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CASE REPORT PAIN MANAGEMENT

Locked-in syndrome post facet joint injection: a case report

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ABSTRACT

Locked-in syndrome (LIS) is a neurological condition characterized by quadriplegia and anarthria with preservation of consciousness. This is a rare, but serious condition as patients with LIS are conscious and possess cognitive function, but cannot move or communicate verbally due to the paralysis of nearly all voluntary muscles in the body. We present a case of a 43-year-old lady who developed locked-in syndrome following a lumbar facet joint injection for chronic spinal pain. The patient developed a sudden onset of quadriplegia and loss of speech around 10 min after the injection. Neurological examination revealed preserved consciousness and alertness, with intact cranial nerve function. Diagnostic work-up, including neuroimaging and laboratory tests, ruled out hemorrhage or other structural lesions. We discuss the potential mechanisms underlying this unexpected complication, explore diagnostic challenges and treatment options. Despite its rarity, this case highlights the importance of careful patient selection, precise procedural technique, and prompt recognition of complications associated with interventional pain procedures.

Abbreviations: ITP - immune thrombocytopenic purpura; LIS - Locked-in syndrome; SLE - systemic lupus erythematous; SIJ - sacroiliac joint

Keywords: Locked-In Syndrome; Facet Joint Injection; Complication; Neurological; Case Report

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1. INTRODUCTION

Facet joint injections are common interventional pain procedures for the management of chronic spinal pain, particularly in the lumbar and cervical regions.[1] While generally considered safe, they can occasionally lead to adverse events, including infection (such as meningitis and epidural abscess), nerve injury like phrenic nerve palsy, intrathecal injection, pneumothorax, and vascular injury.[1] Locked-in syndrome (LIS) is an exceedingly rare but catastrophic complication characterized by quadriplegia and an inability to speak, with intact consciousness and vertical eye movement.[2] Neurological complications following facet joint injections are rare but can be devastating. Therefore, we present a case of LIS following a facet joint injection and discuss possible etiologies and management strategies.

2. CASE REPORT

A 43-year-old lady with a history of chronic low back pain secondary to degenerative lumbar spine disease reported to our pain clinic for evaluation and management. She was also a known case of systemic lupus erythematous (SLE) with immune thrombocytopenic purpura (ITP). The patient had undergone multiple conservative treatments, including physical therapy and pharmacotherapy, with inadequate relief. A thorough evaluation, including diagnostic imaging confirmed facet joint arthropathy at the right L3/L4, L4/L5, L5/S1 and left L3/L4 levels, together with bilateral sacroiliac joint (SIJ) involvement and piriformis syndrome. After discussion of the risks and benefits, the patient consented to an interventional pain procedure. Therefore, the decision was made to proceed with a fluoroscopy-guided facet joint injection at levels L3–S1, bilateral SIJ, and piriformis steroid injection. Her analgesic agents prior to the procedure were tablet dihydrocodeine 30 mg three times a day (TDS) and tablet etoricoxib 90 mg once daily (OD). She was electively admitted to our ward one day prior to the procedures.

The procedure was performed under sterile conditions and the patient was positioned prone on the procedure table. Area and point of injection were identified using anatomical landmarks and a C-arm guidance with contrast technique. After local anesthesia and under fluoroscopic guidance, a 22-gauge spinal needle was advanced into the bilateral piriformis muscles and bilateral SIJ. A mixture of local anesthetic and corticosteroid was injected (50 mg triamcinolone acetonide (Shincort) plus lidocaine 1% in a 10 ml solution), 3 ml for piriformis muscle and 2 ml for SIJ on each side. Following this, we proceeded to inject facet joints of the right L3/L4, L4/L5, L5/S1, and left L3/L4, using fluoroscopic guidance. A mixture of solution 2 ml were injected for each facet without apparent difficulty or patient discomfort. The vital signs remained stable during the procedure, with blood pressure (BP) ranging from 130 to 160 mof 100% under room air.

