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Diagnostic and therapeutic perspectives on low back pain in women with hypertension

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ABSTRACT

Low back pain (LBP) affects approximately 9–12% of the global population at any given time. Most cases are nonspecific, occurring without a clear anatomical cause, and are typically managed with conservative approaches such as physiotherapy and light activity. This case involved a woman with LBP due to lumbar spondyloarthrosis and herniated nucleus pulposus (HNP) accompanied by hypertension. Her history of elevated blood pressure and frequent heavy lifting were recognized as significant risk factors, potentially affecting organ perfusion. The management provided in this case was appropriate and comprehensive, combining pharmacotherapy, physiotherapy as supportive therapy to enhance functional recovery, and comorbidity management. This integrated approach demonstrated effective clinical outcomes in addressing both the musculoskeletal and cardiovascular aspects of the patient's condition.

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INTRODUCTION

Low back pain (LBP) affects approximately 9–12% of the global population at any given time and increase each year (García-Moreno et al., 2022). It affect almost all age and sometime lead to dissability (Christe et al., 2021; Mescouto et al., 2022; Ogbeivor & Elsabbagh, 2021). Most cases are nonspecific, occurring without a clear anatomical cause, and are usually managed with conservative approaches such as physiotherapy and light activity. However, some cases are associated with mechanical or non-mechanical conditions, including disc degeneration, spondylolisthesis, or inflammatory and infectious diseases, which require specific evaluation and intervention. Diagnostic assessment often involves identifying "red flags," such as fever, weight loss, neurological symptoms, or persistent pain, which may indicate vertebral infection, tumor, or chronic inflammatory disorders (Deyo & Weinstein, 2001).

In 2020, an estimated 619 million people worldwide experienced LBP, and this number is projected to rise to 843 million by 2050, in line with global population growth and aging. The main modifiable risk factors include workplace ergonomics, smoking, and high body mass index (BMI).

Notably, every 5% increase in BMI raises the risk of developing LBP by up to 35% (Balagué et al., 2012).

LBP refers to pain localized in the lower back, which may present as localized pain, radicular pain, or both. The discomfort is typically felt between the lower rib margin and the gluteal folds, in the lumbar or lumbosacral region, and is often accompanied by pain radiating to the legs and feet (Pinzon, 2012; Tampubolon & Fransysca, 2021). LBP is classified as a musculoskeletal disorder but may also involve psychological components and is frequently caused by improper body movement patterns (Haritsah et al., 2024). Symptoms may include soreness, aching, pain, or discomfort in the lumbosacral and sacroiliac areas, with possible radiation to the lower limbs. This condition is more common among workers routinely engaged in activities such as lifting, carrying, pushing, or pulling heavy loads (Lubis, 2015). Heavy lifting is the most significant factor for low back pain, quadrupling the risk. Age is also a significant risk factor related to structural degeneration and reduced physical capacity with age. Hypertension can act as a comorbidity, worsening low back pain (Bae et al., 2015).

Hypertension, a prevalent comorbidity in adults, may exacerbate LBP by compromising tissue perfusion and amplifying inflammatory responses. Chronic hypertension induces endothelial dysfunction and capillary rarefaction, reducing microvascular blood flow to spinal and paraspinal tissues. This perfusion deficit delays tissue repair and lowers mechanical tolerance, predisposing the spine to accelerated degeneration. At the same time, hypertension promotes systemic inflammation and oxidative stress, which sensitize pain pathways and contribute to chronic pain persistence. Together, impaired perfusion and heightened inflammation create a pathophysiological environment that worsens LBP severity and hinders recovery (Xiao & Harrison, 2020).

Individuals with both LBP and hypertension require comprehensive management strategies that address musculoskeletal dysfunction while also controlling cardiovascular risk factors. This case report presents a patient with LBP caused by lumbar spondyloarthrosis and herniated nucleus pulposus (HNP) in the context of hypertension, highlighting diagnostic considerations and integrated management approaches.

RESEARCH METHOD

This study employed a case study design to examine a 53-year-old female patient presenting with low back pain resulting from lumbar spondyloarthrosis and herniated nucleus pulposus, accompanied by a history of hypertension. Data were collected through direct observation of the patient's condition and treatment, as well as from relevant journal articles and textbooks that provided valuable insights into low back pain associated with hypertension. Following data collection, the authors systematically organized and documented the findings to present a detailed case analysis.

RESULTS AND DISCUSSIONS

A 53-year-old woman presented to the emergency department with complaints of persistent right pelvic pain described as a burning sensation radiating to the tips of her right toes. One week prior, the patient had fallen in a seated position. She had a history of hypertension, well-controlled with amlodipine and hydrochlorothiazide, and reported a long-standing habit of lifting heavy loads since a young age.

