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Thoracic spinal epidural abscess caused by fishbone perforation

A case report and review of literature

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Abstract

Rationale: Ingestion of a fishbone is a common cause of esophageal injury, but spinal epidural abscess (SEA) is a rare condition due to the esophageal penetration by a swallowed fishbone. Prompt diagnosis can be seldom made owing to incomplete patient history taking and difficulties in imaging evidence identification.

Patient concerns: We describe the case of a 62-year-old woman who was stuck in her throat by a fishbone, and complained of back pain, paresis of the lower limbs and fever, successively. To our knowledge, this is the first case report that we know of thoracic SEA caused by fishbone perforation.

Diagnoses: About 20 days after the onset of severe back pain, she was diagnosed with SEA based on the clinical presentation and imaging findings.

Interventions: Antibiotic therapy and rehabilitation therapy were carried out afterwards. However, due to exacerbation of her condition, surgical intervention had to be taken eventually.

Outcomes: It is quite unfortunate for this patient to have a poor prognosis due to a delayed diagnosis and an improper management.

Lessons: A number of lessons can be learnt from this case.

Abbreviations: ASIA = American Spinal Injury Association, CRP = C-reactive protein, CT = computed tomography, ESR = erythrocyte sedimentation rate, MRI = magnetic resonance imaging, PCT = procalcitonin, SEA = spinal epidural abscess, WBC = white blood cell.

Keywords: diagnosis, fishbone, management, spinal epidural abscess, surgery

1. Introduction

Spinal epidural abscess (SEA) is a rare clinical condition with a relatively high rate of associated morbidity and mortality.[1,2] It is much rarer due to the esophageal penetration by a swallowed fishbone, though ingestion of a fishbone is a common cause of esophageal injury. As its low incidence limits many health care practitioners exposure to this pathology, approximately 50% of patients are initially misdiagnosed at the time of presentation.[1] Obviously, early diagnosis leading to prompt treatment is regularly rewarded by better recovery.[3] We present a case of thoracic SEA caused by fishbone perforation, which has never been reported in the English literature.

2. Case presentation

A 62-year-old woman consulted the emergency department of a local county hospital due to a sudden onset of severe back pain 6 days ago. She did not have any other past medical history. After admission, digestive tract radiography failed to find any abnormality in her stomach and duodenum. No abnormalities but a few enlarged lymph nodes were revealed by chest computed tomography (CT) scan. Four days later, however, she began to feel nausea and vomit with a frequency of over 10 times per day.

The patient was immediately transferred to Zhangzhou Municipal Hospital and admitted to the emergency department. Routine blood testing was taken and the results are summarized in Table 1 (the first time). On the third day after admission, she complained of weakness and numbness over bilateral lower limbs, as well as difficulty in urinating. An immediately conducted thoracic CT scan found multiple small regions of gas in spinal canal at the level of T4 and below (Fig. 1), which indicated that the patient had a specific infection in the mediastinum and thoracic spinal cord. Further enquiry about her recent history revealed that there was an event of a fishbone getting stuck in her throat 16 days before admission to the local county hospital. It occurred to the physicians that her condition could be related to the fishbone.

Afterwards, the patient was further transferred to the First Affiliated Hospital of Fujian Medical University and admitted to the emergency department. On physical examination, paraplegia (muscle power scored as 0/5 in bilateral lower limbs), decreased

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Ethical approval was not necessary for this report, as it was focused on the patient hospital course and did in no way alter her treatment.

However, the patient provided informed consent for the publication of this case report.

The authors report no conflicts of interest.

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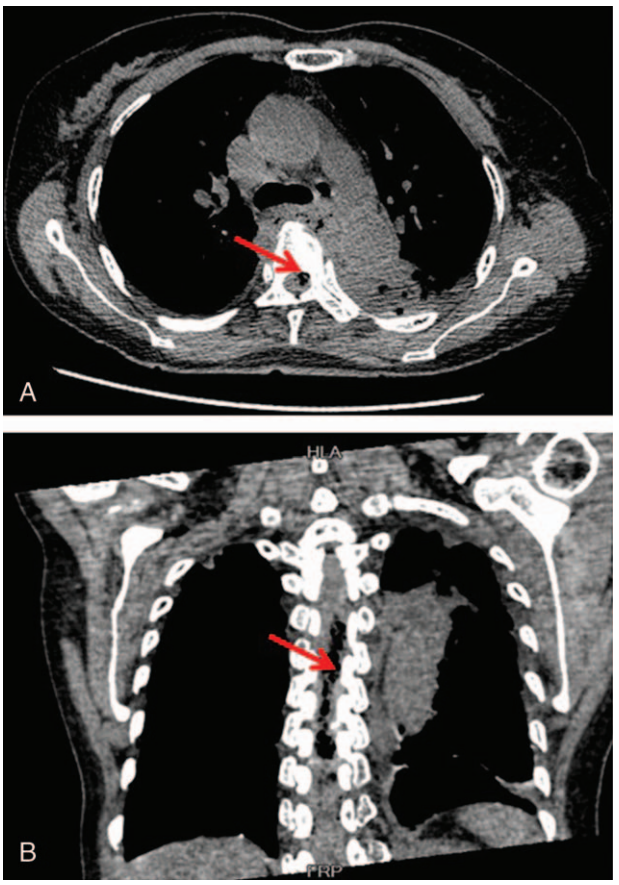