Following the procedure, about 10 min later, the patient complained of visual disturbances in the form of blurring of vision and difficulty in breathing while being transferred from the operating table onto the bedside trolley (prone to supine position). Shortly after that, she rapidly developed sudden-onset complete quadriplegia and loss of speech, with preserved vertical eye movements and blinking. The patient's consciousness level was intact, and she was able to communicate through eye and head movements. Vital signs were taken immediately, with a reading of BP 207/114, PR 64/min, and SpO_2 100% under room air. Facemask oxygen (5 L/min) was given despite a normal SpO_2 reading, in view of the patient complaining of difficulty in breathing. Immediate assessment revealed she was conscious and oriented but unable to speak or move any voluntary muscles except for head and vertical eye movements. Upon neurological assessments, it was found that her motor power was 0/5 from bilateral ankle joints up to bilateral shoulder joints, and her neck motor power was 4/5. Sensation was impaired from T1 and below. There was intact cranial nerve function with no facial asymmetry, including normal eye movements and pupillary responses. Therefore, she was then admitted to

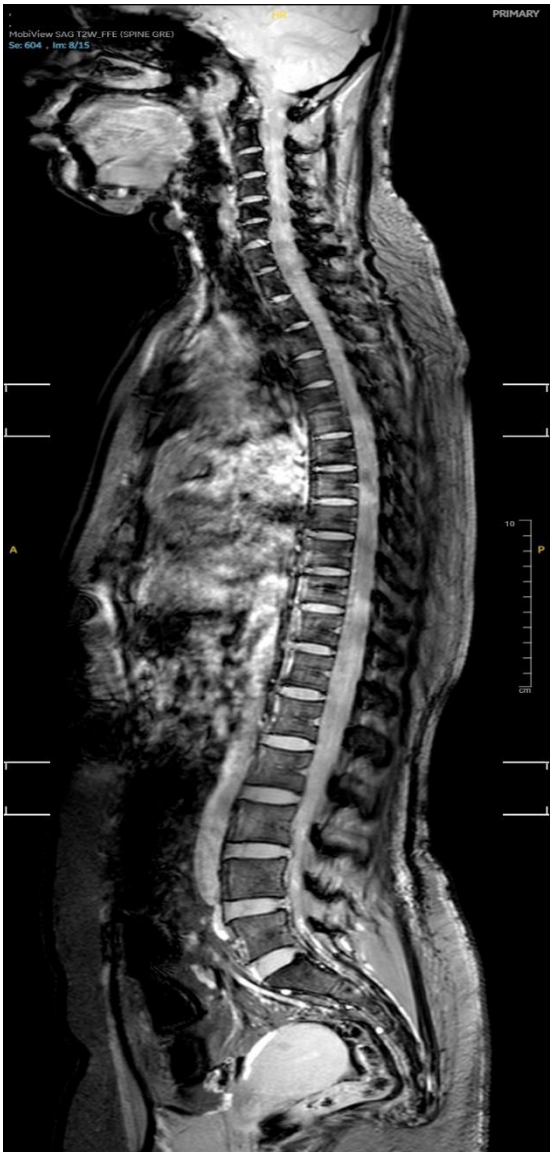

Figure 1: The MRI of the whole spine showed no acute abnormalities, including no evidence of haemorrhage or any spinal cord oedema, lesion, hematoma, or structural lesions. The spinal canal appears spacious, and the visualised brainstem and cerebellum are normal with no tonsillar herniation.

the surgical intensive care unit (SICU) for observation and further evaluation.

