neck malignancies were performed at the MD Anderson Cancer Center, by autologous tissue transfers. Watchful observations were made regarding the Age, Sex, Performance status of the patients, Site distribution of the disease, Preoperative radiation therapy status, Types of defects left over after curative resections, Size of the defects were noted, Nature of defects, Whether through and through, Type of reconstructions done, Nature of
reconstructions done. Whether bridging plates used, whether planned for Osseo integrated mandibular implants. Also observations were made regarding total Operation time and number of preoperative Blood Transfusion. Observations were made in the ICU and Postoperative wards as to the routine protocol of management, any morbidity and the number of hospital stay.

During the month, 75 patients were observed in the out patient clinics during the post operative follow up and evaluated the short term and long term functional and cosmetic outcome.

**OBSERVATIONS**

Total number of patients observed in operation room was 24. The average age was 42yrs. Table-1 shows male,female ratio.

<table>
<thead>
<tr>
<th>Serial</th>
<th>Oro-mandibular site R/B</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Oro-mandibular site S/B</td>
<td>2</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>Mucosa and soft tissue</td>
<td>9</td>
<td>5</td>
<td>14</td>
</tr>
<tr>
<td>3</td>
<td>Pre-operative irradiation received</td>
<td>4</td>
<td>2</td>
<td>6</td>
</tr>
</tbody>
</table>

Table-2 Shows no. of patients observed in the operation room taken for reconstruction(n=24)

The free fibula was planned for those 6 patients who needed only bone with or without mucosa. Whereas anterolateral thigh flap was selected for those 5 patients who needed mucosa and bulk of soft tissue. There was only 1 patient in whom Antero lateral thigh flap was planned in addition to free fibula osteocutaneous flap. It was found to be most suitable for the patient as the soft tissue bulk could be achieved with it and simultaneous harvesting was possible along with the resection surgery, so also because each of the three perforators were of equal size to the peroneal vessels. Three out of thirteen patients with requirement of mucosa and minimal soft tissue were opted for radial fore arm flap

None of the 6 preoperative irradiated patients underwent planning for Osseo integrated dental implants. It was noted that all 3 patients of poor performance group were subjected to the procedure of bridging plate and muscle wrapping over it. Out of such 3 patients, in 2 patients the wrapping of plate was with the muscle and the anterior rectus sheath from a Vascularized free rectus abdominis myocutaneous flap, in the other patient it was wrapped with a pedicled pectoralis major myocutaneous flap to minimize the operative time in view of low performance status of the patient.

Except in the patient of mucosal defect where only skin graft was put, the operating time for all other free and pedicle flaps ranged from 8-9 hours.

In none of the cases of bony reconstructions free scapula, ilium nor rib was suitable.

Table3-shows no. of patients observed in outpatient clinic(n=75)

The evaluation of free iliac and scapular flaps in term of their results was ignored as the number was too small to evaluate.

Aesthetic and functional outcome was 100% in all free flaps but 25-75% in those with pedicle flaps and the one with bridging plate reconstruction.

Adequate mouth opening, speech intelligibility and oral competence were 83-100% in the free flap patients where as it ranged from 25-75% in the group of pedicle flaps and the bridging plate. Speech intelligibility, mouth opening and oral competency were not applicable to the
trapezius pedicle flap patients as they were all extra oral head neck reconstruction.

Speech intelligibility was 83% in gracilis group which considered being very good as all of those were for reconstruction of tongue defects

The functional outcome in all 3 cases of Osseo integrated implants and prosthesis were excellent with regards to chewing quality and deglutition

Donor site morbidity was found in the both pedicle flaps and the case of Bridging plate reconstruction in the form of clinical or sub clinical muscle weakness. Fibula free flap harvest appeared to be associated with acceptable donor site morbidity and preservation of good foot and ankle function in most individuals.

There was one major long term complication in the one bridging plate reconstructed mandible after a period of 6 years and the stump of native mandible was visible and the patient was planned for a revision repair.

**Table-2**

No. of patients observed in the operation room taken for reconstruction(n=24)

