Systemic approach to Low Back Pain and Spondyloarthritis (SpA)

Rakesh Kumar Jagdish[1,*], MK Bhatnagar[2]

[1]Assistant Professor Medicine & Incharge Rheumatology Clinic, [2]Professor, Dept. of Medicine, Santosh Medical College & Hospital, Ghaziabad

***Corresponding Author:**
Email: dr.rkj.kapil@gmail.com

Abstract

Low back pain is most common outpatient presentation just next to common cold and leading cause of loss of productivity, morbidity and disability. There are many causes for LBA, as it is a symptom only, not a diagnosis, so it is referred as "illness in search of a disease". Over 50%of all patients with low back pain recovers within a week time while 90%at one month without any intervention. Recurrence is quite common about 40%cases mostly within six months. It can be acute (pain less than 3 month duration), or chronic (pain more than 3 months duration), need consideration of "red and yellow flags". Broadly it can be classified as non inflammatory(mechanical) type and inflammatory type(spondyloarthropathy). Non inflammatory causes are most common (90%) but understanding and appropriate management need to be done for inflammatory type (SpA), as it is a major cause for preventable deformities and various systemic inflammatory complications. SpA clinically has 3 components-1.Axial SpA, 2.Peripheral SpA, 3.Extra-articular manifestations of SpA. Careful understanding and accurate diagnosis needs to be done for its appropriate management and avoidance of unnecessary blood and radiological testing and finical burden on patients. Multidisciplinary approach is most appropriate in management and early recovery.

Keywords: Low back Ache (LBA), mechanical low back pain, Red flags, Yellow flags, Spondyloarthropathy (SpA), ASAS criteria, Non radiographic SpA.

Introduction

Lower Back Pain is a broad clinical term; it is a symptom, not a diagnosis of specific disease. There are a variety of causes – the most common cause is mechanical back pain due to muscle, tendon, or ligamentous strain. Several structures in the back, including the joints, discs and connective tissues, may contribute to symptoms.

Low back pain is the second most common cause of absence from the work place among people younger than 55 years, second only to the common cold,[(1)] with about 90% of people suffering from it at some point in their lives.[(2)] In many countries, chronic low back pain is the most common cause of long-term disability in middle age.[(3)] Low back pain is a major public health problem, not only because of the high prevalence and incidence of low back problems but also because of the important consequences which are disability, the use of health services, sickness absence, early retirement[(4)] and lost working days[(5,6)] especially in the information technology (IT) and BPO sectors, in 80% of patients sitting for more than 8 hours a day in front of computer.[(7)] In up to 85% of individuals, no specific cause can be isolated. In a recent study,[(8)] 79% of patients with acute low back pain will see one physician and 21% will see multiple physicians including: chiropractors, orthopaedic surgeons, neurosurgeons, physiatrists, rheumatologists, and physical therapists. By the 3-month interval of continued low back pain, 54% of patients will seek out multiple healthcare specialists.[(9)]

Classification

1. According to duration-Acute/chronic
2. According to character of pain
3. Inflammatory verses non inflammatory (mechanical)

Acute Low Back Pain

It is defined as pain between bottom of rib cage and inferior gluteal folds with no serious underlying pathology. Some patients may experience pain in upper legs with back pain. As such, acute back pain is often referred to as mechanical low back pain, duration is usually less than 4 weeks, and term sub acute used in some studies for duration of less than 12 weeks.[(10)] Acute back pain frequently recurs and resolves on its own, regardless of treatment.

Specific causes account for less than 20% of cases of back pain: the probability that a particular case of back pain has a specific cause is only 0.2%.[(11)] Work-up for a patient with acute low back pain should focus on ruling out more serious pathology by screening for "**red flags"**[(12,13)] as follows.

Red flags for potentially serious conditions in acute LBP		
From history		Suggestive of
1	Age over 50 or under 20 yrs	Tumour /infection
2	The pain is constant, without exacerbating and remitting factors and getting worse.	Tumour /infection
3	Constitutional symptoms- Fever and/or weight loss.	Tumour /infection
4	Steroid use, IV drug use, risk of UTI and/or immuno-suppressed.	Infection
5	History of cancer	Metastatic disease
6	Pain is worse at night or when supine	Malignant
7	Major trauma such as accident or fall from height	Fracture/NV injury
8	Minor trauma or strenuous lifting in older or potentially osteoporotic patient	Fracture/NV injury
9	Neurological signs such as weakness, numbness, saddle anaesthesia or bowel/bladder incontinence	Cauda equina syndrome
10	Bilateral progressive neurological deficit covering several dermatomes	Cauda equina syndrome
From examination		Suggestive of
1	Unexplained fever	Infection/inflammatory arthritis
2	Unexplained weight loss	Infection/Malignancy
3	Percussion tenderness over the spine	Infection/Malignancy
4	Abdominal, rectal, or pelvic mass	Malignancy
5	Patrick's sign(FABER) or heel percussion sign	Inflammatory arthritis
6	Straight leg or reverse straight leg–raising signs	Disc prolapsed
7	Progressive focal neurologic deficit	Cauda equina syndrome
8	Hyperreflexia with upgoing toes	Myelopathy

In the absence of Red Flags, treatment for acute LBP is conservative.

Chronic back pain

Chronic back pain is low back pain of unspecified pathology that persists longer than 3 months.[14] It is not acute back pain with an extended duration.[15] The evolution of chronic LBP is complex, with physiological, psychological, and psychosocial influences. Depression is often associated with chronic back pain but malingering is uncommon. Work-up for chronic back pain (and acute LBP) should include a screen for "**yellow flags**"[16] that indicate risk for chronic disability.

Yellow flags for potential risk for chronic Low back pain(LBP)			
Physical /Clinical		**Psychological**	
1	SpA features –enthesitis, restriction of spinal movements, dactylitis etc	1	A belief that back pain is harmful or potentially severely disabling
2	Hamstring tightness	2	Withdrawal from social interaction/ divorced/ widowed/ no children
3	Prolonged sitting posture	3	Taking prolonged bed rest for back pain.
4	Previous history of LBP	4	Overprotective family or lack of support
5	High Oswestry score	5	Poor job satisfaction, unemployed
6	Increased age	6	Less educated, less seniority, low wages
7	Female gender	7	Fear-avoidance behaviour (avoiding a movement or activity due to misplaced anticipation of pain) and reduced activity.

Pearl: No rest: An important treatment goal for both acute and chronic back pain is for the patient to be active as soon as possible, if at all required then not for more than 24-48 hours. Bed rest is not useful, it may be harmful, leading to chronicity.[17]

According to character/nature of low back pain

a. **Local pain** is caused by injury/ trauma to pain-sensitive structures that compress or irritate sensory nerve endings. The site of the pain is near the affected part of the back.
b. **Referred pain** may arise from abdominal or pelvic viscera.
c. **Spine origin pain** may be located in the back or referred to the buttocks or legs. Referred pain may explain instances where the pain crosses multiple dermatomes without evidence of nerve root compression.
d. **Radicular** back pain is typically sharp and radiates from the low back to a leg within the territory of a nerve root. Exacerbated by coughing, sneezing, or voluntary contraction of abdominal muscles. The description of the pain alone often fails to distinguish between referred pain and radiculopathy.
e. **Muscle spasm associated pain** is commonly associated with many spine disorders. The spasms are accompanied by poor sitting posture, frequent forward bending, heavy lifting, tense paraspinal muscles, and dull or achy pain in the paraspinal region.
f. **Logical/illogical pain**: Some patients involved in accidents or work-related injuries may **exaggerate** their pain for the purpose of compensation or for psychological reasons.

