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A Case of Spinal Epidural Lipomatosis Presenting as a Stroke Mimic

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Introduction:

Spinal Epidural Lipomatosis (SEL) an excess of normal adipose tissue in the epidural space of the spinal canal, typically becoming symptomatic when cord compression results. First described in 1975 by Lee et al (Lee M, 1975), it is a relatively rare disease. Unfortunately, it is often diagnosed once patients have developed marked symptoms and can have dire complications. Oftentimes SEL is completely asymptomatic, and symptoms on initial presentation are vague. Mostly commonly it presents as worsening chronic back pain (Daniel R. Fassett M.D. M.B.A., 2004) with progressive lower extremity weakness and occasional cauda equina syndrome (Wells AJ, 2014), but this is of course dependent on the level of the culprit lesions. What is unique about this case as compared to other instances of SLE, is that it presented as a stroke mimic. Ultimately little is known about the pathogenesis of this disease process, but generally SEL can be attributed to the following: exogenous steroids, excess endogenous steroid production from endocrine abnormalities, obesity, postsurgical changes, and idiopathic disease (Daniel R. Fassett M.D. M.B.A., 2004). With back pain being one of the most commonly encountered chief complaints in the ED, SLE proves itself as an example as to the importance of broad differentials and should be considered in cases with symptoms consistent with spinal cord impingement.

Case Report:

A 57-year-old male, 158 cm, 100.4 kg, presented to the emergency department for evaluation via EMS for right sided upper and lower extremity weakness with onset 3 days prior to arrival. Patient had been urged to present to the ED for evaluation by his family and PCP but had been adamant about not being hospitalized. As his weakness worsened his wife called 911. On arrival to the ED his presenting complaints were severe headache, inability to fully move his right lower and upper extremity, and acute on chronic back pain with radiation to right sided extremities. During initial triage he was noted to have right arm drift, right lower extremity weakness with no effort against gravity, and some mild dysarthria.

Past medical history was notable for HIV, Hepatitis C, hypertension, hyperlipidemia, and prior CVA with sensory/mild speech deficits. Surgical history was notable for exploratory laparotomy secondary to a stab wound as well as laminectomy in 1986 for a herniated disc. Patient was also noted to have a history of prior intravenous drug abuse and was currently non-compliant with most medications.

Vital signs on arrival were a blood pressure 119/59 mmHg, heart rate 57 beats per minute, respirations 19 breaths per minute, and temperature of 98.4 degrees Fahrenheit orally with oxygen saturation of 95% on room air.

The patient was awake, alert, and in moderate distress secondary to pain from his headache and back pain with weakness of his right upper extremity and inability to move his right lower extremity. He was normocephalic and atraumatic without obvious signs of head trauma. Neck was supple without midline tenderness or step-offs. Cardiac exam revealed normal rate without appreciable murmurs, rubs, gallops. Abdomen was obese, non-distended, without peritoneal signs, soft, without tenderness. Pulmonary examination was clear to auscultation and otherwise unremarkable. On neurological examination, patient had mild dysarthria, motor strength 3/5 on right upper extremity, 1/5 on the right lower extremity with 5/5 strength to his left upper and lower extremities. Sensation was intact throughout.

A stroke alert/response was initiated, patient point of care glucose testing was found to be 152, and he was taken down to CT scan. Initial non-contrast head CT was without acute intracranial abnormality. CT angiography of the neck/head was also without significant large vessel occlusion or high-grade stenosis and was essentially unremarkable. Remainder of work-up was unremarkable with normal hemoglobin without leukocytosis and cardiac enzymes, sodium, potassium, chloride, bicarbonate, BUN, creatinine all were within normal limits.
While the work-up was being pursued, patient had continued to complain of back right sided extremity pain which was being addressed with morphine. Patient had denied any signs or symptoms of saddle anesthesia and continued to report his back pain as acute on chronic pain that he's had "for years" from prior herniated discs. Given the extensive history of IVDA as well as prior spinal surgery, an MRI of the thoracic as well as lumbar spine were ordered. This revealed epidural lipomatosis involving posterior aspect of thoracic spinal canal with anterior displacement and compression of thecal sac. This was present at multiple levels but most prominent at T4-T8. Lumbar MRI revealed spondylosis and degenerative changes at L3-S1. Ultimately the patient was admitted to the hospital for further neurology and surgical evaluation but left against medical advice shortly afterwards.

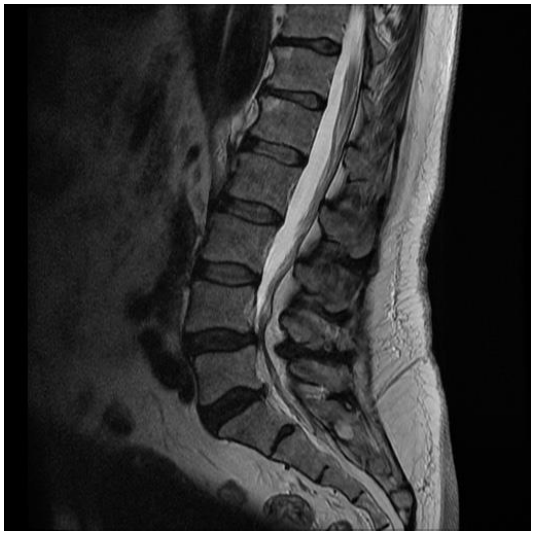

Figure 1: An example of fat deposits in the lumbar spine causing SLE
Case courtesy of Dr Matthew Lukies, Radiopaedia.org, rID: 57661