<table>
<thead>
<tr>
<th>Defects</th>
<th># of Defects</th>
<th>Size of defects</th>
<th>Poor performance status</th>
<th>Free fibula</th>
<th>Combined fibula</th>
<th>ALT</th>
<th>RFA</th>
<th>Gracilis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Bony mandibular only</td>
<td>1</td>
<td>6</td>
<td></td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 Bone and mucosa lining</td>
<td>7</td>
<td>6</td>
<td>2</td>
<td>5</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 Bone, mucosa, soft tissue and skin</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 Mucosa only</td>
<td>1</td>
<td>10</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 Mucosa and soft tissue</td>
<td>13</td>
<td>13</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>24</strong></td>
<td><strong>13</strong></td>
<td><strong>2</strong></td>
<td><strong>6</strong></td>
<td><strong>1</strong></td>
<td><strong>5</strong></td>
<td><strong>3</strong></td>
<td><strong>3</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Defects</th>
<th>Bridging plates with muscle wrapping</th>
<th>Pec Major Pedicle Flap</th>
<th>Trapezius pedicle</th>
<th>Skin graft</th>
<th>Free scapula</th>
<th>Free ilium</th>
<th>Free Rib</th>
<th>Plan for osseo integrated implant</th>
<th># of Periop Blood transfusion</th>
<th>Total hospital stay in days</th>
<th>Operation time in hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Bony mandibular only</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2 Bone and mucosa lining</td>
<td></td>
<td></td>
<td>3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3 Bone, mucosa, soft tissue and skin</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4 Mucosa only</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5 Mucosa and soft tissue</td>
<td></td>
<td></td>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td></td>
<td></td>
<td><strong>3</strong></td>
<td><strong>1</strong></td>
<td><strong>1</strong></td>
<td><strong>0</strong></td>
<td><strong>0</strong></td>
<td></td>
<td></td>
<td><strong>6</strong></td>
<td><strong>14</strong></td>
</tr>
<tr>
<td>Nature of reconstructions</td>
<td># of patients</td>
<td>Mouth opening acceptable</td>
<td>Speech intelligible</td>
<td>Deglutition to solids and or semisolids</td>
<td>Fair aesthetic appearance</td>
<td>Oral competence</td>
<td>Donor site morbidity</td>
<td>Complications</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------------------------------</td>
<td>---------------</td>
<td>--------------------------</td>
<td>---------------------</td>
<td>----------------------------------------</td>
<td>--------------------------</td>
<td>-----------------</td>
<td>---------------------</td>
<td>---------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free iliac osteoseptocutaneous flap</td>
<td>1</td>
<td>1(100%)</td>
<td>1(100%)</td>
<td>1(100%)</td>
<td>1(100%)</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free scapula osteomyocutaneous flap</td>
<td>1</td>
<td>1(100%)</td>
<td>1(100%)</td>
<td>1(100%)</td>
<td>1(100%)</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free radial forearm flap-</td>
<td>11</td>
<td>10(90%)</td>
<td>10(90%)</td>
<td>9(82%)</td>
<td>11(100%)</td>
<td>11(100%)</td>
<td>Nil</td>
<td>Nil</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free antero lateral thigh flap-single padded</td>
<td>12</td>
<td>11(92%)</td>
<td>11(92%)</td>
<td>11(92%)</td>
<td>12(100%)</td>
<td>11(92%)</td>
<td>Nil</td>
<td>Nil</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free antero lateral thigh flap-double padded</td>
<td>5</td>
<td>4(80%)</td>
<td>4(80%)</td>
<td>4(80%)</td>
<td>4(80%)</td>
<td>Nil</td>
<td>Partial flap necrosis</td>
<td>one wound infection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined Free fibula and rectus abdominis free flap</td>
<td>2</td>
<td>2(100%)</td>
<td>2(100%)</td>
<td>2(100%)</td>
<td>2(100%)</td>
<td>2(100%)</td>
<td>Nil</td>
<td>Nil</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Combined Free fibula and anterolateral thigh</td>
<td>6</td>
<td>5(83%)</td>
<td>6(100%)</td>
<td>6(100%)</td>
<td>6(100%)</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Free scapular osteocutaneous flap</td>
<td>1</td>
<td>1(100%)</td>
<td>1(100%)</td>
<td>1(100%)</td>
<td>1(100%)</td>
<td>1(100%)</td>
<td>Nil</td>
<td>Nil</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bridging plate and musculoe</td>
<td>4</td>
<td>3(75%)</td>
<td>3(75%)</td>
<td>3(75%)</td>
<td>3(75%)</td>
<td>3(75%)</td>
<td>2(50%)</td>
<td>One plate extrusion</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transverse gracili free flap for tongue reconstruction</td>
<td>6</td>
<td>5(83%)</td>
<td>5(83%)</td>
<td>5(83%)</td>
<td>6(100%)</td>
<td>5(83%)</td>
<td>Nil</td>
<td>Nil</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pectoralis major pedicle flap</td>
<td>4</td>
<td>3(75%)</td>
<td>1(25%)</td>
<td>2(50%)</td>
<td>1(25%)</td>
<td>1(25%)</td>
<td>3(75%)</td>
<td>One wound infection</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trapezius pedicle flap</td>
<td>3</td>
<td>2(66.7%)</td>
<td>Not applicable</td>
<td>2(66.7%)</td>
<td>1(67%)</td>
<td>2(66.7%)</td>
<td>One wound infection</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Free fibula osteocutaneous flap</td>
<td>16</td>
<td>14(87.5%)</td>
<td>15(94%)</td>
<td>15(94%)</td>
<td>16(100%)</td>
<td>15(94%)</td>
<td>Nil</td>
<td>Nil</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Osseo integrated implants</td>
<td>3</td>
<td>3(100%)</td>
<td>3(100%)</td>
<td>3(100%)</td>
<td>3(100%)</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td></td>
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<tr>
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</tbody>
</table>

**DISCUSSION**

The current state of mandibular reconstruction is the result of an evolution in techniques the past 60 years. Refinements in microvascular technique, biomedical advances in plating technology and instrumentation, and an understanding of donor site angiosomes have made reliable mandibular reconstruction a reality. Historically, free bone grafts were frequently used for mandibular reconstructions. Autogenous bone grafts from the calvarium, rib, ilium, tibia, fibula, scapula, and radius have been used. Over the past twenty years, however, the use of Vascularized bone grafts has become the state of the art for mandibular reconstruction. The most common donor sites for osseous free tissue transfer include the fibula, scapula, ileac crest and radius. The use of reconstruction plates also remains an option in the appropriately selected patient population. Each of these options is described as follows.

**Alloplastic implants** - use of mandibular reconstruction plates is typically indicated in patients with poor performance status or in cases where the
soft tissue defect in oral cavity or oropharynx is more extensive than the bony mandibular defect. In patients in whom a mandibular continuity is restored with a reconstruction plate.

**Micro vascular reconstruction with osseous free tissue transfer** – however remains the preferred technique for mandibular reconstruction. The latest innovation in the screw and plate technology is the development of self drilling, self tapping screws and locking manipulates. Locking manipulates use double threaded screws that lock to both bone and the plate thus creating additional stability. The main advantage of primary oromandibular reconstruction using free vascularized bone –containing flaps is improved oral function from (1) maintenance of mandibular and soft tissue architecture and (2) dental rehabilitation through Osseo integrated implants. Bone dimensions, volume, quality and the ability of the bone to withstand masticatory forces are important factors in achieving successful Osseo integration. Inigo et al studied the results of three different techniques for mandibular reconstruction after hemimandibulectomy. Those were (1) full thickness galeoparietal bone flap (2) free iliac crest graft and (3) free fibular grafts. The free fibular flap demonstrated good results and good occlusion on the non operated side.

Radial forearm osteofasciocutaneous free flap is a less popular choice for mandibular reconstruction. The radius is typically suitable for small mandibular defects and more so the bone stalk is not suitable for Osseo integrated dental implants. The donor site morbidity can include limited range of motion, grip strength, and supination.

The fibula is the workhorse of modern day mandibular reconstruction. The fibula can be used to reconstruct bony defects as long as 30 cm in length. Preoperative evaluation of lower extremity vasculature is recommended to evaluate vascular disease precluding transfer. Chang et al describe successful treatment of malocclusion following fibular free flaps mandibular reconstruction by performing an osteotomies at the junction of fibula and native mandible...Munoz et al found that only one flap out of twenty six had flap necrosis. The average length of fibula graft was 10.96 cm. Disa et al concluded that the retention of fibula height seen in their study indicated that fibula bone mass was preserved after free flap mandibular reconstruction. Furthermore those findings were not affected by the site of reconstruction, patient age, length of follow up, adjuvant radiation therapy, or presence of Osseo integrated dental implants...Bhathena from Tata memorial hospital Mumbai, India described the Sliding mandibulectomy as a simple method of mandibular reconstruction.