Inflammatory verses non inflammatory pain

Points	Inflamatory	Non-Inflamatory
EMS-early morning stiffness	More than 30 min	Less than 30 min
Activity	Decrease with activity	Increase with activity
Rest	Pain more at rest	Decrease with rest
Association	SpA features may present	Non specific mostly
Diurnal variation	Worse in early morning	Worse in evening
Constitutional features	Usually present	Usually absent
Radiographs	Sacroilitis, Vertebral ankylosis, syndesmophytes	Osteophytes, malalignment
Example	SpA,RA	Mechanical LBA

History

1	ODP(origin, duration, progression),Aggravating/Reliving factor/severity/intensity/quality of pain
2	Inflammatory/Mechanical type
3	Red flag-acute pain/bowel/bladder/planter/power
4	Yellow flag-chronic-psychological features
5	VAS-0 to 10,intial and follow up visit
6	Character/nature of pain
7	Medication received earlier if any
8	Functional limitation if any

History includes the onset, intensity, duration, quality, frequency, radiation, severity, associated symptoms, and aggravating and reliving factors. It is important to ascertain whether the pain is chronic or acute because the diagnosis and treatment may vary. **The patient should be screened and treated for depression, especially if the pain is chronic**. Bowel and bladder incontinence should be explored because it could indicate a significant neurologic disorder such as Cauda equina syndrome. One should document the patient's current functional level and the level at which the patient is pain free. Finally, the goals of the patient are extremely important, such as walking, sleeping, or even competitive running.

Several pain assessment tools have been established to objectify pain. These tools include the Visual Analog Scale,[18] McGill Pain Questionnaire (MPQ), Short-Form McGill Pain Questionnaire (SF-MPQ), Western Ontario and McMaster Universities Osteoarthritis Index, and various behavioural and physiologic testing instruments.[19]

The Visual Analog Scale is probably the most used and simple tool for pain evaluation. It is a simple analog scale that measures pain perception on a scale from 0 to 10, where "0" is graded as "no pain" and "10" is graded "as bad as it gets." Patients place a mark between the 0 and 10 (100-mm) line that corresponds to the level of pain they are experiencing. Studies show that a change of 13 mm or more can be considered statistically important and clinically relevant.[18] The shortcoming of the Visual Analog Scale is that it does not account for function, depression, or different types of pain symptoms.

History Pearls: Red and Yellow flags need to be considered.

Physical Examination: The physical examination begins with examination of the musculoskeletal, osteopathic systems neuromuscular and vascular system.

Musculoskeletal Examination: The patient should be evaluated **sitting, standing, supine, and prone**.

The normal **spine** has a cervical and lumbar lordosis, and a thoracic kyphosis. Exaggeration of these normal alignments may result in hyperkyphosis of the thoracic spine or hyperlordosis of the lumbar spine. Inspection may reveal a lateral curvature of the spine (scoliosis) or an asymmetry in the prominence of the paraspinal muscles, suggesting muscle spasm. Obesity that results in a heavy paunch and pregnancy in its later stages, can, however, distort the curvature of the spine and result in back pain. In the case of pregnancy, the pain usually ameliorates once the child is delivered.

Inspection	Peripheral arthritis for SpA, psoriatic skin changes /nail changes, acne(SAPHO), café-au-lait spots (neurofibromatosis), hairy patches (Spina bifida), abdominal fullness for referred pain
Feel	Bone pain(osteoporosis), generalised myelgia(vitamin D deficiency) spinous process tenderness (fracture, tumor, infection), SI joint pain (ankylosing spondylitis), chest expansion <2.5cm (ankylosing spondylitis), step at L5 (spondylolisthesis),renal angle tenderness(pyelonephritis)
Move:	BJHS(hypermobile joints), pain on bending toward affected side, or on flexion (Lumbar disc disease), pain on extension (Facet joint or spinal stenosis), range of motion of involved joints

Rheumatological –GALS Screen	
Gait	Gait and stance of patients
Arms	Shoulder/elbow/wrist/hand/nail and muscular examination
Legs	Hip/knee/ankle/feet/nail and muscular examination
Spine	Examination of spine and various special tests
Skin	Head to toe skin and general examination, including eyes, ear, oral cavity to look for any clue of extra articular rheumatological menifestations

Special Tests

- **FABER test (Patrick's test)** - consisting of flexion, abduction, and external rotation of the hip, can evaluate both hip and sacroiliac dysfunction.
- **FAIR test- Piriformis syndrome** is a compression of the sciatic nerve as it courses under or through the piriformis muscle. Patients complain of gluteal and hip pain that is exacerbated with flexion, adduction, and internal rotation of the hip(**FAIR**), that is, piriformis stretch.
- **Schober's Test**: assesses the amount of lumbar flexion. Make two pen-marks, one at 10cm above the PSIS, the other 5cm below it. Upon flexion, the distance should increase >5cm. Decreased ROM of lumbar spine suggests ankylosing spondylitis.
- **Straight Leg Raise (SLR) test:** performed with the patient supine and the lower extremity slowly raised. With a positive test, radicular symptoms will be present at less than 70 degrees of flexion, indicating irritation of the sciatic nerve.[21] Studies show that the test is very sensitive to lumbar disk herniation but not highly specific.[22] Nonneurologic etiologies of a positive SLR (False positive) include tight hamstrings, muscle spasms, and sprained posterior longitudinal ligament sprain.
- **Lasegue's test** involves dorsiflexing the foot during the SLR.
- The **crossed SLR sign** is positive when flexion of one leg reproduces the usual pain in the opposite leg or buttocks. The crossed SLR sign is less sensitive but more specific for disk herniation than the SLR sign. The nerve or nerve root lesion is always on the side of the pain.
- The **reverse SLR sign** is elicited by standing the patient next to the examination table and passively extending each leg with the knee fully extended. This maneuver, which stretches the L2-L4 nerve roots, lumbosacral plexus, and femoral nerve, is considered positive if the patient's usual back or limb pain is reproduced.

Neuro-Musculoskeletal Examination: Complete a thorough **neurological exam (UMN and LMN),** including gait, ankle reflex (S1), knee reflex (L4), power, sensation (look for saddle anesthesia and anal sphincter tone, plus check dermatomes along lower limb).

Neurolologic- sign	**UMN**	**LMN**
Nutrition	Wasting is minimal, due to disuse atrophy	Atrophy and wasting are cardinal features
Muscle tone and spasticity	Clasp knife spasticity(hypertonia)	Facidity (hypotonia)
Muscle power testing	Paralysis of voluntary muscle groups	Paralysis of individual muscle supplied by segment or nerve
Sensory dermatomes and peripheral nerve testing	-see table below-	-
Deep tendon reflexes	Brisk	Lost

Hoffmann's sign and Babinski's reflex(planter)	Present	Not present

When evaluating low back pain, the superficial cremasteric reflex and the superficial anal reflex can indicate a significant upper motor neuron lesion corresponding to L1, L2 and S2, S3, S4, respectively.[20] The finding of an unexplained upper motor neuron pathologic process on physical examination should be immediately correlated with head and spine magnetic resonance imaging (MRI) or computed tomography (CT) scans. Ataxia, tremor, and dysmetria will be present if a pathologic process exists in the cerebellum or posterior fossa, useful particularly when patient has cervical symptoms also.