Given the concerning neurological deficits, urgent neuroimaging studies were performed, including brain and whole spine magnetic resonance imaging (MRI). It showed no acute abnormalities, including no evidence of hemorrhage or any spinal cord edema, lesion, hematoma, or structural lesions (Figure 1). The spinal canal appeared spacious, and the visualized brainstem and cerebellum were normal with no tonsillar herniation. The previous pathology revealed a posterior disc bulge at C3/C4, C4/C5, C5/C6 (Figure 2) and at L3/L4 levels (Figure 3). Laboratory tests, including complete blood count, comprehensive metabolic studies, coagulation studies, and inflammatory markers, were within normal limits. Based on clinical presentation, investigative findings, and possible association with the facet injection, a diagnosis of locked-in syndrome was established. The patient was diagnosed with locked-in syndrome secondary to the facet joint injection. For multidisciplinary care in the SICU, she was referred to the neurology, orthopedic spine, and rehabilitation teams. We started her on intravenous (IV) Parentrovite (preparation of vitamin B complex with vitamin C) and piracetam 1.2 grams twice daily (BID).

She slowly regained her muscle power as she was able to move her left upper limbs by 4 h of SICU admission; her right upper limb by 18 h, and her complete return to baseline muscle power for all limbs by 42 h later. The patient was discharged from the SICU to the general ward on day two of SICU admission and discharged home on day three. Upon discharge, she was comfortable in room air, able to ambulate, able to urinate, and passing motion; pain reduction by 30% was achieved. She was discharged with tablet diclofenac sodium 50 mg TDS and tablet paracetamol 1 gram four times daily (QID) as analgesic medications. The next appointment at the pain clinic was given two weeks later.

3. DISCUSSION

The development of LIS following a facet joint injection is an extremely rare and alarming complication. To our knowledge, this is the first reported case of LIS following a lumbar facet joint injection. LIS typically arises from vascular insults to the brainstem, most commonly the anterior pons.[2,3] The ventral pons is a critical region housing descending motor pathways essential for voluntary movement. Lesions in this area lead to the characteristic motor deficits of LIS while sparing sensory pathways and the reticular activating system, thus preserving consciousness and cognitive function.[2,3]

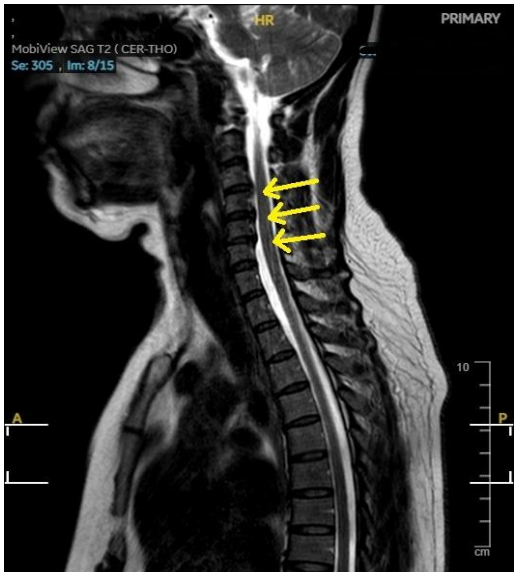

Figure 2: The MRI of the whole spine (focus on cervical) showed a posterior disc bulge seen at C3/C4, C4/C5, and C5/C6 (yellow arrows).

The classical type of LIS is characterized by complete immobility while preserving the ability to blink, move the eyes vertically, and remain conscious. The other type is the incomplete form, which is defined as the classical form with a few minor motor function additions.[3] In our case, the diagnosis of LIS was made at an initial phase because of the patient's typical presentation post procedure. There was a possibility she had an incomplete form of LIS. Based on the study, facet joint injections have been reported to be associated with an overall low rate of adverse events of 0.84% per case and 1.63% per patient.[4] The list of complications reported from highest to lowest includes pain at the injection site, somnolence, vasovagal reactions, intravascular injections, transient non-positional headaches, facial flushing, symptom aggravations, steroid-clogged needles, spine infections, and uncontrolled hypertension.[4] None of the reports showed the production of LIS post-facet injection. Therefore, we consider our case to be a unique presentation of a complication.