Recently the osteocutaneous fibula flap is gaining popularity due to its developments in the surgical techniques including endosseous implants, and dental prosthetic rehabilitation which has been considered as the final goal of treatment. Therefore from the initial tumor surgery until the definite oral rehabilitation, not only maxillofacial surgeons but also an increasing number of clinicians who deal with oral implants and implant related prostodontics treatment are involved in the reconstructive treatment of oral cancer patients. They found from their study that oral functions were greatly affected by the extent of soft tissue defect caused by the tumor surgery but not by the that of bony defect. Their results suggest that
irrespective of the sizes of mandible defect, dental prosthetic rehabilitation was worth performing in cases of soft tissues defects only involving floor of mouth. For through and through large oromandibular defects which may require a large skin flap in addition to the bone and mucosal lining repair, the anterolateral thigh flap seems to be best for the following reasons: (1) its pedicle the lateral circumflex femoral system, has several major branches, A, B, C of equal size for anastomosis to the peroneal vessels. (2) As the majority of such patients with multiple previous surgery has lost recipient vessels near the mandible, the longest vascular pedicle is required. (3) There is no need for positional changes and simultaneous flap elevation with the tumor resection is possible. (4) Use of the fibula allows for reconstruction of the entire mandible, if necessary. (5) Some of the shortcomings of the individual sites for massive composite osteocutaneous flaps are minimized because each component consists of two donor sites. (6) Operating time for this flap elevation is minimized, compared to that for massive composite osteocutaneous flaps, because the individual components can be obtained simultaneously by two teams.

Vascularized bone grafts although has become the preferred method of mandible reconstruction, the technique is considered to increase both operating time and blood loss, which might be associated with an increased morbidity and mortality. Okura et al conducted a retrospective analysis of 100 cases who underwent immediate bridging plate reconstruction. They opined the use of bridging plates was an option for lateral mandible reconstruction with no history of preoperative irradiation to avoid risk from blood transfusion. But anterolateral defects and preoperative radiotherapy immersed as an independent adverse factor for plate survival.

Observations and the extensive review of literature enlightens about the wide spread use of free flaps as it carries lot of advantages over pedicle flaps or bridging plates only. As per review of literature, the fibula was the bone that matches very well to the mandible with regards to bone length, thickness, allows multiple osteotomies for bending and gets perfect angulations for the mandible reconstruct. This is followed by clavicle, scapula, ileac crest, metacarpals, radius, and rib in that order. But during our observations fibula was found to be most commonly used in 7 out of 10 mandibular reconstruction followed by bridging plate in 3 out of 10 such cases owing to poor performance status. Additional soft tissue and outer skin defects were covered by combined anterolateral thigh, radial fore arm or a gracilis myocutaneous flap in rest 14 cases. Functional outcome was much better with free flaps as they are mostly innervated ones. Tongue movement, speech and deglutition were optimum with free flaps.

**CONCLUSION**

Vascularized osseous free tissue transfer is the preferred reconstructive modality today for oromandibular reconstruction after curative surgery for head and neck malignancy. Free fibula with or without other flap combinations has shown excellent long term aesthetic and functional outcomes. Anterolateral myocutaneous flap can offer good amount of skin, mucosal lining and soft tissue for reconstruction. Gracilis myo cutaneous neurovascular repair can give a good functional tongue. However for low perfor-
mance patients, bridging plate wrapped by muscle reconstruction can be an alternative option. Refinements in technique and developments of new direction of tissue engineering in the field of Osseointegrated implants and prosthesis will offer a near normal native mandible function as well as excellent cosmetic outlook in addition to an oncological cure.

ACKNOWLEDGEMENT.
This work has been supported by a UICC International Cancer Technology Transfer Fellowship funded by the American Society of Clinical Oncology.

REFERENCES
Case Report

TRICALCIUM PHOSPHATE BONE GRAFT IN HISTIOCYTOSIS - X SCAPULA

Gurdeep S. Ratra¹, Shankar Acharya¹, A. K. Kochhar¹

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Sir Gangaram Hospital, New Delhi

INTRODUCTION

Histiocytosis-X (Langerhan's Cell Histiocytosis) is a complex of syndromes of unknown etiology involving proliferation of specialized types of histiocytes similar to Langerhan’s cell of epidermis accompanied by varying proportions of eosinophils and chronic inflammatory cells¹,². We present here, a case report of Histiocytosis-X involving the scapula where we have used Tricalcium Phosphate (TCP) graft. We are not aware of any similar case in literature using this type of graft. In small children with large defects, TCP grafts seem to be a good substitute.

CASE REPORT

A 5-year-old male child presented with pain and swelling on the posterior aspect of the left scapula since 4 months. The pain was worse at night and localized around the scapula. There was no history of trauma. He had occasional, low-grade intermittent fever.

Clinical examination revealed a large, firm, tender swelling in the infraspinous region of left scapula. Active left shoulder movements, especially abduction were painful. There was some local warmth but no muscle wasting.

Blood investigations were normal (TLC-11200; DLC-P : 73, L : 21, E : 4, M : 2) except an elevated ESR (54).

X-Ray of left shoulder showed an osteolytic lesion involving the infraspinous portion of scapula.

CT-Scan revealed an expansile lytic lesion in relation to infraspinous part of body and spine of left scapula with evidence of cortical destruction and local soft tissue involvement. The radiological differential diagnosis was eosinophilic granuloma/sarcoma.

CT guided FNAC yielded a hemorrhagic aspirate showing oval and spindle shaped cells with large oval indented nuclei with interspersed lymphocytes, polymorphs, eosinophils and large no. of histiocytic cells.

There was no evidence of granuloma, necrosis or malignancy. PAS stain showed glycogen in few cells. Picture was suggestive of Histiocytosis-X.

At surgery, large swelling involving spinous process with part of body of scapula upto the inferior margin of the glenoid was seen. Cavity of about 5 x 3 x 2 cm. was carefully curetted and yielded soft brownish granulation tissue streaked with yellow necrotic material and patches of
hemorrhage. Per-operatively sent frozen sample showed no malignant cells but was inconclusive of any specific diagnosis. After thorough curettage, the cavity was filled with chron OS (TCP) synthetic bone graft.

Post-operative X-Ray and CT-Scan suggested complete curettage of the lesion filled with TCP graft (Fig. 1).

![Post-op X-Ray showing tumor cavity filled with chron OS.](image)

**Fig. 1:** Post-op X-Ray showing tumor cavity filled with chron OS.

Final biopsy report showed a cellular lesion with histiocytes, eosinophils, multinucleated giant cells (Fig. 2). Immunohistochemistry was done, special stains were used and the final diagnosis was put as Langerhans' Cell Histiocytosis.

![Histopathology (HPE) slide of tumor tissue.](image)

**Fig. 2:** Histopathology (HPE) slide of tumor tissue.

Post-operative period remained uneventful and the patient was discharged 3rd day after the surgery.

Follow up at 4 years shows no recurrence with good graft incorporation.