Upon arrival at the emergency department, her vital signs were as follows: blood pressure 140/80 mmHg, heart rate 86 beats/min, respiratory rate 20 breaths/min, and temperature 36.4 °C. The main complaint of right pelvic pain had been present for the past five months and had worsened over the last three days. The pain was constant, with a burning quality radiating to the

right toes, relieved when lying supine at rest but aggravated by sitting, standing, walking, and physical activity. The pain limited her mobility.

She denied any family history of hypertension, diabetes mellitus, or similar complaints. Personal and social history revealed a routine of lifting heavy loads since early adulthood, regular consumption of vegetables and fruits, but no regular exercise habits. Physical examination of the head, neck, thorax, abdomen, and extremities was within normal limits. Neurological examination of the right lower extremity revealed positive Lasegue, Patrick, Contra-Patrick, and Bragard tests. A lumbar MRI was performed on December 17, 2024.

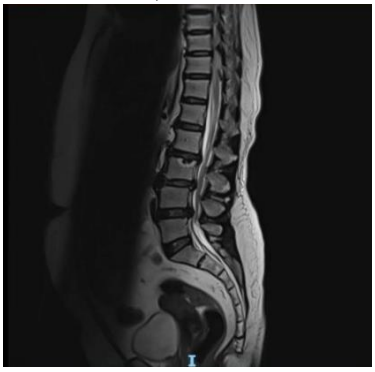

Figure 1. Lumbar MRI showing compression of the lumbar II, III, and IV nerve roots bilaterally, and herniated nucleus pulposus (HNP)

The MRI findings indicated compression of the traversing nerve roots at L II, L III, and L IV on both the right and left sides, with HNP characterized by disc protrusion at intervertebral levels VL II–III, VL III–IV, and VL IV–V. Magnetic resonance myelography demonstrated cerebrospinal fluid (CSF) flow obstruction at the levels of VL II–III, VL III–IV, and VL IV–V, consistent with HNP. In addition, plain radiographs of the lumbosacral vertebrae revealed lumbar spondyloarthrosis at levels L3–L4.

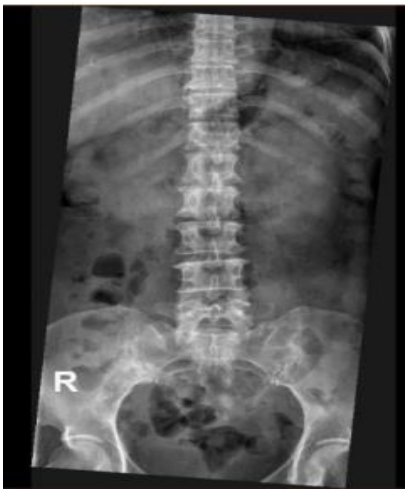

Figure 2. Plain radiograph of the lumbosacral vertebrae showing lumbar spondyloarthrosis at L3–L4

Based on comprehensive physical and diagnostic examinations, the patient was diagnosed with low back pain (LBP) secondary to lumbar spondyloarthrosis at L3–L4 and herniated nucleus pulposus (HNP), with comorbid hypertension. The patient received comprehensive management, including Ketorolac 30 mg every 8 hours, Mecobalamin 1000 mcg every 12 hours, Methylprednisolone 62.5 mg every 12 hours, Gabapentin 100 mg twice daily, Fiotram 100 mg twice

daily, Atorvastatin 20 mg once daily, Ondansetron 4 mg every 8 hours, and Ceftriaxone 1 g every 12 hours. Supportive therapy included physiotherapy.

LBP most commonly occurs at the L4–L5 or L5–S1 levels, which correspond to specific dermatomes. Loss of sensory reflexes in these dermatomes can lead to decreased deep tendon reflexes and muscle weakness (Splittgerber, 2024). Mechanical LBP is often caused by excessive use of muscles, either from prolonged static posture or incorrect body mechanics over an extended period. In such cases, back muscles contract to maintain normal posture or respond to mechanical overloading during physical activity. Overuse of the muscles may result in ischemia or inflammation, leading to pain and muscle spasms, which in turn limit lower back mobility (Deyo & Weinstein, 2001).

The diagnostic process for LBP begins with a thorough history, physical examination, and relevant investigations. History-taking should include pain duration (acute, subacute, or chronic), location and radiation (axial or radicular), severity (VAS or numeric scale), pain characteristics (burning, dull, electric shock-like), aggravating or relieving factors, social and occupational history, and psychological status (Koes et al., 2006). Physical examination involves observing gait, spinal alignment, and palpating for tenderness along the vertebral column and paravertebral muscle spasm [6]. Neurological examination is crucial for assessing pain distribution according to nerve root involvement, motor strength, sensory function, and reflexes of the lower extremities (Harris et al., 2017).

Provocative tests help localize the pain source, such as the Lasegue test for sciatic nerve irritation, Patrick and contra-Patrick tests for sacroiliac joint pain, Bragard and Sicard tests for nerve irritation with additional ankle or toe dorsiflexion, and the femoral nerve stretch test (FNST) for L2–L4 root involvement (Van Zundert et al., 2011). The Valsalva maneuver can also be used to assess pain associated with increased intrathecal pressure (Chou et al., 2007). Screening for red flags—including fever, weight loss, and trauma is essential to rule out serious conditions such as infection, malignancy, or fracture (Balagué et al., 2012).

