LOW BACKACHE – PATHOPHYSIOLOGY AND MANAGEMENT(INCLUDING FAILED BACK SYNDROME)

LOW BACKACHE

- PREVALANCE AND MAGNITUDE OF THE PROBLEM
- LBA is extremely prevalent with lifetime prevalence of 60-90%
- Annual incidence -5%
- One of the most common reason for people to seek medical attention
- Accounts for up to 15% of sick leave from work and hence a major health and economic burden

LBA-CLINICAL SYNDROMES

Myofascial syndrome
 Neural compression syndrome
 Mechanical (instability) syndrome
 Inflammatory syndrome
 Neuropathic syndrome
 Psychosocioeconomic syndrome

LBA-Classification according to presentation

> ACUTE : <6 WEEKS

Most are non specific, only 10-20% have anatomical diagnosis, 80-90% improve within 1 month

- SUBACUTE : 6 WEEKS 3 MONTHS In 10% of cases pain present beyond 6 weeks.
- CHRONIC : >3 MONTHS Only 5% have pain beyond 3 months Structural diagnosis is possible in 50% cases

LBA : ETIOLOGY

Mechanical / Musculoskeletal pain minor trauma muscle strain sprain **Degenerative spine disorders** lumbar disc herniation lumbar canal stenosis spondylolisthesis

LBA : ETIOLOGY

Non Degenerative causes

- 1. Metabolic : Osteoporosis, osteomalacia
 2. Inflammatory : Ankylosing spondylitis, Reiter's disease, psoariasis, enteropathic arthritis, fibromyositis, rheumatoid arthritis
- 3. Infectious : pyogenic, granulomatous
 - 4. Neoplastic

5. Juxtafacet cysts

LBA : ETIOLOGY

Extra spinal causes 1. Hip diseases : Trochanteric bursitis Degenerative arthritis of hip 2. Pelvic and lower abdominal diseases Endometriosis Sigmoid diverticulitis Post. wall DU **Retro peritoneal tumor Dissected aortic aneurysm**

LBA – Anatomical considerations

Pain sensitive structures of low back

1. Lumbar spine, sacrum

- 2. Sacro iliac articulations
- 3. Coccyx
- 4. Muscles, tendons, ligaments
- 5. Neural elements : cauda equina

nerve roots peripheral nerves

INNERVATION

- Posterior primary ramus innervates the vertebral and para vertebral osseo musculo ligamentous structures
- Recurrent nerve of Lushka, a branch of post primary ramus, receives sensory branches from dura, PLL, facet joint capsules, erector spinae, annulus fibrosis but not nucleus pulposus

DEGENERATIV SPINE DISEASES (DSD)

Progressive deterioration of structures of spine :

- 1. Disc abnormalities
- 2. Facet joint abnormalities
- 3. Osteophyte formation
- 4. Spondylolisthesis
- 5. Hypertrophy of ligamentum flavum

ETIOLOGY OF DSD

Cumulative effects of micro trauma / macro trauma

- > Osteoporosis
- Cigarette smoking
- > Obesity

Loss of abdominal and paraspinal muscle tone

PATHOPHYSIOLOGY OF DSD

- Pathological disc alterations
 - 1. Nuclear degeneration :
 - proteoglycan content of disc decreases with age disc dessication (loss of hydration) annular tears develop
 - 2. Nuclear prolapse : due to increased nuclear pressure under mechanical loads
 - 3. Nuclear fibrosis : due to mucoid degeneration and ingrowth of fibrous tissue
 - 4. Disc resorption
 - 5. Loss of disc space and osteophyte formation

PATHOPHYSIOLOGY OF DSD

 Concurrent changes in facet joints Synovitis
 Synovial tags in joints
 Capsular tears
 Capsular laxity
 Degeneration of articular cartilage
 Osteophyte formation and hypertrophy of articular facets and ligaments

These changes may produce spinal stenosis which can lead to neural compromise

PATHO PHYSIOLOGY OF SCIATICA

Incompletely understood ; possible mechanisms include :

Mechanical pressure on nerve root :

-edema

-altered nutrient transport

-inhibition of axonal conduction

Role of inflammation

- evidence of inflammatory cells in disc specimen removed at surgery

-presence of phospholipase A2, an inflammatory mediator in disc specimen

 Experimental application of nucleus pulposus without compression of nerve roots result in alteration of blood flow and nerve conduction velocities

LUMBAR CANAL SENOSIS

CLASSIFICATION: Depending on location : 1. central canal stenosis : decreased **AP diameter** 2. foraminal stenosis 3. lateral canal stenosis : height less than 3 mm Depending on aetiology 1. congenital 2. acquired

PATHO PHYSIOLOGY OF LCS

- Progressive narrowing of spinal canal attributed to :
- Acquired degenerative changes such as thickened laminae
- Medially impinging arthritic facets
- Infolding of hypertrophied yellow ligament
- Hyperlordosis
- Ossification of PLL

PATHO PHYSIOLOGY OF LCS

Radiculopathy and neurogenic claudication associated with LCS are attributed to

- Direct mechanical compression
- Indirect vascular insufficiency leading to decreased oxygenation of lumbar nerve

LBA : MANAGEMENT PRINCIPLES

INITIAL CLINICAL ASSESSMENT: Major Goal : To detect 'RED FLAGS' that may indicate potentially serious spinal or nonspinal pathology such as ; fracture tumor infection cauda equina syndrome

