



## Case of the Month

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### This “TUNA FISH” is Missing Something

#### Introduction

“TUNA FISH” is a very popular mnemonic that we use to recall the red flags for low back pain. It is just one out of ostensibly innumerable mnemonics used every day by Emergency Physicians to rapidly organize and memorize vital pieces of information pertinent to clinical practice. We have got to maintain fresh and readily available knowledge about anything at any time. However, does this seemingly accessible memory tool work all the time? The following is a comprehensive discussion of an encountered rare and crucial neurosurgical diagnosis, presenting to the Emergency Department initially with low back pain with no “TUNA FISH” red flags!

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- U** UNEXPLAINED WEIGHT LOSS
- N** NEUROLOGIC SYMPTOMS
- A** AGE >50
- F** FEVER
- I** INTRAVENOUS DRUG USE
- S** STEROID USE
- H** HISTORY OF CANCER

## Case Summary

A 28-year-old male, known to have lumbar disc disease on conservative management and Hemophilia A, was encountered on his third visit to the Emergency Department (ED) complaining of worsening back pain that started 1 week prior to presentation. The pain was reportedly sharp, located in the lower back, worsening with any movement, radiating to both thighs, more to the right, with recently developing bilateral lower limb numbness. No reported loss of bowel or bladder control and no saddle anesthesia was present. He stated that he regularly performs weight-lifting exercises with no recent increase in the practiced weight. He denied any recent trauma or fall. On his first ED visit, the complaint was solely low back pain radiating to bilateral lower limbs, for which he underwent X-Ray imaging of the lumbo-sacral spine, reported with no significant findings. Hence, he was diagnosed with Lumbar muscle spasm and discharged on analgesics with a referral to the Neurosurgery clinic.

On his follow up visit at the Neurosurgery clinic, the patient was booked for a Magnetic-Resonance Imaging (MRI) of the lumbosacral spine and discharged on more potent analgesics. However, due to worsening pain not adequately responding to the analgesics, patient sought another medical opinion and had emergent MRI done at another facility, which showed large epidural hematoma extending from L5 to S1 and compressing L5 nerve roots.

On examination the patient was in mild distress, walking into the room with antalgic gait. His vital signs were within normal limits. On inspection, there was an old lipoma at the lumbar region. No tenderness was evident on palpation. Pain was reproducible, however, with lumbar flexion and extension. A positive straight leg raise test was elicited on the right side.

Patient was initiated on intravenous (IV) analgesics and Factor VIII replacement therapy at 50IU/Kg to achieve 100% desired increase. The neurology service was consulted and were still in doubt of the provided report and requested a confirmatory report from the radiologist in house. Based on the confirmatory report, the patient was diagnosed with spontaneous spinal epidural hematoma (SSEH) and admitted for observation and conservative management. A follow up MRI study after 2 weeks showed resolving hematoma.

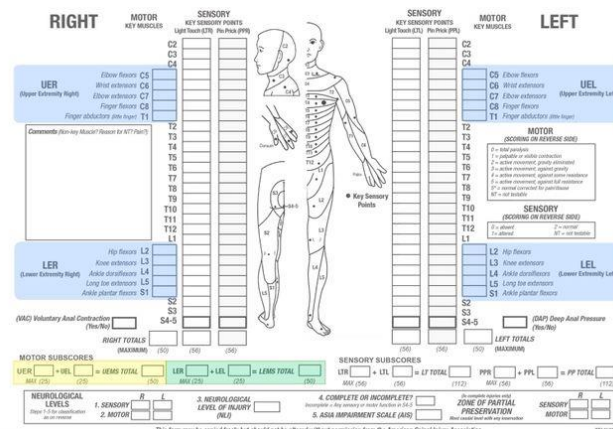
## Discussion

**Spontaneous Spinal Epidural Hematoma (SSEH)** is relatively rare neurological condition, with an estimated annual prevalence of 1 in 100,000 individuals and slightly higher incidence in males compared to females (1.4:1).<sup>(3)</sup> There is still no conclusive definition of the term “Spontaneous” in SSEH. Some authors exclusively label idiopathic hematomas as “Spontaneous”, while several more extend the definition to include hematomas that are secondary to vascular malformation, hypertension, coagulopathy, and tumors.

**The Most Common Affected Site** is the cervicothoracic or thoracolumbar spinal area, usually posterior to the thecal sac. There has been a dispute regarding the source of bleed. Some researchers have postulated that these hematomas develop from a ruptured epidural venous plexus, referring to its valveless structures and thin walls that makes it vulnerable to any sudden increase in intra-thecal pressure. However, some authors argue that epidural venous pressure is not strong enough to overcome the intravascular-intrathecal pressure gradient and produce a mass effect. This, in turn, supports the proposed hypothesis of arterial origin of the epidural spinal hematomas. A relatively fair supposition is that, when there is a rapid progressive neurologic decline, the source is likely to be arterial, while a slow progression of symptoms likely indicates venous source.

**Clinical Manifestation** ranges from simple back pain with radiculopathy to complete paraplegia or quadriplegia due to cord compression, depending on the location and degree of spinal cord compression. Due to the fact that the initial symptom is usually intense back pain mimicking that of disc prolapse, SSEHs diagnosis might be missed or overlooked. Hence, ED physicians should expand their list of differentials for back pain and keep a high index of suspicion with coexisting predisposing factors for epidural bleed.