Supporting investigations include laboratory and imaging studies. Laboratory tests may involve erythrocyte sedimentation rate, leukocyte count, protein electrophoresis, and calcium and phosphatase levels to detect infection or neoplasms (Modic & Ross, 2007). Plain radiographs (AP, lateral, oblique) help identify disc space narrowing, osteophytes, or spondylolisthesis. MRI is particularly useful for assessing soft tissues, the spinal cord, and disc herniation, especially in postoperative patients or those suspected of having infection or tumors. CT scans provide better visualization of bone structures, while CT myelography helps localize preoperative lesions and evaluate possible disc sequestration (Moore et al., 2015).

In this case, the patient experienced chronic burning pain radiating to the lower limb, aggravated by activity and relieved by rest, with a history of heavy lifting. Neurological examination revealed positive Lasegue, Patrick, contra-Patrick, and Bragard tests, indicating a spinal origin of the pain. MRI findings of lumbar spondyloarthrosis and HNP confirmed the diagnosis of LBP.

The primary goals of acute LBP management are to relieve pain, restore functional activity, reduce work absenteeism, and educate patients on pain-coping strategies to prevent progression to chronicity. Management consists of three main approaches: treatment of the underlying cause, surgical intervention, and conservative therapy. When LBP results from intra-abdominal pathology or spinal metastases, therapy targets the primary disease. Surgery is considered when there is a strong indication, whereas cases without surgical indications are treated conservatively with limited bed rest, bracing, physiotherapy (including thermotherapy and traction), muscle strengthening exercises, orthosis, and pharmacological therapy such as antibiotics, NSAIDs, and analgesics. Nerve blocks and psychotherapy may be indicated for chronic or psychogenic pain (Jacobs et al., 2011; Sun et al., 2005).

The American Family Physician guidelines recommend that for first-visit LBP patients without red flags, education should emphasize that most cases resolve spontaneously, advising patients to remain active and avoid excessive twisting or bending. First-line medications include NSAIDs or acetaminophen, with muscle relaxants such as diazepam or tizanidine for severe pain. Short-term opioid use may be considered if necessary. For recurrent episodes, physical therapy is advised. If symptoms do not improve within 2–4 weeks, alternative NSAIDs, physiotherapy referral, or spine subspecialist consultation should be considered (Wiffen et al., 2017).

For this patient, comprehensive management was essential, given the chronic musculoskeletal pain, neuropathic symptoms, and cardiovascular comorbidity. Ketorolac, a nonsteroidal anti-inflammatory drug (NSAID), was used for acute pain relief due to its potent analgesic and anti-inflammatory effects, though it requires caution in hypertensive patients as it may elevate blood pressure (Peloso et al., 2004). Mecobalamin (vitamin B12) supported nerve regeneration and alleviated neuropathic symptoms (Cholesterol Treatment Trialists' (CTT) Collaboration et al., 2015), while systemic corticosteroid methylprednisolone was used to reduce local inflammation caused by nerve root compression (Christofaki & Papaioannou, 2014). Gabapentin was prescribed to treat chronic neuropathic pain often associated with HNP (Pappas et al., 2016). Fiotram, a tramadol-paracetamol combination, provided dual analgesia for moderate-to-severe pain through opioid and non-opioid mechanisms (Hayden et al., 2005). Atorvastatin was administered for dyslipidemia management as part of cardiovascular risk reduction in hypertensive patients (Cholesterol Treatment Trialists' (CTT) Collaboration et al., 2015). Ondansetron, a serotonin antagonist, was used to manage nausea or gastrointestinal side effects from analgesics or steroids (Christofaki & Papaioannou, 2014), and ceftriaxone was given empirically to prevent or treat suspected infection. Supportive physiotherapy played a critical role in restoring muscle function, improving mobility, and accelerating recovery from LBP (Hayden et al., 2005). This comprehensive approach aimed to alleviate symptoms, prevent complications, and improve the patient's quality of life.

CONCLUSION

This case highlights the complex interplay between low back pain and hypertension, where impaired tissue perfusion and heightened inflammation contribute to pain persistence and delayed recovery. For broader clinical practice, patients presenting with LBP and comorbid hypertension should be managed through an integrated, multidisciplinary approach. Comprehensive care should combine appropriate analgesics, physiotherapy, and lifestyle modification with strict blood pressure monitoring and cardiovascular risk management. Clinicians are advised to use NSAIDs cautiously, prioritize non-pharmacological interventions when possible, and coordinate care with primary care or cardiology specialists. Early recognition of risk factors such as heavy lifting, aging, and hypertension is essential to prevent chronicity and optimize functional recovery. These strategies may improve clinical outcomes and quality of life in patients with LBP complicated by hypertension.

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