**DISCUSSION**

Histiocytosis-X includes a wide range of clinical presentations from the localized and self healing eosinophilic granuloma to the potentially lethal disseminated types. Male to female ratio is 2:1 and peak age is between 5 and 15 years.

While eosinophilic granuloma is rarely extraskeletal, Letterer- Siwe disease and Hand-Schuller-Christian disease may occur in skeletal as well as extraskeletal sites. The preferred localization (60%) is in the axial skeletal, in this order: cranium, pelvis, vertebrae, ribs, mandible, clavicle, scapula. In the appendicular skeleton, proximal femur, femur shaft, humerus and tibia are involved.

The symptoms include pain and moderate swelling with occasional pathological fracture. Laboratory tests may show a slight increase in ESR and rarely a mild peripheral eosinophilia.

A solitary lesion may resolve spontaneously. The accepted treatment modalities include curettage / local injection of steroids in case of Eosinophilic granuloma while Hand-Schuller-Christian & Letterer- Siwe disease are managed by corticosteroids / vinblastine / methotrexate/ etoposide / low dose radiation therapy.

In our case, frozen sample sent per-operatively was inconclusive but had stamped the lesion as being benign, so it was decided to curette it completely and fill the cavity with synthetic bone graft. Complete curettage was confirmed by post-operative CT-Scan.

In small children, after curettage of benign tumors, the problem is ‘how to fill
the cavity? In such situations, harvesting autograft is difficult as the child is quite small and the autograft site yield may not be adequate. In such situations, allograft-freeze dried / bone bank bone is used. However, problems of allograft bone remain. Tricalcium Phosphate is a known viable bone graft substitutes. In our case, we used TCP graft as an alternative form of bone graft and got good result.

REFERENCES
Case Report

ACUTE MIXED LINEAGE LEUKAEMIA - A CASE REPORT.

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ABSTRACT:

Acute Mixed Lineage Leukemia is relatively a rare entity. The clinicopathological features in a 24 years old patient who was initially diagnosed as a case of Non Hodgkins Lymphoma and subsequently proved as a case of Acute Mixed Lineage Leukemia is presented here with due to its diagnostic dilemma.

The age old conventional methods are not sufficient to confirm the diagnosis of this disease. Immunophenotyping plays a pivotal role in further classification, diagnosis and prognostication for conflicting cases of hematological malignancy.

Key words: Mixed Lineage Leukemia, Immunophenotyping

INTRODUCTION:

Leukemia has been originally described by Virchow in 1845. The incidence of acute leukemia is less than 2% of all cancers¹. These diseases are the first and second leading causes of death due to cancer in the united states in men & women respectively, under the age of 40 years².

Acute leukemias are clonal, uncontrolled neoplastic proliferation of immature cells of the haematopoietic system characterized by aberrant or arrested differentiation, that can be broadly grouped as either lymphocytic or myelogenous and can be identified phenotypically & genetically. The acute leukemias are characterized by a rapid clinical course that usually necessitates immediate treatment. Morphology and cytochemistry are insufficient to distinguish acute myelogenous leukemia from acute lymphatic leukemia in 15-20% cases of acute leukemia³. With the addition of immunohistochemistry to recognize B cell, T cell and myeloid antigen, it is possible to diagnose most of these difficult cases.

Sometimes 15% patients of acute leukemia demonstrate lymphoid as well as myeloid antigen expression. These patients are termed as acute mixed lineage leukemia. According to Mirro et al the term acute mixed lineage leukemia has been proposed for acute leukemia in which the malignant cells simultaneously express differentiation associated characteristic of more than one cell lineage⁴.

Due to its rarity and difficulty in diagnosis with conventional methods we hereby report a case of acute mixed lineage leukemia which had been initially
diagnosed and treated as a case of Non Hodgkin’s Lymphoma.

CASE HISTORY

A 24 yrs Hindu male clinically presented with left level II and III cervical lymphadenopathy and some maculo papular rashes at left temporal area of skull as well as left shoulder region in March 2001. Histopathology of the cervical lymph node was diffuse large cell Non- Hodgkin’s Lymphoma. Routine work up did not reveal any abnormalities except splenomegaly. His peripheral smear was normal. Bone marrow aspiration cytology was not done as the patient was treated outside. Patient had received seven cycles of chemotherapy i.e combination of cyclophosphamide, adriamycin, vincristine and oral prednisolone. There was complete disappearance of enlargement of lymph nodes as well as skin lesions.

The patient was asymptomatic for 6 months after completion of chemotherapy. Subsequently he developed fever off & on, and appearance of maculo papular rashes on right leg. On clinical examination, there was no peripheral lymphadenopathy but spleen was enlarged & palpable. Radiological examination of chest was normal, ultrasonography of abdomen and pelvis revealed only splenomegaly. Bone marrow aspiration & cytology was showing only increase in mature lymphocytes. The patient was on constant followup.

In January 2003 the patient developed enlargement of left inguinal lymph nodes as well as multiple erythematous nodular lesion in left temporal area, on chest wall and abdominal wall. On clinical examination, there was persistent splenomegaly. Biopsy of inguinal lymph node as well as that of skin nodule was done. Complete blood cell count revealed the following: white blood cell count 22,000/cmm, with 43% blasts (Fig -1), hemoglobin 7.4 gm % and platelet count 32,000/cmm. Bone marrow aspiration and biopsy was also done to know the bone marrow involvement.

Histopathology report of inguinal lymphnode as well as skin nodule revealed Non- Hodgkin’s Lymphoma (Lymphoblastic type). Bone marrow biopsy also revealed involvement by high grade NHL.

Bone marrow aspiration was hypercellular, blasts were of 80% among which 15% blasts were myeloperoxidase stain positive and others were negative. Hence surface maker analysis was repeated on peripheral blood which was showing 43% blasts among which 27% was myeloperoxidase stain positive - suggestive of myeloid leukemia.
Subsequently immunohisto-chemistry of the Lymph node was done which was showing Tdt +ve, CD20 +ve in back ground lymphocyte, CD3 +ve & CD43 +ve.

Immuno histochemistry of bone marrow biopsy revealed CD 20 and CD 3 -ve.

Immuno histochemistry findings on peripheral blood was: CD 33 - 42%, Cyto CD 3 - 22%, Cyto CD 13-65%, anti-MPO - 21%, Tdt -ve.

In view of MPO, anti-MPO ,CD13, CD 33 & Cyto CD 3 positivity this case was finally diagnosed as a case of acute mixed lineage leukaemia.