**The Initial Assessment** of SSEH should address two main factors; a) the degree of neurological deficit and b) time interval between symptoms and surgical intervention. These two factors play a role in post-operative outcomes. Patients who develop severe symptoms involving four or more of the spinal segments within a short time frame tend to have worse outcomes. Examples of the clinical tools used to assess the severity of neurological deficits include: ASIA Impairment scale (Figure1. A) and the Frankel scale (Figure1. B).

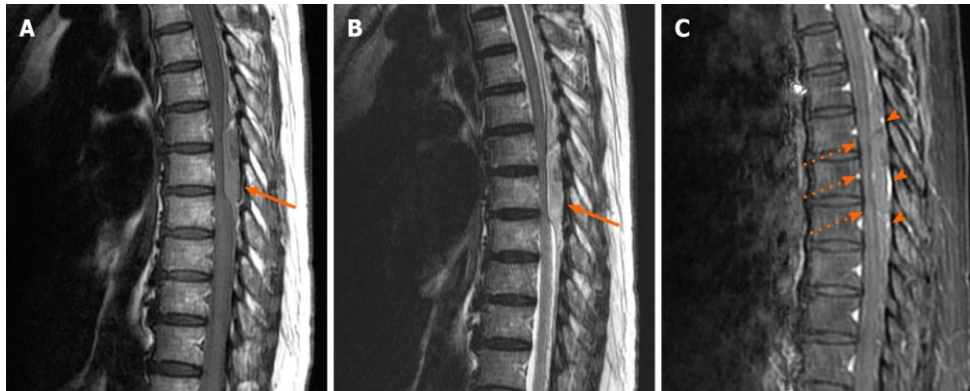


**Figure1. A:** ASIA Impairment scale

Frankel grading	Description
A	Complete motor and sensory loss
B	Complete motor loss, incomplete sensory loss
C	Incomplete motor loss without practical use
D	Incomplete motor loss, able to ambulate with or without walking aids
E	Free of neurological symptoms

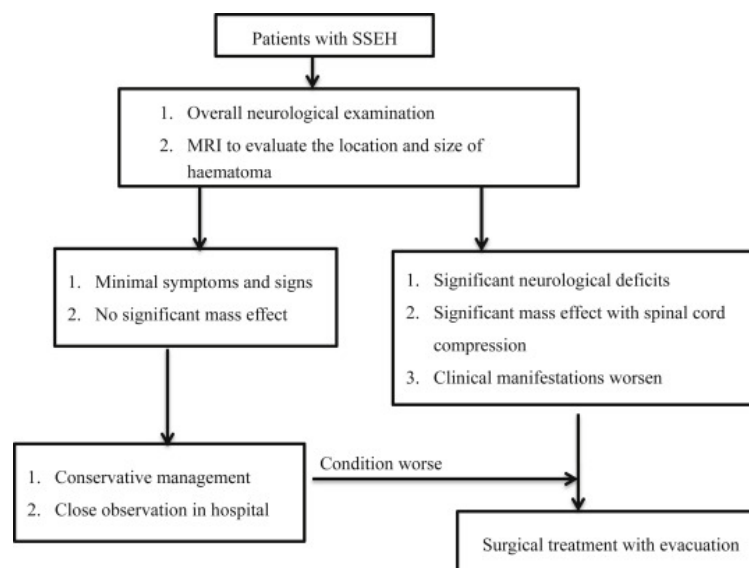
**Figure1. B:** Frankel scale

The **Imaging Modality** of choice is an emergent MRI when SSHE is suspected. Within the first 24 hours the hematoma typically appears isointense on T1-weighted (Figure2.A) and hyperintense on T2-weighted MRI (Figure2.B). After 24 hours, the hematoma often appears hyperintense on both T1- and T2-weighted images. Chronic hematomas become hypointense on both T1- and T2-weighted images. Active bleeding into the hematoma will reveal a central area of enhancement when contrast is used (Figure2.C).<sup>(4)</sup>



**Figure2:** Spinal magnetic resonance imaging demonstrating an intraspinal epidural hematoma at the T4 to the T8 levels (arrows). A: Sagittal T1-weighted image (T1WI); B: Sagittal T2-weighted image; C: Sagittal T1WI with enhancement showing mildly thin peripheral enhancement (arrowheads) and the spinal cord is compressed and flattened (dashed thin arrows)

The **Management** of SSHE is surgical decompression and hematoma evacuation through laminectomy. Non-operative management is reserved only for candidates with improving neurological deficits in the early phase or with coexistence of coagulopathy. These patients should be monitored with serial examinations while on strict bed rest. Patients managed non-operatively may benefit from dexamethasone to decrease the compressive effect of edema. Shown below (Figure3.) is a treatment strategy suggested by *Tao Lan* and colleagues, published by the *journal of orthopedic translation in 2015*.<sup>(8)</sup>



**Figure 3:** Treatment strategy for patients with spontaneous spinal epidural hematoma (SSEH)

## Conclusion

While this patient was known to have lumbar disc disease, a significantly crucial and time sensitive red flag was overlooked in this case. Hemophilia patients with acute on chronic low back pain that is not improving with analgesics is a striking history that should trigger prompt suspicion of spontaneous spinal epidural hematoma. Perhaps you can add “History of Coagulopathy” to your “TUNA FISH” mnemonic

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