	Examination Findings- Lumbosacral Radiculopathy			
Lumbosacral Nerve Roots	**Reflex**	**Sensory**	**Motor**	**Pain Distribution**
L2	—	Upper anterior thigh	Psoas (hip flexion)	Anterior thigh
L3	—	Lower anterior thigh Anterior knee	Psoas (hip flexion) Quadriceps (knee extension) Thigh adduction	Anterior thigh, knee
L4	Knee reflex	Medial calf	Quadriceps (knee extension) Thigh adduction Tibialis anterior (foot dorsiflexion)	Knee, medial calf Anterolateral thigh
L5	Medial hamstring reflex	Dorsal surface—foot Lateral calf	Peroneii (foot eversion)Tibialis anterior (foot dorsiflexion) Gluteus medius (hip abduction) Toe dorsiflexors	Lateral calf, dorsal foot, posterolateral thigh, buttocks
S1	Ankle reflex	Plantar surface—foot Lateral aspect—foot	Gastrocnemius/soleus (foot plantar flexion) Abductor hallucis (toe flexors) Gluteus maximus (hip extension)	Bottom foot, posterior calf, posterior thigh, buttocks

Vascular System

Palpate all peripheral arterial pulses	**Palpation of the pulses in the lower extremity should be a routine part of the evaluation of the patient with low back pain.**
Evaluation for claudication	Vascular claudication presents as bilateral leg pain that begins at a fixed distance when ambulating and is relieved by standing. Pseudoclaudication, or neurogenic claudication (associated with spinal stenosis), is relieved only by sitting or forward flexion of the lumbar spine.[21]
Detection of any ischemic changes in the lower extremity.	

Examination Pearl

1. Examination of **abdomen and rectum** in the same setting while doing SLR/planter testing ,while patient is still supine, for assessment of referred pain, eg [pancreatitis, abdominal aortic aneurysm (AAA)] or percussion over the costovertebral angles (pyelonephritis) do abdominal
2. In a patient with simple mechanical back pain, without symptoms of nerve root compression and no reason to consider recurrent malignancy, osteoporosis, or HIV infection, the yield from clinical examination is low. However, the history and physical exam are essential to determine the presence of any red flags.

Differential diagnosis of back pain

Differential Diagnosis of Back Pain		
Neurologic		**Short description**
Upper motor neuron lesions		
1	Metabolic	Global deep tendon hyperreflexia and upper motor neuron findings. Syndromes include a thyroid pathologic lesion, electrolyte imbalances, and drug toxicities
2	Space-occupying lesions	History of cancer.

3	Parkinson's disease	May present with low back pain and leg pain. Physical findings are increased tone and shuffling gait.
4	Cerebrovascular accident	Due to increased tone or even pain syndromes such as complex regional pain syndromes(CRPS) or hand-shoulder syndrome
5	Central disk herniation	Can produce myelopathy
6	Myelopathy	Myelopathy, both cervical and lumbar, can result from central disk herniation and severe spinal stenosis or chronic disease processes such as the human immunodeficiency virus (HIV) infection
7	Congenital: Spina bifida	Hairy back
Lower motor neuron		
1	Disk herniation: posterolateral	Disk herniation is a common cause of sciatica; however, not all disk herniations result in a painful process. Intervertebral foraminal narrowing/ Disk-Osteophytes complex/ Internal disk disruption/ LSS with neurogenic claudication/ Uncovertebral joint disease
2	Neuropathy	Especially diabetic neuropathy can present with back pain
3	Lumbar foraminal stenosis	Spinal stenosis is a narrowing of the spinal canal or foramen (or both). Patients with central canal stenosis will have bilateral neurologic symptoms, whereas patients with foraminal stenosis will have unilateral symptoms.
4	Myopathy	Myopathy is an abnormal breakdown of muscle and may present as pain involving the back and proximal region of the leg. It is usually bilateral and involves the proximal muscle groups.
Mixed upper and lower motor neuron disease		
1	Cauda equina syndrome	Real surgical emergency
2	Multiple sclerosis	Can present with back pain ,other signs of MS can be there
3	Lumbar spinal stenosis	Patients with central canal stenosis will have bilateral neurologic symptoms,
Vascular		
1	Claudication	Fixed distance limb pain
2	AAA(abdominal aortic aneurysm)	Palpate all pulses and look for bruit also
3	Osteonecrosis	Hip and knee joints are common sites
Rheumatological		
1	Spondyloarthropathy(SpA)	SpA clinically has 3 components- 1. Axial SpA, 2.Peripheral SpA, 3.Extra-articular manifestations of SpA**. For details see text**
2	DISH (Diffuse idiopathic skeletal hyperostosis)	Hypercalcification of the anterior and posterior longitudinal ligament and is found in men with more than 50 years of age with metabolic syndrome component. X-ray films will demonstrate large bone spurs and thick syndesmophytes that fuse regions of the spine, like candle wax flowing. Patients complain of diffuse, nonradicular back pain.
3	SAPHO	**S**ynovitis, **A**cne, **P**ustolosis, **H**yperostosis, **O**steitis, (Pustular Skin Disease + Osteitis or Arthritis), Arthritis of Amphiarthroses (Saddle joints): AC (AcromioClavicular), SC (SternoClavicular), MS (ManubrioSternal) Joints.
4	Ocronosis/ Alkaptonuria	Deficiency of enzyme homogentisate 1, 2 dioxygenase(HGO). Triad of dark urine on addition of alkali,ocronotic pigmentation at axilla/pinna/sclera/other area and arthritis of spine and large joints. Intervertiveral disc calcification, hand and feet spared
5	BJHS(Benign joint hypermobility syndrome)	BJHS can present with variable features articular/ periarticular/ extraarticular. Many patients with BJHS can present with low back pain, usually after prolonged sitting or standing, it can be associated with with spondylosis and spondylolisthesis. Diagnosis is clinical with modified Beighton score 4/9. Always rule out more serious cause of hypermobile joints like Marfans syndrome, Ehler-danlos syndrome and Osteogenesis imperfecta.