The development of LIS following facet joint injection raises questions regarding the underlying mechanism and potential contributing factors. While direct spinal cord injury or vascular compromise during the procedure cannot be entirely ruled out, the absence of acute structural abnormalities on imaging suggests alternative etiologies. Several hypotheses can be considered to explain this occurrence. The exact mechanism underlying this phenomenon remains uncertain but is hypothesized to involve inadvertent intravascular injection leading to embolic or thrombotic occlusion of the pontine arteries, resulting in ischemic injury to the

ventral pons. The literature states that the most common cause of LIS is a vascular complication in the form of a hemorrhagic or ischemic stroke.[3] In the context of facet joint injection, direct spinal cord injury, vascular injury, or embolic events may precipitate LIS. In our case, while direct spinal cord injury was unlikely given the fluoroscopic guidance, vascular compromise secondary to embolization or vasospasm cannot be excluded. Additionally, individual patient factors, such as pre-existing vascular compromise or anatomical variations, may predispose certain individuals to this complication. Our patient had an underlying SLE with ITP that may postulate the vascular variation of abnormalities.

The other precise mechanism underlying LIS post-facet injection may be related to inadvertent intrathecal injection. As a plane of synovial joints, lumbar facet joints create the articular pillars that support the entire vertebral column structurally and are located between the pedicle and lamina of the same vertebra.[5] The spectrum of facet joint disease, includes facet joint effusion, synovial cysts, osteoarthritis of the facet joints, septic arthritis of the facet joint, rheumatoid arthritis (RhA), and many more.[5] With the diverse spectrum of facet joint diseases, the pathophysiology of LIS post-facet injection is thought to result from the inadvertent injection of a local anesthetic (LA) or corticosteroid into the spinal cord pathway. The proximity of the spinal cord to the facet joints raises the possibility of injecting local anesthetics into the spinal circulation. This could lead to the LA effect, resulting in high spinal cord paralysis manifestations. However, in our case, we used lidocaine 1% as LA with diluted Shincort, which is unlikely to cause paralysis in the patient.

Diagnostic evaluation of LIS should include neuroimaging to rule out brainstem lesions, EEG to assess cortical function, and CSF analysis to exclude infectious or inflammatory causes.[3] Nerve conduction studies and EMG can help differentiate peripheral from central nervous system involvement.[3] In this case, we only proceeded with the neuroimaging technique because the motor power was regained to normal function after 48 h in the SICU. The management of LIS is primarily supportive, focusing on maintaining airway patency, providing nutritional support, and preventing complications such as pressure ulcers and contractures.[2,3] We managed the patient accordingly in the SICU with proper follow-up in the general ward.

To mitigate the risk of severe complications such as LIS, several preventive strategies should be considered in clinical practice. A detailed patient history and assessment of risk factors for vascular or neurological complications should guide decision-making. Pre-procedural imaging to assess vascular anatomy may be warranted in high-risk patients. Utilizing fluoroscopy or ultrasound guidance to ensure accurate needle placement can significantly reduce the risk of inadvertent injections, either vascular or intrathecally. Real-time visualization of the needle trajectory helps avoid critical structures. Employing the lowest effective dose of LA and steroids minimizes potential neurotoxic effects. Careful consideration of dosage and volume can reduce the risk of adverse events.

4. CONCLUSION

Locked-in syndrome is an exceedingly rare but potentially devastating complication of interventional pain procedures with profound implications for the patients' quality of life. Clinicians should be aware of this potential possibility when performing these procedures and maintain a high index of suspicion for any complications. Prompt recognition and appropriate management are essential for optimizing patient outcomes. This case underscores the importance of thorough pre-procedural evaluation, meticulous technique, and vigilant monitoring for early detection of neurological complications.

5. Ethical considerations

Written consent of the patient was obtained to publish her story for educational purposes.

6. Author contribution

MRH: Conduct the case, literature review, manuscript writing
SI, SKH, KI: Supervise the case, review of the manuscript, final approval of manuscript

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