Patient was advised for bone marrow transplant. Due to the nonavaibility of sibling donor he was treated with MCP-841 protocol of Acute lymphatic leukemia as there was predominance of MPO negative blasts and immunohistochemistry of lymph node was showing lymphoid markers. After completion of Induction chemotherapy, bone marrow of the patient did not show remission, hence he was offered high dose arabinoside C (ARC-C) therapy & till then the bone marrow of the patient did not enter into remission, for which patient discontinued the treatment & ultimately died in December 2003.

DISCUSSION:

Acute mixed lineage leukemia is a rare entity and due to its rarity no established treatment exist for the same. In majority of cases of leukemia FAB criteria of AML and conventional classification for ALL supplemented with appropriate cytochemistry is sufficient to diagnose and treat the concerned type of acute leukemia. Additional phenotypic data i.e surface marker analysis usually adds to confirm the diagnosis & to avoid the confliction.

Flow cytometric analysis of blood to determine the cluster determination markers (CD) is now-a-days used to distinguish the acute lymphatic leukemia and acute myelogenous leukemia from normal hemopoietic precursor cells. CD markers have high sensitivity and specificity. Patients having expression of CD markers CD13, CD15, CD33, CD34 are diagnosed as acute myelogenous leukemia. Similarly those patients who express CD markers - CD2, CD3, CD5, CD7, CD10, CD19, CD20, CD22 are diagnosed as acute lymphatic leukemia.

According to two recently reported large series, approximately 46% of acute lymphatic leukemia cases and 48% of acute myeloid leukemia cases have aberrant expression of a single antigen associated with another cell lineage, most commonly CD20 and CD7 in AML and CD33 in ALL. In present case we are having two lineage of blasts having expression of CD33, CD13 suggestive of myeloid markers in bone marrow as well as CD3, CD20 suggestive of lymphoid marker in both bone marrow and lymph node.

Uncommonly, the lineage of origin is not clear, either 2 separate blast populations, 1 myeloid and the other lymphoid, or a single blast population having immunophenotypic evidence of both myeloid and lymphoid differentiation is encountered. The new World Health Organization classification system refers to the former entity as bilineal (i.e
Mixed Lineage) acute leukemia and refers to the latter entity as biphenotypic acute leukemia, and it categorizes them as subtypes of the group “acute leukemia of ambiguous lineage.” Leukemias of ambiguous lineage are believed to be derived from marrow stem cells having the capacity for expressing antigens from more than 1 cell line. In an effort to distinguish between biphenotypic acute leukemia and those cases of ALL and AML having aberrant expression of markers of other lineage(s), the authors included an earlier scoring system by the European Group for the Immunological characterization of acute leukemias in which antigens detected by the flow cytometric immunophenotyping of cases were assigned a score of 2, 1, or 0.5 depending on the specificity of a particular antigen for myeloid, T-lymphoid, or B-lymphoid lineage (Table 1). Cases having a score greater than 2 for both the myeloid and either the T- or B-cell lines are designated biphenotypic acute leukemias.

**Table 1:**

<table>
<thead>
<tr>
<th>Score</th>
<th>B Lymphoid</th>
<th>T Lymphoid</th>
<th>Myeloid</th>
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<tbody>
<tr>
<td>2</td>
<td>Cyt CD 79a</td>
<td>CD 3 N/Cyt Anti-TCR</td>
<td>MPO</td>
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<tr>
<td></td>
<td>Cyt IgM</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cyt CD 22</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>CD 19</td>
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<td></td>
<td>CD 10</td>
<td>CD 8</td>
<td>CD 33</td>
</tr>
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<td>CD 14</td>
</tr>
<tr>
<td>0.5</td>
<td>CD 24</td>
<td>CD 7</td>
<td>CD 15</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CD 1a</td>
<td>CD 64</td>
</tr>
</tbody>
</table>

In the present case report as the patient presented with multiple cervical adenopathy, patient had undergone biopsy of the lymph node which was suggestive of Non-Hodgkin’s Lymphoma.

The patient did not undergo the bone marrow aspiration and cytology during the diagnosis as the case was dealt with a general practitioner.

Basing upon the diagnosis of Non-Hodgkin’s Lymphoma, patient was treated with chemotherapy for which he had complete disappearance of the disease. Subsequently the patient presented with inguinal lymphadenopathy and patient came to A.H. Regional Cancer Centre, Cuttack, where the biopsy of the lymph node was Non-Hodgkin’s lymphoma (lymphoblastic type). Simultaneously bone marrow aspiration and cytology revealed blast cell of 80% out of which 15% were MPO+ve. So the bone marrow picture was that of mixed population of blasts i.e lymphoblast and myeloblast. Due to this conflict in bone marrow picture the patient was subjected for immunophenotyping.

Immunohistochemistry of lymphnode was Tdt - positive, CD20 +ve in background lymphocytes, CD3 +ve & CD43+ve. Again the immunohistochemistry of bone marrow biopsy was CD20 & CD3 -ve.

Since bone marrow aspiration was hypercellular showing combination of MPO +ve and MPO -ve blast, surface marker analysis was done from peripheral blood which also revealed
combination of CD markers of myeloid series i.e CD13, CD33 and CD marker of lymphoid series i.e Cyto CD3.

So basing upon the immunohistochemical finding, as per WHO Classification the present case was confirmed to be a case of acute mixed lineage leukemia.

There is no standard guideline for the treatment of acute mixed lineage leukemia. Such patients are candidates for bone marrow transplant. In this case, as the patient was not having sibling donor for bone marrow transplant the patient was offered MCP-841 i.e treatment approach for acute lymphoblastic leukemia. Since the bone marrow was not in remission after the induction, the patient had been offered high dose of ARA-C. But still then the patient's bone marrow was not in remission which proves that the prognosis of the disease is poor and patient discontinued the treatment and died due to disease.

CONCLUSION:

Clinicopathological proved cases of Non Hodgkins Lymphoma, should be subjected to bone marrow aspiration and cytology and biopsy. Bone marrow study may also alter the diagnosis of the disease i.e leukemic transformation as well as prognosis. In conflicting situation of bone marrow pictures, patients should be subjected for immunophenotyping which further classifies the disease, by which appropriate treatment approach can be adopted.

REFERENCES

CURRENT PERSPECTIVES ON DIASTOLIC HEART FAILURE

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ABSTRACT

Heart failure is a major public health problem. Clinical studies suggest that a significant proportion of patients with heart failure have preserved ejection fraction, a clinical syndrome commonly referred to as diastolic heart failure (DHF). DHF is associated with significant morbidity and mortality and cost; however, few clinical trials focussing on DHF have been completed. Several myocardial, endocardial and pericardial diseases are responsible for giving rise to DHF. Though clinical features can help in differentiating DHF from systolic heart failure, Doppler ecocardiographic evaluation is often necessary for establishing the diagnosis of DHF. Treatment of DHF is often empirical. The therapy is directed at reducing volume overload, slowing heart rate, controlling hypertension and relieving myocardial ischemia.