6	Osteoporosis	Patients present with "aches and pains all over," also suspect in patients with Recent wrist fracture, Previous low trauma fractures, # from fall from standing and Loss of height/kyphosis (? Vertebral #), Age > 50 and female sex, Recurrent falls, Glucocorticoid use, Family History of osteoporosis, Secondary causes e.g. Primary hypoparathyroidism (serum Ca), Thyrotoxicosis (TSH), Myeloma (ESR, plasma electrophoresis, BJ proteins), Osteomalacia (serum Ca, Phosphate, ALP), Malabsorption syndrome.
7.	Fibromyalgia	Fibromyalgia (FM) is characterized by chronic widespread musculoskeletal pain and tenderness. FM patients also commonly complain of associated neuropsychological symptoms of fatigue, unrefreshing sleep, cognitive dysfunction, anxiety, and depression.[25] Pain in the left and right side of the body, pain above and below the waist and axial skeletal pain (shoulder and buttock included on each side and lower back pain considered lower segment pain) for at least 3 months.
8.	Chronic fatigue syndrome	Chronic fatigue syndrome (CFS) is a disorder characterized by persistent and unexplained fatigue resulting in severe impairment in daily functioning. Besides intense fatigue, most patients with CFS report concomitant symptoms such as pain, cognitive dysfunction, and unrefreshing sleep. Additional symptoms can include headache, sore throat, tender lymph nodes, muscle aches, joint aches, feverishness, difficulty sleeping, psychiatric problems, allergies, and abdominal cramps. Treatment consists of Cognition Behavioral Therpay and graded exercise therapy
Orthopedic		
1	Facet Joint Pain	Onset in minutes to hours, lasts days to weeks, and is worse on extension. Often associated with osteophytes, Facet (zygapophyseal) joint pain may account for up to 15% to 40% of low back pain. Patients have low back pain that may radiate to the buttocks which is made worse by extension and rotation of the lumbar spine. Radiologic studies may demonstrate facet arthritis but most likely will show no abnormality.
2	Lumbar sprain/strain/ Whiplash injury	Overall, the main sites of low back pain are the posterior longitu- dinal ligament, the interspinous ligaments, the nerve roots and dural coverings, the facet joints, and the deep muscles.
3	Lumbar spondylosis	**Lumbar spondylolisthesis** is a slippage usually of the fifth lumbar segment due to a fracture of the pars interarticularis, and those affected have low back pain that radiates to the coccyx or lateral aspect of the leg. Plain x-ray films are all that are usually needed to make the diagnosis.
4	Vertebral fracture	Trauma—falls, motor vehicle accidents. Atraumatic fractures—osteoporosis, neoplastic infiltration, exogenous steroids, osteomyelitis
5	Coccygodynia	**Coccygodynia** is the complaint of pain at the base of the spine; etiology is unknown. Trauma, intraosseous lipoma, chordoma, and giant cell tumor have been postulated as the cause. Imaging studies usually show no abnormality.
6	Kyphoscoliosis	Spinal curvature problem
7	Infection	Vertebral osteomyelitis/ Spinal epidural abscess/ Septic disk (discitis)/ Meningitis/ Lumbar arachnoiditis
8	Neoplasm	metastatic, hematologic, primary bone tumours
OSTEOPATHIC-posture related		
1	Somatic dysfunction	Somatic dysfunctions, both segmental and intersegment, may result in significant pain.
2	Posture related	Evaluation of posture should be part of every osteopathic evaluation. According to Cailliet,[23] 15 80% to 90% of low back pain is related to poor posture.
3	Referred pain	Referred pain from the viscera, known as **viscerosomatic reflexes**. These reflexes can be seen with pathologic lesions in the prostate, stomach, colon, uterus, kidney, urinary bladder, liver, and spleen
4	Piriformis syndrome	Piriformis syndrome is a compression of the sciatic nerve as it courses under or through the piriformis muscle. Patients complain of gluteal and hip pain that is

		exacerbated with flexion, adduction, and internal rotation of the hip (FAIR), that is, piriformis stretch.

Detail discussion about spondyloarthropathies (SPA) –Inflammatory low back ache

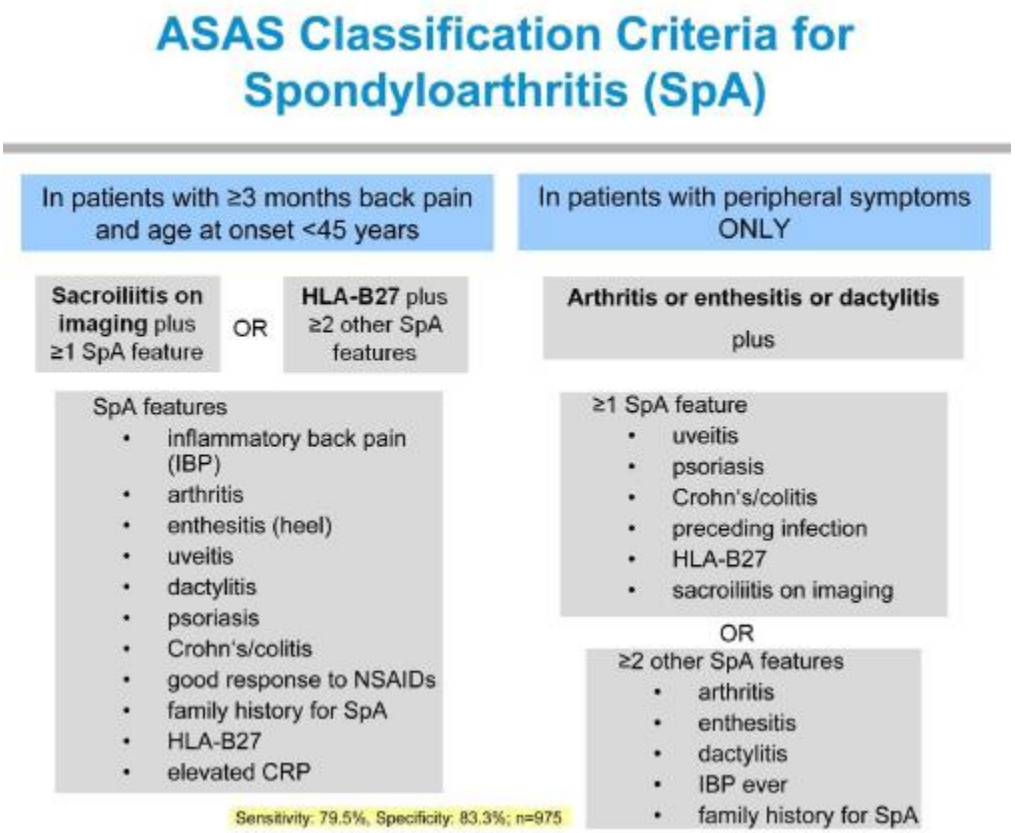
Spondyloarthropathies (SpAs) are classically described as a family of interrelated yet distinguishable disorders which include **A**nkylosing spondylitis (**AS**), **R**eactive arthritis (ReA), **P**soriatic arthritis(PsA), **I**nflammatory bowel disease (IBD) associated, **U**ndifferentiated spondyloarthropathy (USpA) and **J**uvenile chronic arthritis-SpA variant (Mnemonic-PAIR JU). **SpA clinically has 3 components-1.Axial SpA, 2.Peripheral SpA, 3.Extra-articular manifestations of SpA.**

Using **ASAS CRITERIA[(27)]-** SpA defined as patients with age less than 45 yrs predominantly axial (spinal) involvement with at least 3 month history of SpA features from total 11 defined SpA features and,

1. **HLA B 27 +**status without radiological sacroilitis with at least **two** SpA features **OR**
2. Sacroilitis on **MRI** (but not on x ray) with **one** SpA features.

Predominantly peripheral features are more common in younger age and females with predominant enthesitis, so also called as enthesitis related arthritis. These patients develop axial involvement with back pain later in life and may progress to classical AS if not taken care at early stages.

Also some patient may not manifest sacroilitis on x ray, but manifest on MRI of sacroiliac joint, this condition termed as non radiographic/pre-radiographic SpA. It can be diagnosed by HLA B27 criteria without any evidence of sacroilitis at all.



Criteria for AS - predominantly axial (spinal) involvement with at least 3 month history of SpA features and age less than 45 yrs.

Within this axial SpA group, you then have 2 subgroups:

1. **Ankylosing Spondylitis (AS) –** this is the diagnosis when the X-ray changes for sacroilitis are clearly present. SpA with radiological sacroilitis + 1 SpA features- found in 30% of total patients with inflammatory LBA using ASAS criteria.
2. **Non-radiographic axial spondyloarthritis** – this is the type of axial SpA in which the X-ray changes are not present. Term pre/non-radiographic axial SpA or **nr-axSpA** is used for SpA without radiological sacroilitis but

HLA B 27 +status +2 SpA features found in up to 2/3 rd of all patients with inflammatory LBA using ASAS criteria.[2]

Non-radiographic axial SpA is diagnosed when there is a typical history of inflammatory back pain, with other clinical criteria pointing to it or by typical changes on MRI showing active inflammation of the bone.

Pre/Non-radiographic axial spondyloarthritis (SpA) must be differentiated from undiffertiated SpA- which is predominantly peripheral involvement using ASAS criteria.