AHCPR classification of back problems > CLINICAL CATEGORY : 1. potentially serious spinal condition

2. sciatica

3. nonspecific back problems

LBA : MANAGEMENT PRINCIPLES ACUTE BACKACHE



LBA – TREATMENT PRINCIPLES > CONSERVATIVE / NON SURGICAL

> SURGICAL

> ANALGESICS

- initially use NSAIDS or acetaminophen
- Opioids: for short term period only(2-3 weeks)

- dubious role of muscle relaxants in LBA

BED REST :

Objective : to reduce symptoms by

- reducing pressure on nerve roots by decreasing intra discal pressure which is lowest in supine semi fowler position

- reducing movements which cause pain

AHCPR recommendations : Majority will not require bed rest.

-bed rest for 2-4 days may be an option for those with initial severe radicular symptoms

 prolonged bed rest (> 4 days) appears to be worse for patients by producing weakness, stiffness, increased pain

 ACTIVITY MODIFICATIONS : Risk factors : jobs requiring -heavy / repetitive lifting -asymmetrical postures -prolonged sitting / standing

GOAL : to achieve a tolerable level of discomfort

AHCPR recommendations :

- temporarily limit the risk factors

 then establish activity goals to help return to full functional status

> EXERCISE

AHCPR recommendations - use low stress aerobics during 1st month like walking cycling - after 1st month – do conditioning exercises for trunk muscles - use gradually escalating exercise grade

EDUCATION

- Explain the condition to the patient
- > Positive reassurance
- Proper posture, sleeping positions, lifting techniques

SPINAL MANIPULATION THERAPY

- useful in facet slippage with radiculopathy

 doubtful role in acute backache without radiculopathy

-use during initial 4 weeks only when 'RED FLAGS' are ruled out

EPIDURAL INJECTIONS OF CORTICOSTEROIDS

-recommended only for short term relief of radicular pain when control on oral medications is inadequate

Not recommended by AHCPR panel for acute LBA

- > Oral steroids
- > Anti depressants
- > TENS
- > Ultrasound
- Lumbar corsets
 Facet joint injections
 Acupuncture

SURGICAL TREATMENT OF LBA

> URGENT SURGERY is indicated in

- cauda equina syndrome
- progressive neurological deficits
- profound motor weakness
- rarely in intractable severe pain

SURGICAL TREATMENT OF LBA

ROUTINE SURGERY is indicated in

 4-8 weeks of symptoms, not improving with time, and with radiologically identified abnormality that correlates with findings on history and physical findings

 - <4 weeks of symptoms with potentially serious spinal conditions

SURGICAL OPTIONS OF LBA

 Central or para central PIVD

Far lateral or foraminal PIVD

LCS

 Standard discectomy; microdiscectomy; endoscopic disc excision; laser disc decompression; chemo papain

Partial or total facetectomy; endoscopic technique, extra canal approach

 Simple decompressive laminectomy; laminectomy and fusion

FAILED BACK SYNDROME

 DEFINITION : The failure of lumbar spine therapy to relieve pain and incapacitation
 Multifactorial- organic, psychological and social factors

Failure rate of lumbar discectomy is 8-25%

- INCORRECT INITIAL DIAGNOSIS (most common cause)
 - Incorrect pre op imaging
 - Clinical findings not correlated with imaging
 - Missed associated pre op conditions

eg. Trochanteric bursitis, diabetic amyotrophy, hip/knee arthropathy, myofascial pain syndrome, occult pelvic malignancy

PERSISTANT NERVE ROOT / CAUDA EQUINA COMPRESSION :

- Residual disc material

- Recurrent disc herniation at the same level / another level
 - Epidural fibrosis

 Lumbar spinal stenosis associated with midline fusion recurring over many years at the same level recurring at different levels

> SEGMENTAL INSTABILITY :

 associated with laminectomy and total facetectomy leading to spondylolisthesis

- post op scoliosis
- lateral rotational instability

- Macnab' consider 'traction spurs' as an indication of segmental instability

- Transitional syndrome ; seen in spinal fusions

- Permanent nerve root injury deafferentiation pain
- > Adhesive arachnoiditis related to sub arachnoid bleed ; best cure is prevention
- Discitis present 2-4 weeks after surgery
- Non anatomical factors :
 - -poor patient motivation
 - primary gains ; avoidance of unpleasant tasks
 - -secondary gains
 - psycho social factors

FBS-MANAGEMENT GUIDELINES

Proper clinical and radiological assessment to ascertain one of the above mentioned causes

- Rehabilitation programmes to be started early
- Pharmacological management -NSAIDS -Anti depressants
- Psycho social management

FBS-MANAGEMENT GUIDELINES

> SURGICAL MANAGEMENT :

Success rate of re operation 25-80% Indicated in 2 clinico anatomical conditions 1. Neural compressive process : LCS PIVD

2. Lumbar segmental instability

FBS-MANAGEMENT GUIDELINES



NEUROPATHIC PAIN PROCEDURES-SCS

North et.al (Neurosurgery 1993) reported that in carefully selected patients suffering from end stage FBS and treated with implantable spinal cord stimulation – 50% pain relief in long term with substantial improvement in QUALITY OF LIFE

Bees et.al (J.pain sym and management 1997) has shown better response to spinal cord stimulation than to re operation