INTRODUCTION

Heart failure is a principal complication of virtually all forms of heart diseases. The prevalence of heart failure increases dramatically with age, occurring in 1–2 percent of persons aged 48 to 54 years and upto 10 per cent of individuals older than 75 year. For individuals free of heart failure at 40 years of age, the remaining life time risk of developing heart failure is 21 per cent for men and 20.3 percent for women. There is a trend towards increased morbidity and mortality related to heart failure (HF). HF can be caused by systolic dysfunction leading to a defect in expulsion of blood (systolic HF) or by diastolic dysfunction leading to defect in ventricular filling (diastolic HF). This article shall attempt to review briefly the emerging concepts on aetiology, pathophysiology, diagnosis and management of diastolic heart failure (DHF).

DEFINITION OF DHF

Diastolic HF is usually defined as HF with normal systolic function or preserved ejection fraction, (EF). However, definite guidelines have been given for diagnosis of DHF that consider clinical presentation as well as documentation of systolic and diastolic function (Table-1).3

AETIOLOGY

Various myocardial, endocardial and epicardial / pericardial factors can give rise to DHF (Table-2).

PATHOPHYSIOLOGY

Diastole starts with isovolemic relaxation, which is energy - dependent process, followed by rapid ventricular filling, diastasis and finally atrial contraction.
Factors affecting either phase can contribute to DHF. Intrinsic factors causing DHF occur primarily as one of two mechanisms: (1) Impaired relaxation, (2) Increased stiffness. Extrinsic factors, such as pericardial restriction, may also cause DHF.

Ventricular diastolic relaxation, an energy dependent process, may be impaired by decreased energy availability or by changes in calcium homeostasis. ATP is required for actin-myosin cross-bridge dissociation and the reuptake of calcium into the sarcoplasmic reticulum. Conditions associated with decreased ATP availability, such as ischemia, increased diastolic calcium concentration, or a delay in the decline of diastolic calcium concentration, may impair relaxation and cause diastolic dysfunction.

Ventricular stiffness can be caused by ventricular hypertrophy. This hypertrophy is often secondary to hypertension or aortic stenosis. Disproportionate growth of the non-myocardial extracellular matrix leads to ventricular stiffness. The hypertrophied heart becomes stiff leading to raised filling pressures.

LABORATORY DIAGNOSIS OF DIASTOLIC DYSFUNCTION

The Doppler echocardiography has emerged as an important tool that provides reliable and useful data on diastolic function of the left ventricle. Measurement of transmitral flow is a convenient and effective way of assessing diastolic function. Blood flow across the mitral value occurs in 2 phases: an early transmitral flow (E-wave) and a late flow with atrial contraction (A-wave). The relative contribution of each is expressed as a ratio (E/A). In healthy young individuals, the E/A ratio exceeds 1. (Fig. -1) In delayed relaxation pattern, the E/A ratio is reversed (E/A = <1) with increased deceleration time (DT). With further disease progression, left ventricular compliance becomes reduced and filling pressure begins to rise. This results in E/A ratio exceeding 1.

(Pseudonormalisation pattern). The pseudonormalisation pattern is differentiated from the normal filling pattern by a shortened DT. In patients with severe decrease in left ventricular compliance, the left atrial pressure is markedly elevated. This results in restrictive filling pattern (E/A > 1) with a very short DT.

Analysis of pulmonary venous flow and colour M-mode Doppler echocardiography are other ancillary procedures in assessing diastolic dysfunction comprehensively.
SYSTOLIC VERSUS DIASTOLIC HF

A number of clinical features and laboratory findings characterise systolic and diastolic HF (Table 3). However, it is important to recognise that clinical features of HF may be similar whether LV systolic function is normal or depressed, underscoring the need for evaluation of both systolic and diastolic function in all patients with HF.

TREATMENT OF DIASTOLIC HF

Current strategy for the management of DHF focuses on symptomatic relief and modification of underlying causes of HF. This is because of the fact that in contrast to many clinical trials in systolic HF, there are few studies on therapy for DHF. Reduction of pulmonary and systemic congestion is done by salt restriction, diuretics, angiotensin converting enzyme inhibitors (ACEI) and angiotensin receptor blockers (ARBs). Judicious use of diuretics is essential, because excessive reduction of preload can result in a low cardiac output state. Exercise has been shown to reduce symptoms in the patients with DHF.

Targeting underlying causes such as coronary artery disease, valve abnormalities and hypertension is vital for optimal management. Tachycardia which increases myocardial oxygen demand and simultaneously decreases coronary perfusion is poorly tolerated in patients with DHF. Hence, proper rate control should be done with β-blocker and non-dihydropyridine calcium blockers (Verapamil, diltiazem).

Data support the use of ACEI and ARB in the treatment of DHF. ACEI has been shown to decrease LV hypertrophy and improve LV relaxation. In the Losartan Intervention for endpoint reduction in hypertension (LIFE) study, losartan therapy was associated with regression of LV mass and improvement of diastolic filling parameters. β-blockers are frequently used in patients with DHF because of its properties like controlling heart rate, hypertension and causing regression of LV hypertrophy.

Aldosterone inhibitors may play a role in the treatment of DHF. Because aldosterone stimulates collagen and cardiac fibrosis, blocking aldosterone may contribute to improved cardiac function. The randomised Aldactone evaluation study (RALES) has already demonstrated decreased mortality among patients with systolic HF.

CONCLUSION

Diastolic heart failure is common and accounts for more than 50% of heart failure cases among the elderly. As with systolic heart failure, DHF is associated with significant morbidity and mortality. Clinical features of HF associated with a normal left ventricular ejection points to the diagnosis of DHF. Diagnosis of definite DHF is established by echocardiographic assessment of various diastolic abnormal patterns of transmitial flow velocity. Pulmonary venous flow pattern and colour M-mode Doppler echocardiography are contributory. Maintenance of euvoilema, ventricular rate control in atrial fibrillation and management of hypertension are key elements of therapy for DHF. More randomised clinical findings are necessary to lay down definite guidelines for treatment of diastolic heart failure.
Table 1
Diagnosis of Diastolic Heart Failure