Undiffertiated SpA – these are the patients who do not full fill the criteria for diagnosis of particular SpA type, it can develop into axial predominant or peripheral predominant SpA. Reactive arthritis has significant overlap with undiffertiated SpA particularly when evidence of preceding infection is lacking or early axial spa when even preradilological evidence of sacroilitis had not been developed. Patents who do not progress to radiographic changes or other clinical features of AS will continue to be classified as undifferentiated SpA. So ASAS criteria have been revised recently, separately for axial disease(not include possibility of reactive arthritis) and peripheral disease(include possibility of reactive arthritis). Using these criteria SACROILITIS /HLA B27 positivity/ LBA none is essential to diagnose PERIPHERAL SpA. History related to reactive arthritis (ReA) is not included in ASAS criteria for axial disease/LBA as ReA is predominantly peripheral arthritis rather than axial.

SPA Features: 11 SpA features are: (Mnemonic - **ABCDEFGHI, PU)**

A. Arthritis Past or present active synovitis diagnosed by a doctor. Axial and extra axial joints –girdle/ root joints (hip and shoulder joint) involve about 35% patients. In peripheral joints knee is most commonly involved. Temperomandibular joints are also involved in 10 % of pts**.**

B. **Inflammatory back pain** according to experts: four out of five of the following parameters present: (1) age at onset 40 years, (2) insidious onset, (3) improvement with exercise, (4) no improvement with rest, (5) pain at night (with improvement upon getting up).

C. **CRP-** increased CRP above upper normal limit in the presence of back pain, after exclusion of other causes for elevated CRP concentration.

D. **DACTYLITIS** Past or present dactylitis (Sausage-like toe or digit) diagnosed by a doctor.

E. **ENTHESITIS** Heel enthesitis: past or present spontaneous pain or tenderness at examination at the site of the insertion of the Achilles tendon or plantar fascia at the calcaneus.

F. **Family history** Presence in first-degree or second-degree relatives of any of the following: (a) ankylosing spondylitis, (b) psoriasis, (c) uveitis, (d) reactive arthritis, (e) inflammatory bowel disease.

G. **Good response to NSAIDs**. At 24–48 h after a full dose of NSAID the back pain is not present anymore or much better.

H. **HLA B27** Positive testing according to standard laboratory techniques.

I. **IBD FEATURES**. Past or present Crohn disease or ulcerative colitis diagnosed by a doctor.

P.PSORIASIS. Past or present psoriasis diagnosed by a doctor.

U.UVITIS. Past or present anterior uveitis, confirmed by an ophthalmologist.

All these are early features ,some of the other features are usually present in late and advance cases so we need to know them as well to understand in better way that what we are going to prevent with early diagnosis and early treatment.

Other features of AS

1. **Constitunal Symptoms**: low grade fever, weight loss and fatigue.
2. **Skeletal**
   a. Bamboo spine- spinal inflammation leading to fusion and thoracic kyphosis .Mostly during the first 10 years of illness if not treated early leading to restriction of neck or other spinal movements.
   b. Metabolic bone disease (new bone formation at sites of spinal and peripheral enthesitis, syndesmophytosis, and bone loss in the form of osteopenia, osteoporosis).
   c. Chest tightness with pluritic type of chest pain, accentuated with coughing /sneezing with mild to moderate reduction in chest expansion can be present in early stage of AS with tenderness on sternocostal/costosternal joints.
   d. Bone spur formation at enthesitis sites eg heel /planter fasciitis.
3. **Extra skeletal menifestations**: (Mnemonic -**A5)**
   a. Eye disease: **A**. Acute anterior uveitis/iridiocyclytis
   b. Cardiovascular System: **A**. Ascending aortitis/Aortic Regurgitation
   c. Respiratory System: **A**: Apical fibrosis of lungs progressively.
   d. Neurological **A**: Atalanto axial joint subluxaxation /Archodonitis /arachnoidal adhesions.
   e. Renal **A**: IgA nephropathy. Rarely renal Amyloidosis.

Investigations: It is recommended that no laboratory investigations be ordered for patients with uncomplicated mechanical low back pain. If there is suspicion about a systemic cause of low back pain such as inflammatory arthritis, connective tissue disease, or infection, then **CBC, ESR/CRP, HLA B27**, and other markers may be appropriate.

ESR/CRP: In general, ESR/CRP would be a good first investigation when an infection or systemic condition is suspected, such as RA or connective tissue disease

HLA B27: In case of inflammatory LBA of more than 3 month duration or peripheral arthritis predominantly of lower limb large joint asymmetric type even without back pain. Positive in about 90% patients with ankylosing spondylitis and indicate propensity for severe disease.

Imaging: No imaging is recommended for acute mechanical low back pain in the absence of red flags. Keep in mind that imaging can be used to help confirm a clinical diagnosis, but cannot confirm that a particular structure is the cause of a patient's pain. Patients with uncomplicated acute low back pain and no red flags, who are between 20 and 50 years old, do not require imaging. In chronic low back pain, it may be appropriate to take AP and lateral lumbosacral x-rays. A bone scan can also be considered. If the pain radiates below the knees, an MRI may be indicated. Many doctors order elaborate studies when non-specific back pain is presented, including X-rays and magnetic resonance imaging, with little guidance to treatment decisions being the result. It has been demonstrated that 20% to 30% of healthy, asymptomatic patients have CT- or MRI-evident herniated disks without any significant indentation, so need not to advice radiological investigation to nonspecific low back patients, to avoid confusion in diagnosis and unnecessary cost.

Indications for Imaging Modalities

1. **X-Ray:** X-ray remains the imaging of first choice for investigation of suspected OA (disc space uniformity), tumor, trauma, spondylolisthesis, and ankylosing spondylitis. It is also acceptable to order x-rays of the lumbar spine in the case of chronic LBP. Minor abnormalities are very common on x-ray films of the lumbar spine.

In general, a lumbar x-ray is a low yield test. However, the dose of radiation from a set of lumbar spine x-rays is 120 times that of a chest x-ray. The incidence of cancers induced by radiation following x-rays of the lumbar spine may be around 1 in 25 000.

2. **CT:** CT is the test of choice to investigate pain suspected to be from multi-segmental bony stenosis, and fracture. CT is most helpful if osseous abnormality is clinically suspected, as abnormal findings are commonly found on CTs of asymptomatic patients. CT is commonly used along with MRI to investigate spinal trauma or tumours and is also commonly used for OA.
3. **Bone Scan:** This is a useful test to investigate osteomyelitis, primary or metastatic bony neoplasm, RSDS, occult fractures and spondyloarthropathy (i.e. facet or SI joint pain). The test is quite sensitive for infections and tumours, but false positives are common in the elderly due to the presence of OA. False negatives may occur with diffuse bony metastases and multiple myeloma.
4. **MRI:** MRI is the primary diagnostic tool when Cauda equina or malignancy is suspected, or if there is a previous history of cancer or complaint of progressively worsening radiculopathy over 4 months. MRI is also the best test for osteomyelitis as it can detail the extent of damage, but bone scans and white blood cell scans are more commonly used due to availability. In confirmation of sacroilitis when it is not visible on simple x ray pelvis for sacroiliac joints .bone marrow edema may be visible in early stage in MRI.

Investigation Pearl

a. Make diagnosis clinically with very minimal and relevant uses of investigations.
b. Do not offer X-ray of the lumbar spine for the management of non-specific low back pain.
c. Consider MRI (magnetic resonance imaging) when a diagnosis of spinal malignancy, infection, fracture, Cauda equina syndrome or ankylosing spondylitis or another inflammatory disorder is suspected.
d. Only offer an MRI scan for non-specific low back pain within the context of referral for an opinion on spinal fusion.