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Discussion:

The exact mechanism and pathogenesis of SEL is currently unknown, however it is grouped into four causative categories: exogenous steroid 55.3% of cases, endogenous steroid hormonal disease 3.2% of cases, obesity-associated disease 24.5% of cases, and 17% believed to be idiopathic (Guy R. Fogel MD, 2005). It is important to note that idiopathic cases have previously included cases in which obesity was an associated factor. However, recent trend has been to account obesity as a separate causative factor and for "idiopathic" cases to include non-obese individuals (Kim K, 2019). Exogenous steroids are regarded as the most significant and common factor in the development of SLE (Guy R. Fogel MD, 2005). Also, males are commonly more at risk as compared to female populations (Al-Khawaja D, 2008).

Our patient was unique in this case as he did not appear to be taking any recent exogenous steroids, as this is the single most important risk factor for the development of SLE. The proposed mechanism for this is that exogenous steroids cause one to develop excess adipose tissues on the face, neck, trunk and mediastinum and could thusly predispose to accumulation of fat in the epidural space. Development of SLE in patients receiving steroids was not correlated with dosing (Christian Kocha, 2000) and can occur with doses as small as 5mg of prednisone a day. However, most cases are associated with longer therapy over 1 months duration or with doses of prednisone exceeding 30mg per day (Toshniwal PK, 1987). It should be noted that while this patient did not suffer from any primary endocrine disorders, diseases such as hypothyroidism, Cushing syndrome, carcinoid tumor have been previously associated with the development of SLE (Kim K, 2019).

Still our patient did possess certain risk factors. Primarily the patient was obese with a BMI of 40. He also had a reported history of spinal surgery, with a history of intravenous drug abuse and was HIV positive though only intermittently compliant with medications. These factors placed him at higher risk for the development of SLE as compared to the general population. Obesity is a well-known criteria associated with this diagnosis, but there have also been reports that HIV positive patients are also at risk for slightly higher incidences of SLE. This is also especially true for those on highly active antiretroviral therapy (HAART) (Kim K, 2019).

Initial Symptoms of SLE typically consist of chronic back pain, but as the lesions enlarge and cause nerve or spinal cord compression a constellation of symptoms can develop: lower extremity weakness, paresthesias, numbness, radiculopathy, myelopathy, claudication, paraplegia and loss of bowel and bladder control (Kim K, 2019). In our patient's case, he presented with the classic duo of back pain and lower extremity weakness. In addition, his lesion was also in the most common areas for SLE as thoracic involvement account for nearly 60% of cases (Lumbar involvement accounts for 39 to 42% of cases and cervical SEL has not yet been reported) (Daniel R. Fassett M.D. M.B.A., 2004). Still the most striking aspect of his case is that he initially presented as a stroke mimic. His overall clinical picture was complicated by several factors. His pain was described as acute on chronic and he was noted to have some slight unexplained dysarthria on initial presentation. This was further complicated by his prior history of CVA.

Treatment for SEL can be divided into three major categories: Surgical, cessation of offending agent, reversal/treatment of predisposing disease. Given the small number of cases of SLE that have been diagnosed and managed, no clinical trials of different therapies have been compared for superiority. Thus, it remains important to take and individualized approach to each patient and to assess risks and benefits of surgery as compared to conservative management after magnetic resonance imaging and computed topography for grading of severity. Ultimately, weight loss and cessation of offending agent/correction of endocrine disorders has been shown to be the most effective treatments for milder or more chronic cases. Multi-level decompressive laminectomy and debulking remain a mainstay for acute cord compression secondary to SEL, but mortality rates 1 year post operatively have been shown to be as high as 22% (Fessler R, 1992). This is mostly attributed to this patient base usually being immunocompromised and with a high number of comorbidities. When applicable, it is there fore suggested that conservative management with weight loss, cessation of offending agent, and management of causative disease states be performed when possible.

Conclusion:

Spinal epidural lipamotasis is rare and misleading disease that commonly presents on initial eval with vague and non-descripts symptoms of back pain and radiculopathy. It can quickly progress to rapid spinal cord impingement and in the right clinical setting require extensive surgical management with high rates of mortality. This rare diagnosis should remain at the forefront of a clinician's mind in the right clinical setting: acute on chronic back pain with lower extremity weakness, or other neurological deficits, especially in the setting of hyper-cortisol states, prior spinal surgery, or HIV positive patients. Early diagnosis, though difficult, could have drastic consequences for a patient and lead to far improved outcomes. A brief review of available literature was conducted, and SLE has never before been identified as a stroke mimic (Vilela, 2017) (Boulter, 2014) (Huff, 2002) (Hosseininezhad, 2017). Thusly, should especially be remembered in situations where CVA has been ruled out and the patient has sufficient risk factors.