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Possible DHF</th>
<th>Probable DHF</th>
<th>Definite DHF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Definitive evidence of DHF</td>
<td>Signs and symptoms of HF, supporting laboratory tests, and response to diuretics</td>
<td>Signs, Symptoms of HF supporting laboratory tests, and response to diuretics</td>
<td>Signs and symptoms of HF, supporting, laboratory tests, and response to diuretics.</td>
</tr>
<tr>
<td>Objective evidence of normal LV systolic function</td>
<td>LVEF &gt;50% but not at time of HF event</td>
<td>LVEF &gt;50% within 72 hours of event</td>
<td>LVEF &gt; 50% within 72 hours of event</td>
</tr>
<tr>
<td>Objective evidence of LV diastolic dysfunction</td>
<td>No conclusive information</td>
<td>No conclusive information</td>
<td>Abnormal LV relaxation, filling and/or distensibility at cardiac catheterisation</td>
</tr>
</tbody>
</table>

Table 2
Causes of Diastolic Heart Failure

Myocardial
A. Impaired relaxation
   • Epicardial or microvascular ischaemia
   • Myocyte hypertrophy
   • Cardiomyopathies
   • Hypothyroidism
   • Ageing.
B) Increase passive stiffness
   • Diffuse fibrosis
   • Myocyte hypertrophy
   • Post-infarct Scarring
   • Infiltrative (Amyloidosis)
C) Endocardial
   • Fibroelastosis
   • Mitral stenosis
   • Tricuspid stenosis
D) Epicardial / Pericardial
   • Pericardial constriction
   • Pericardial tamponade
E) Coronary microcirculation
   • Capillary compression
   • Venous engorgement
F) Miscellaneous
   • Volume overload of the contralateral ventricle
   • Extrinsic compression by tumour.
### Table - 3

**Systolic Vs. Diastolic HF**

<table>
<thead>
<tr>
<th>Parameters History</th>
<th>Systolic</th>
<th>Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery</td>
<td>+++</td>
<td>++</td>
</tr>
<tr>
<td>Hypertension</td>
<td>++</td>
<td>++++</td>
</tr>
<tr>
<td>Diabetes</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>Valvular Heart disease</td>
<td>++++</td>
<td>+</td>
</tr>
<tr>
<td>Paroxysmal dyspnoea</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td><strong>Physical Examination</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Jugular vein distension</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Rales</td>
<td>++</td>
<td>+</td>
</tr>
<tr>
<td>Oedema</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td><strong>S3</strong></td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td><strong>S4</strong></td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td><strong>Chest X-ray</strong></td>
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<td></td>
</tr>
<tr>
<td>Cardiomegaly</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Pulmonary Congestion</td>
<td>+++</td>
<td>+++</td>
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<tr>
<td><strong>Echocardiogram</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Left ventricular hypertrophy</td>
<td>++</td>
<td>++++</td>
</tr>
<tr>
<td>Left Ventricular Dilatation</td>
<td>++</td>
<td></td>
</tr>
<tr>
<td>Reduced ejection fraction</td>
<td>++++</td>
<td>-</td>
</tr>
</tbody>
</table>

Plus signs indicate “suggestive” (the number reflects relative weight). Minus signs indicate “not very suggestive.”
REFERENCES


Letter to Editor

BENEFICIAL EFFECT OF WATER EXTRACT OF LEAF OF AEGLE MARMELOS (BALE) ON G.I. TRACT

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1. 4th Semistar  MBBS Student, 2. Faculty, Dept of Pharmacology, 3. Faculty Dept. of Bio Chemistry
S.C.B. Medical College, Cuttack, Orissa - 753007

INTRODUCTION:
Aegle marmelos Corr (Beng, Bael) common household plant in India. Each part of the plant has rich medicinal properties. Insulin like action of leaf of A marmelos has also been probed along with its antiinflammatory activity in rats. According to unani system Bael leaves are good appetizer and alaxitric. It is a common local practice to chew 3-4 leaves of this plant in any type of loose motion.

The present investigation has been carried out to screen the activity of A. marmelos on three different animal models.

MATERIALS & METHODS:
The study was conducted under 3 headings.
1) Effect of BALE in Rabbits - In Vitro Study:
   Effect of Graded doses of water extract of Bael leaf (BLE) on intestinal motility (small intestine) of rabbit.
   For the study, a piece of Rabbit's intestine was mounted on the isolated organ bath filled with Tyrode solution and the effect of different doses of BLE was observed on a smoked drum placed on student's kymograph.
   The effect of effective dose of BLE was also observed in presence of antagonist like atropine and Labetolol (Combined α and β blocker) to probe for the mechanism.

2. Effect of BLE in Rabbits - In Vivo Study
   The weight of the stool, its consistency and number of beads of stool of the rabbit was observed in a closed cage with graded doses of BLE administered orally with the help of a stomach tube.

3. Effect of BLE in Frogs - In Vivo Study:
   Effect of Graded doses of BLE was also observed on mucous ciliary movement of Frog's oesophagus.

RESULTS:
Effect of BLE on Intestinal Motility of Rabbit (In Vivo preparation) -
There was significant increase in the weight of the stool starting from day 3 to day 7 of experiment (P<0.05). There was also increase in fibre content of the stool but number of beads in stool was unaltered. On administration of 12ml and 14ml of stock of BLE on day 6 and day 7 respectively, two rabbits succumbed which was found out to be lethal dose. (Table - 1)

Table 1

Gross examination on abdominal opening suggested a gross dilatation of stomach and Large intestine with full gall bladder. (Fig. 1).

All the above findings suggest the intestinal relaxing feature of BLE with increase in fibre content and increase in biliary secretion.

II) Effect of BLE on oesophageal ciliary movement of Frog:
There was a significant increase in transit time of placed mustard seed in the oesophagus of frog in the presence of BLE suggesting decrease in ciliary movement by BLE. Shortened Ciliary movement time due to acetylcholine was prolonged in the presence of BLE, suggesting probable atrople like action of BLE. (Table 2)

III) Effect of BLE on Intestinal Motility of Rabbit:
There was significant decrease in number of intestinal contraction along with decrease in amplitude of contraction. There was no significant action on tone of contraction by BLE. The onset of action was prolonged compared to the known drugs like acetylcholine.

The effect of BLE on intestine was unaltered in the presence of alpha and beta blocker like Labetolol suggesting no adrenergic involvement. To add further the stimulant action of known acetylcholine was decreased in the presence of BALE, further suggesting anticholinergic mechanism of BLE on intestine. (Fig. 3)

CONCLUSION:
Thus all the above finding suggests that BLE has significant relaxant effect on intestinal smooth muscle of rabbit and it decrease the ciliary movement of frog's oesophagus. All these effect is due to atropine like principle present in BLE. Further study confirming these findings will throw light on definite role of this valuable plant on GI tract of humans.