Management of acute low back pain

Key Points of Management

- The most of patients do not have severe (bilateral LE motor weakness and sensory loss) or progressive neurological deficits and so need conservative management.
- Strong analgesics such as opioids are generally not indicated.
- The patient with mechanical low back pain should be encouraged to recognize the pain as part of the healing process and attempt to continue to exercise while tolerating some pain.
- Screening for red and yellow flags; patient reassurance and education; and symptom management.
- NSAIDs and acetaminophen are the first line agents for pain relief.
- If NSAIDS are contraindicated (CHF, allergies, renal failure) muscle relaxants or weak opioids are alternatives.
- For severe back pain, stronger opioids can be used. Patients should be advised regarding the side effects associated with muscle relaxant and opioids use.
- Activities may be modified according to pain tolerance, however, it is important to emphasize that patients should make every effort to a gradual return to normal activity.
- Chiropractic manipulation[32] has been shown to provide pain relief for acute LBP but there is no significant difference in patient outcome between

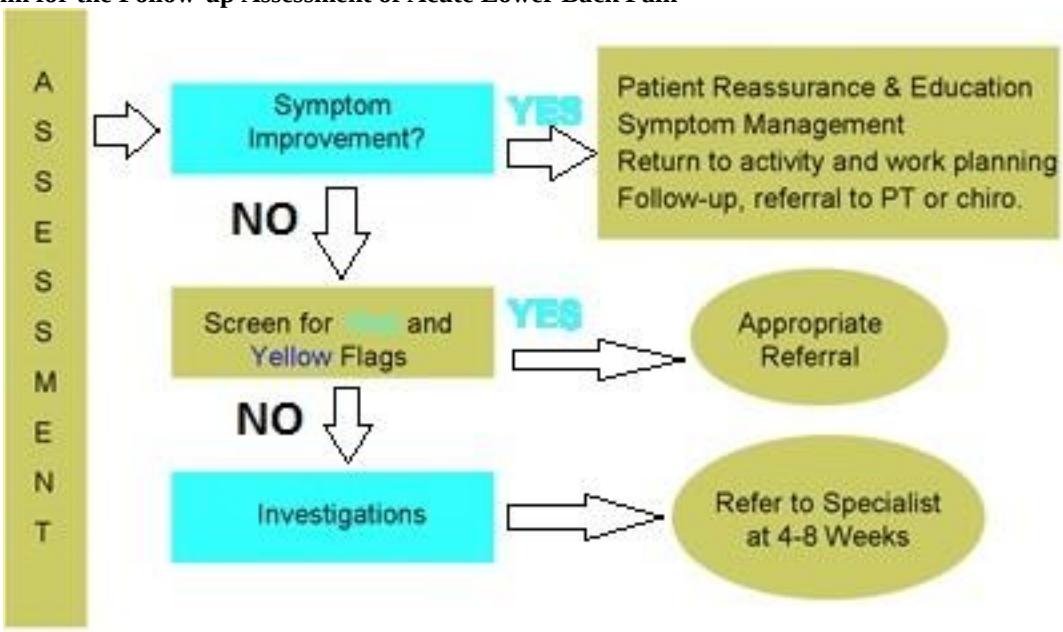
spinal manipulative therapy, general practitioner care, and physical therapy.

- Osteopathic manipulation, Yoga, Acupuncture, Spa therapy and other forms of moist heat and physical therapy

Time range for various interventions

2 days	Bed rest should be minimized to no more than **2 days**.
every **2 weeks**	Ongoing symptom review and management are also important. The patient should be reassessed every **two weeks** after the initial assessment. Each assessment should include a review of symptoms to screen for red/yellow flags.
Few weeks.	Usually radiculopathy will resolve within a **few weeks**.
2-4 weeks,	For patients with mechanical LBP who do not improve within **2-4 weeks,** treatment consists of conservative measures such as weak analgesics and increased activity level.
4-6 weeks	Imaging or referral should be considered after **4-6 weeks**.
> 6 weeks	For lumbar disc herniation with pain lasting > **6 weeks,** CT and MRI are the modalities of choice.
Urgent	Patients who exhibit symptoms of Cauda equina syndrome require **urgent** referral to neurosurgery.
Initial 2-3 weeks	During follow-up assessments of acute LBP, physiotherapy may play a role in the long-term return to work/normal activities plan. It is recommended that patients not be referred for physical therapy in the initial 2-3 weeks of onset of pain.
6 weeks	Consider referral if the patient has unremitting pain 6 weeks after symptom onset.

Algorithm for the Follow-up Assessment of Acute Lower Back Pain



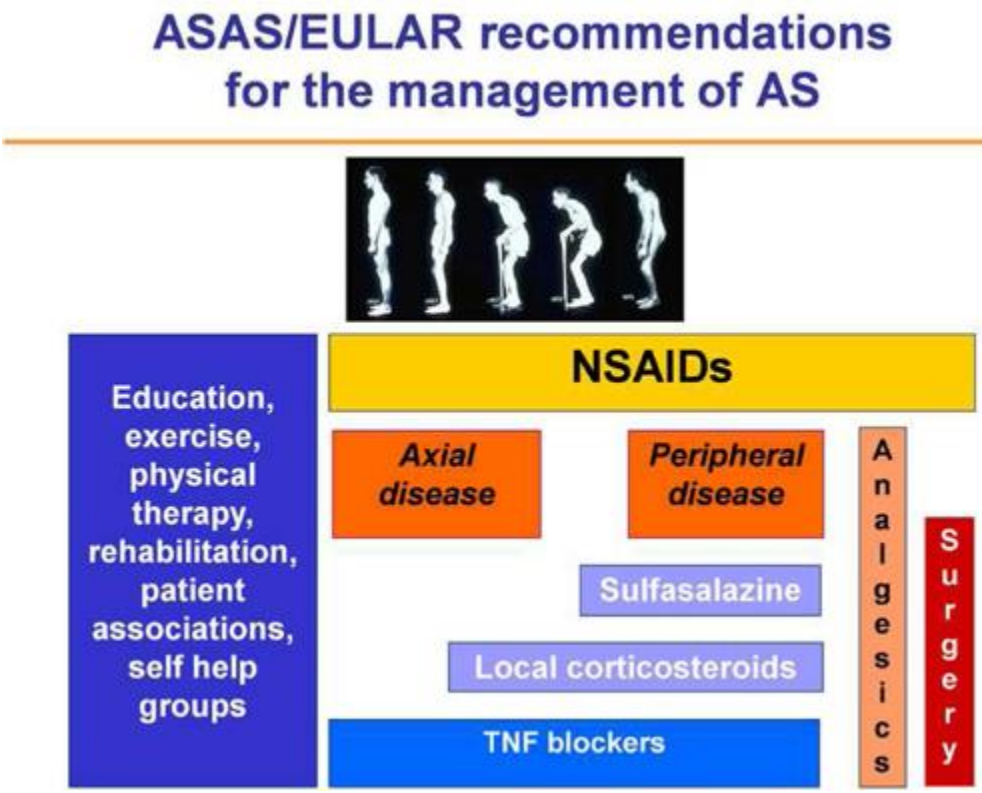
Management of chronic pain

Inflamatory Type (SpA)

NSAIDS- continuous therapy with NSAIDS retards radiographic disease progression. Various studies suggested a possible disease-controlling effect of continuous therapy.