TABLE 1

| Day of | Dose of | Wt. of Stool | No of | Other | colour |
| drug adm. | BLE | In Gm | Beads | findings |
| Day 1 | 1ml of stock | 35 ± 2.5 | 180 | Stool beads are 4-5 clusters | black |
| Day 2 | 2ml | 20 ± 1.6 | 220 | | |
| Day 3 | 3ml | 28 ± 2.2 | 180 | | |
| Day 4 | 4ml | 26 ± 1.7 | 180 | | |
| Day 5 | 5ml | 24 ± 1.8 | 180 | | |
| Day 6 | 6ml | 22 ± 2.0 | 180 | | |
| Day 7 | 7ml | 20 ± 2.2 | 180 | | |

*p value < 0.05  **p value < 0.01  ***p value < 0.001, Unpaired t test of wt of stool between-day

Table 2

<table>
<thead>
<tr>
<th>DRUGS</th>
<th>DOSE</th>
<th>Frog-1</th>
<th>Frog-2</th>
<th>Frog-3</th>
<th>Frog-4</th>
<th>Frog-5</th>
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<tbody>
<tr>
<td>N. Sine</td>
<td>1 drop</td>
<td>90 secs</td>
<td>86 secs</td>
<td>92 secs</td>
<td>90 secs</td>
<td>92 secs</td>
</tr>
<tr>
<td>Acetyl Choline</td>
<td>1 drop</td>
<td>30 secs</td>
<td>37 secs</td>
<td>36 secs</td>
<td>30 secs</td>
<td>34 secs</td>
</tr>
<tr>
<td>BLE</td>
<td>1 drop</td>
<td>120 secs</td>
<td>125 secs</td>
<td>132 secs</td>
<td>98 secs</td>
<td>90 secs</td>
</tr>
<tr>
<td>BLE + ACH</td>
<td>1 drop</td>
<td>120 secs</td>
<td>125 secs</td>
<td>132 secs</td>
<td>142 secs</td>
<td>104 secs</td>
</tr>
<tr>
<td>ATR</td>
<td>6 drops</td>
<td>130 secs</td>
<td>148 secs</td>
<td>126 secs</td>
<td>104 secs</td>
<td>120 secs</td>
</tr>
</tbody>
</table>
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**News & Views**

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### 58th Annual State Conference of Indian Medical Association, Orissa State Branch

**Date**: 10th and 11th February 2007  
**Venue**: Panthaniwas, Barkul (on the beach of Chilika Lake)  
**Hostel by**: IMA, Balugaon-Banpur Branch  

#### Registration Fee:

<table>
<thead>
<tr>
<th>Date</th>
<th>Delegate</th>
<th>Accompanying Person (Above 7 years)</th>
</tr>
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<tbody>
<tr>
<td>Upto 30.11.2006</td>
<td>300/-</td>
<td>250/-</td>
</tr>
<tr>
<td>Upto 31.01.2007</td>
<td>350/-</td>
<td>300/-</td>
</tr>
<tr>
<td>Spot*</td>
<td>400/-</td>
<td>350/-</td>
</tr>
</tbody>
</table>

(*Kits not guaranteed on Spot Registration*)  
The registration fee should be sent in the form of Bank Draft in favour of IMA, Balugaon-Banpur Branch, payable at SBI, Balugaon Branch.  
**For further details, please contact**:
- Dr. Surendranath Patra  
- Secretary Organising Committee  
- At./P.O.: Banpur  
- Dist.: Khurda - 752 031  
- Ph.: 06756-223100 (R)  
- Cell: 94372 86098

---

### 81st Annual Conference of National IMA

**Venue**: S. K. Memorial Hall, Dr. A. K. N. Sinha Nagar, Patna  
**Central Council Meeting**: 27-28 Dec. 06  
**Inauguration**: 28th Dec. 06  
**CME**: 27, 28 & 29 Dec. 06  

#### Contact:
- Organising Secretary, IMA, Bihar State Branch  
- A.K.N. Sinha Path, South East of Gandhi Maidan, Patna - 800004  
- Ph.: 0612-2321542  

The members of IMA Orissa can register for above conference at IMA House, Medical Road, Ranihat, Cuttack  

<table>
<thead>
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<th>Category</th>
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<td>Rs. 800/-</td>
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<tr>
<td>Accomp. Person</td>
<td>Rs. 600/-</td>
<td>Rs. 700/-</td>
<td>Rs.800/-</td>
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<td>Children(Below 10 Yrs.)</td>
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ACTIVITIES OF IMA STATE HEAD QUARTER

1. The 57th Annual State Conference of IMA Orissa State Branch was held on 18th and 19th February 2006 at Mayfair, Puri hosted by IMA Puri Branch.

2. The State Chapter of IMA Academy of Medical Specialities inaugurated on 19.2.2006 by Dr. R. D. Dubey, National Chairman, IMAAMS.

3. The IMA Medicon Library has already been inaugurated on 25.2.2006 by Dr. Kabi Prasad Mishra Sr. Cardiologist, Apollo Hospital, Chennai.

4. Workshop on Filaria conducted at following branches:
   1. Cuttack Branch : 19.11.2005
   2. Balasore Branch : 19.11.2005

5. Workshop on Malaria conducted at following branches:

<table>
<thead>
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<td>Malkangiri</td>
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</table>

6. The following members have attended the Master Trainers workshop on Avian Flue on 11.6.2006 at India Habitat Centre, New Delhi organized by IMA HQ.

a) Dr. M. Abbas, Cuttack as State Co-ordinator
b) Dr. Sreejoy Patnaik, Cuttack as State Secretary
c) Dr. Ashok Singhal of Sambalpur
d) Dr. Saroj Kumar Sahu, Bhubaneswar
e) Dr. Arjuna Sahu, Berhampur

7. Workshop on “Avian Flu” conducted at following branches:
   Berhampur, Bhadrak, Bhubaneswar, Cuttack, Koraput, Rourkela / Rourkela City, Sambalpur, Talcher - Angul from 15.06.2006 to 01.07.2006.

8. Workshop on HIV/AIDS organized by IMA State Branch for Training of Trainers on 29th and 30th July 2006 at Hotel New Marrion, Bhubaneswar with the help of IMA HQ./ NACO/CHAI.

9. The State Secretary, Dr. Sreejoy Patnaik and Dr. M. Abbas, Member CWC have attended 195th Central Working Committee of IMA HQ. at Hotel Hilton, Mount Abu on 20th and 21st May 2006.

10. The State Secretary, Dr. Sreejoy Patnaik and State President, Dr. Krupasindhu Panda have attended the Meeting of All State Presidents and Secretaries of IMA at India Habitat Centre, New Delhi on 30th April 2006.

11. The Hon’ble State Secretary and Dr. M. Abbas have attended two days National Workshop on RNTCP on 13th and 14th April 2006 at Hotel Hill View, Surajkund, Faridabad.
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