SN	Treatment	Level of evidence	Indication
1	Physical therapy and exercise	A	All patients
2	NSAIDs	A	All –especially axial
3	Sulfasalazine	A	Peripheral SPA
4	Methotrexate	B	Peripheral SPA
5	Leflunomide	B	Peripheral SPA

6	Bisphosphonates,	B	Axial SPA
7	Thalidomide	B	Axial SPA
8	Biological –Anti TNF	A	All types(axial, peripheral and extraarticular menifestations)



Mechanical /non inflamatory type

In the absence of red flags, chronic back pain should be treated in a multi-modal but conservative fashion. Early return to work is a priority. The patient's ideas about pain and other yellow flags need to be addressed with appropriate sensitivity.

Overall Approach to Management of Chronic Back Pain

- Management usually with conservative approach with massage, intensive exercise therapy, medications for pain and/or depression, etc.
- Utilize a multi-disciplinary, intensive treatment regimen, if the patient is significantly affected by chronic pain and has failed to improve with trials of first-line treatment.
- Investigate for a specific diagnosis with joint blocks or discography and treat appropriately.
- Consider opioids only for short term use in patients experiencing severe exacerbations of back pain or rarely for those who do not respond to other measures, who are at low risk of drug abuse.
- Depression is common in patients with chronic back pain, screen for it and treat if present.

Levels of action of various management in pain control	Peripheral Level	Spinal Level	Cortical Level
	NSAIDS	Opioids analgesics TENS	Antidepressants anticonvulsant medications
	muscle relaxants		
	epidural steroid injections		
	facet joint blocks		
	injection into trigger points		
	physical stretching exercises and abdominal exercises		
	osteopathic manipulations		

Various pharmacological[30] treatments in treating chronic LBP

Acetaminophen and NSAIDS	First-line medications for managing acute exacerbations of sub acute (pain with duration between 4- 12 weeks) or chronic LBP
Opioids	Opioids only partially relieve the pain, must be used carefully. Consider weak opioids (e.g. Tramadol) for short-term use in patients experiencing severe exacerbations of back pain or rarely for those who do not respond to other measures, who are at low risk of drug abuse.
Antidepressants[28]	There are conflicting results on the benefits .However, depression is a common co-morbidity of chronic back pain and should be assessed for and treated if present.
Muscle relaxants[28]	There is insufficient evidence. Short-term use of muscle relaxants may be considered as adjunctive treatment to analgesics if pain cannot be managed with analgesics alone but caution must be taken in prescribing due to CNS side effects and potential for abuse.
Benzodiazepines	Limited evidence .Prescription of benzodiazepines for long-term treatment of chronic LBP is not recommended.
Anti-epileptics (Gabapentin)	Gabapentin should not be prescribed to treat chronic low back pain as RCTS have shown that it has no significant benefit over placebo.
Herbal medicine	Harpagoside, salacin and capsicum frutescens all have some benefit in pain relief.

Multidisciplinary treatment[12] programs that are intensive (greater than 100 hours), include medical, physical exercise, vocation and behavioural components and are provided by 3 or more health care providers in different fields are more effective in reducing pain, improving function and speeding up return to work than less intensive programme.

Physical activity and exercise[31]

- Advise people with low back pain that staying physically active is likely beneficial.
- Advise people with low back pain to exercise.
- Consider offering a structured exercise programme tailored to the person: This should comprise up to a maximum of eight sessions over a period of up to 12 weeks
- Offer a group supervised exercise programme, in a group of up to 10 people.
- A one-to-one supervised exercise programme may be offered if a group programme is not suitable for a particular person. Exercise programmes may include the following elements: aerobic activity movement instruction muscle strengthening postural control stretching.

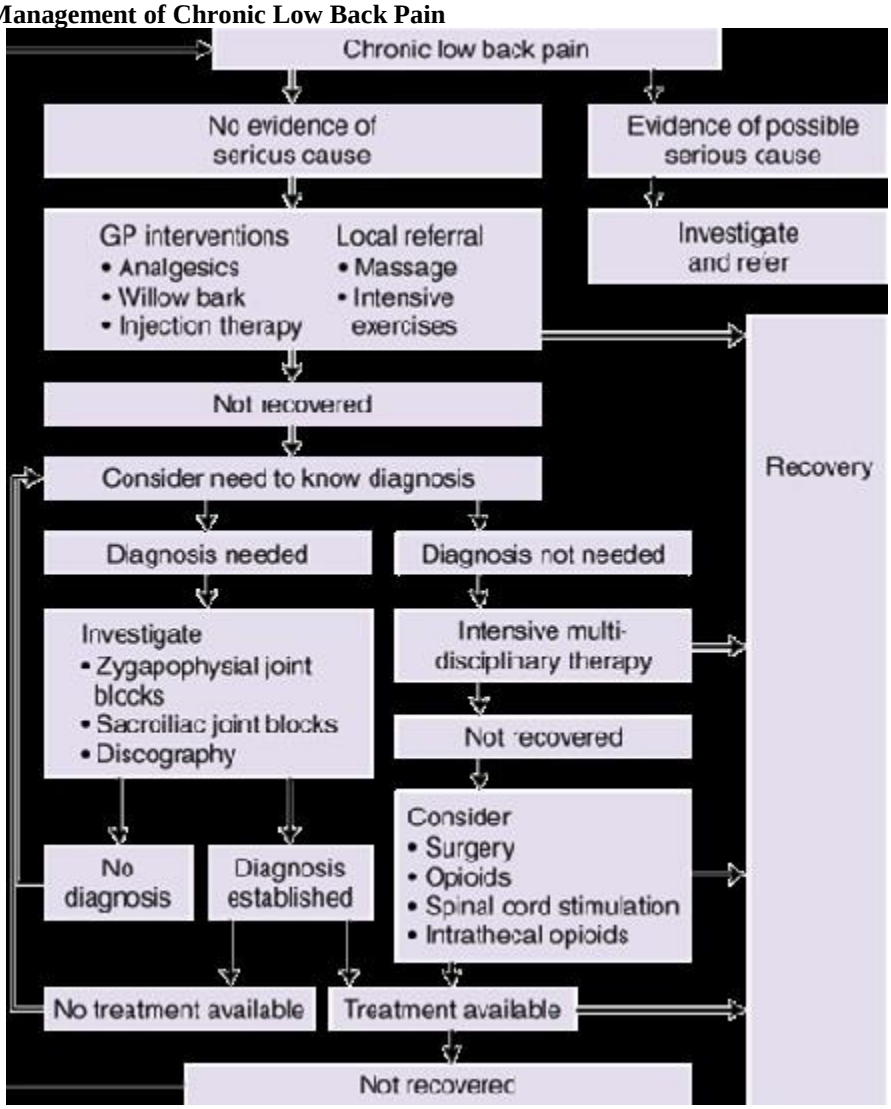
Manual therapy

The manual therapies reviewed were spinal manipulation (a low-amplitude, high-velocity movement at the limit of joint range that takes the joint beyond the passive range of movement), spinal mobilisation (joint movement within the normal range of motion) and massage[33] (manual manipulation or mobilisation of soft tissues). Collectively these are all manual therapy. Mobilisation and massage are performed by a wide variety of practitioners. Manipulation can be performed by chiropractors and osteopaths, as well as by doctors and physiotherapists who have undergone specialist postgraduate training in manipulation. Consider offering a course of manual therapy, including spinal manipulation, comprising up to a maximum of nine sessions over a period of up to 12 weeks. Spinal manipulation is not beneficial after 6 weeks of acute LBP. Hence, chiropractic therapy does not play a role in the management of chronic back pain.

Non pharmacological alternative treatment available	
Multidisciplinary treatment programs	Should be the approach
Exercise therapy[31]	Advise people with low back pain that staying physically active is likely to be Beneficial as discussed above in text.
Acupuncture	It should be prescribed in combination with rehabilitation therapy, if at all required in resistant case.
Cognitive-behavioural therapy	Cognitive-behavioural therapy(35) can mitigate the psychological factors contributing to chronic back pain e.g. chronic fatigue syndrome, fibromyelgia. CBT has been found to be effective in relieving pain and improving function.
Spinal manipulation[29]	Spinal manipulation has been found to be beneficial in reducing pain and improving function, and has been found to have equal effectiveness as analgesics, physical therapy and exercise therapy. Spinal manipulation is not beneficial after 6 weeks of acute LBP.
Massage	Massage may be beneficial in relieving pain and improving function

Treatment not recommended	Bed rest, lumbar supports and traction. naturopathic medicine therapies, laser therapy, interferential therapy Anti-epileptics, facet joint injections, EMG biofeedback, therapeutic ultrasound and traction.
Treatment with conflicting evidence	Epidural steroid injections, trigger point injections, electrical muscle stimulation, laser, TENS, and superficial hot and cold therapy.

Algorithm for Management of Chronic Low Back Pain



Referral for surgery[34]

1. Cauda equina.
2. Progressive or severe neurological deficit.
3. Persistent sciatica for 4-6 weeks.
4. Persistent neurological deficit after 4-6 weeks of conservative treatment.

References

1. Taylor H, Curran NM. The Nuprin Pain Report. New York, NY: Louis Harris & Associates; 1985. Cited in: Raj P. Pain Management: A Comprehensive Review. Los Angeles, Calif: Mosby; 1996; p 406.
2. Frymoyer JD. Back pain and sciatica. N Engl J Med. 1988;318:291–300.
3. Badley EM, Rasooly I, Webster GK. Relative importance of musculo- skeletal disorders as a cause of chronic health problems, disability, and health care utilization: Findings from the 1990 Ontario health survey. J Rheumatol.1994;21:505.
4. Nyman T, Grooten WJ, Wiktorin C, Liwing J, Norman L. Sickness absence and concurrent low back and neck-shoulder pain: Results from the MUSIC-Norrtalje study. Eur Spine J. 2007;16:631.
5. Hoogendoorn WE, Bongers PM, de Vet HC, Ariens GA, van Mechelen W, Boutler LM. High physical work load and low job satisfaction increase the risk of sickness absence due to low back pain. Occup Environ Med.2002;59:323–8.
6. Reme SE, Hagen EM, Eriksen HR. Expectations, perceptions, and physiotherapy predict prolonged sick leave in subacute low back pain. BMC Musculoskelet Disord. 2009;10:139.

7. Bhuyar P, Banerjee A, Pandve H, Padmanabhan P, Patil A, Duggirala S, et al. Mental, physical and social health problems of call centre workers. Indian Psychiatry J. 2008;17:21–5.
8. Carey TS, Garrett JM, Jackman A, Hadler N. Recurrence and care seeking after acute back pain: results of a long-term follow-up study. North Carolina Back Pain Project. Med Care 1999;37:157-164.
9. Gordon M, Greenfield E, Marvin J, Hester C, Lauterbach S. Use of pain assessment tools: is there a preference? J Burn Care Rehabil 1998;19:451-454.
10. Michael Nicholas PhD, Associate Professor, pain management research institute, university of Sydney at royal north shorte hospital. ppt presentation.
11. Bigos SJ, Bowyer O, Braea G, Brown K, Deyo R, Haldeman S, et al. Acute low back pain problems in adults. Clinical practice guideline no. 14. AHCPR Publication No. 95-0642. Rockville (MD): US Department of Health and Human Services; 1994.
12. Ehrlich GE, Khaltaev NG. Low back pain initiative. Geneva: World Health Organization; 1999.
13. Mannel JM Joint pain. Boston: little brown,1964.
14. International Association for the Study of Pain. Classification of Chronic Pain. Pain 1986; Suppl 3:S1-226.
15. Jayson MIV. Why does acute back pain become chronic? Chronic back pain is not acute back pain lasting longer. BMJ 1997;314:1639-40.
16. Guidlines for back pain, year 3 clerkship guid, family medicine department, schulich school of medicine and dentistry.
17. Deyo RA, Diehl AK, Rosenthal M. How many days for acute low back pain? NEJM1986;315:1064-70.
18. Gordon M, Greenfield E, Marvin J, Hester C, Lauterbach S. Use of pain assessment tools: is there a preference? J Burn Care Rehabil 1998;19:451-454.
19. Katz W. Pain Management in Rheumatologic Disorders: A Guide for Clinicians. Drugsmartz Pub; 2000;pp 1-11, 14, 20-21, 110.
20. Hoppenfeld S. Physical Examination of the Spine Extremities. Norwalk, Conn: Appleton & Lange; 1976;pp 237-263.
21. Cole A, Herring S. The Low Back Pain Handbook: A Practical Guide for the Primary Care Clinician. Philadelphia, Pa: Handley & Belfus, Inc; 1997; pp 31, 39,59-90, 92.
22. Kosteljanetz M, Espersen JO, Halaburt H, Miletic T. Predictive value of clinical and surgical findings in patients with lumbago-sciatica. A prospective study (Part I). Acta Neurochir (Wien) 1984;73(1-2):67-76.
23. Cailliet R. Low back pain. In Cailliet R, ed: Soft Tissue Pain and Disability. Philadelphia, Pa: FA Davis;1977.
24. Bozzao A, Gallucci M, Masciocchi C, Aprile I, Barile A, Passariello R. Lumbar disk herniation: MR imaging assessment of natural history in patients treated without surgery. Radiology 1992;185:135-141.
25. Croft P, Burt J, Schollum J, Thomas E, Macfarlane G, Silman A. More pain more tender points: Is fibromyalgia just one end of a continuous spectrum? Annals of Rheumatic Diseases 1996;55:482-5.
26. Hunt IM, Silman AJ, Benjamin S, McBeth J, Macfarlane GJ. The prevalence and associated features of chronic widespread pain in the community using the 'Manchester' definition of chronic widespread pain. Rheumatology 1999;38:275-9.
27. ASAS handbook doi:10.1136/ard.2008.104018 Ann Rheum Dis 2009;68;ii1-ii44.
28. Wolfe F, Smythe HA, Yunus MB, Bennett RM, Bombardier C, Goldenberg DL, et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. Report of the Multicenter Criteria Committee. Arthritis and Rheumatology 1990;33:160-72.
29. Triano JJ, McGregor M, Hondras MA, Brennan PC. Manipulative therapy versus education programs in chronic low back pain.Spine1995;20;948-55.
30. Von Feldt JM, Ehrlich GE. Pharmacologic therapies. Low back pain. Physical Medicine and Rehabilitation Clinics of North America 1998;9:473-85.
31. Timm KE. A randomized control study of active and passive treatments for chronic low back pain following L5 laminectomy. J Orthop Sports Phys Ther 1994;20;276-86.
32. Bigos SJ, Bowyer O, Braea G, Brown K, Deyo R, Haldeman S, et al. Acute low back pain problems in adults. Clinical practice guideline no. 14. AHCPR Publication No. 95-0642. Rockville (MD): US Department of Health and Human Services; 1994.
33. Cherkin DC, Sherman KJ, Deyo RA, Shekelle PG. A review of the evidence for the effectiveness, safety, and cost of acupuncture, massage therapy, and spinal manipulation for back pain. Annals of Internal Medicine 2003;138:898-906.
34. Junge A, Dvorak J. Ahrens S. Predictors of bad and good outcome of lumber disc surgery. Spine;1995;20:460-8.