Figure 1. Sagittal (A) and axial (B) plane CT of spine (taken on September 3, 2015) showing a number of small regions of gas (red arrows) in spinal canal at the level of T4 and below.

sensation below the T4 dermatome was discovered. Her ASIA (American Spinal Injury Association) motor score was 50. Postcontrast CT scan showed pus accumulation with multiple air bubbles in anterior mediastinum and left pleural cavity, spinal canal being involved as well (Fig. 2). Results of routine blood testing for this time are summarized in Table 1. She was given antibiotic therapy with vancomycin (1.0 g/12 hours) and meropenem (1.0 g/12 hours) administered intravenously on the first day of admission. It was on the second day when her blood sample was collected for culture. Unfortunately, neither bacterium nor epiphyte could be detected. On the third day, her temperature rose to 39.8°C. Thoracic paracentesis was conducted with subsequently negative results from abscess smear and cultural examinations, respectively. On the fifth day, magnetic resonance imaging (MRI) of the thoracic vertebra demonstrated epidural abscess at the level of T4 to T5, spondylitis of T4 and T5 with T4-5 discitis and spinal cord compression, causing spinal cord compression at these levels (Fig. 3). On the basis of the clinical presentation and imaging findings, a diagnosis of SEA was made eventually on the day about 1 month after a fishbone was stuck in her throat, or about 20 days after she complained of severe back pain.

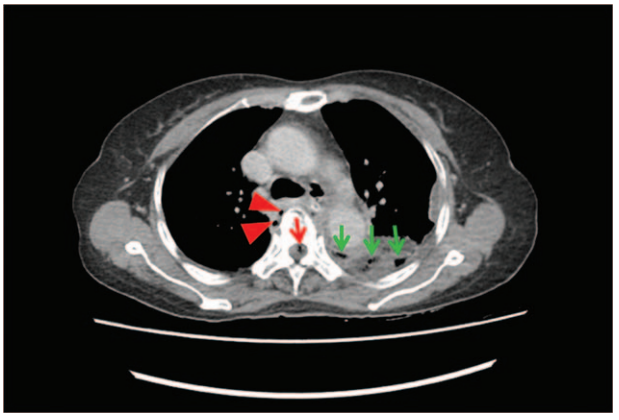

Figure 2. Sagittal post-contrast CT scan image (taken on September 5, 2015) showing pus accumulation with multiple air bubbles in anterior mediastinum (red arrow heads) and left pleural cavity (green arrows), as well as a small air bubble in spinal canal (red arrow).

The patient was treated with antibiotics continuously for another 26 days. Fever subsided and back pain was relieved a few days later. Routine blood testing (the third time) revealed normal results (Table 1). However, the power of the lower limbs remained unchanged. The patient was transferred to the rehabilitation department for comprehensive rehabilitation. Her temperature kept normal during this period. The power of the proximal lower limbs gradually increased to grade 2/5. However, 23 days later, the power of the bilateral lower limbs decreased. Follow-up enhanced MRI observed granulation tissue in the epidural region and epidural abscess collected at the level of T4-5 and below (Fig. 4). She was then transferred to the orthopedic department. On the second day, her temperature rose to 39°C. Laboratory data (the fourth time) are summarized in Table 1. She was given antibiotic therapy with vancomycin (1.0 g/12 hours) and fever subsided 5 days later. The patient was treated with antibiotics continuously for another 17 days before total laminectomy was undertaken at T4 to T5 with percutaneous pedicle screw fixation at T3 to T4 and T6 to T7. The abscess and lesion (composed of sequestrum fragments and inflammatory granulation tissue) were removed without collecting samples for pathology and culture. One day after the operation, she experienced fever and was treated with vancomycin (1.0 g/12 hours) for another 10 days. Later on, the power of the proximal lower limbs gradually increased to grade 2/5. However, there was no improvement in bladder control. The ASIA motor score was still 50. She was then discharged with normal temperature, accompanied with normal results of routine blood testing and erythrocyte sedimentation rate (ESR) level, as well as a slightly high C-reactive protein (CRP) level (Table 1). No further improvement was obtained in motor function and bladder control 6 months postoperatively. Follow-up thoracic MRI showed complete resolution of epidural abscess with fusion of T4-T5 (Fig. 5).

3. Discussion

This case illustrates a rare presentation of SAE caused by fishbone perforation. In those countries where fish is often consumed, fishbone is perhaps the most commonly ingested foreign body that becomes impacted in the upper alimentary tract and causes alimentary tract penetration.[4] Quite luckily, fishbone is often lodged in the tonsil or tongue and easily removed. Nevertheless, whenever a fishbone is stuck in the throat without removal, serious complications may occur. This case highlights the need to consider the possibility of a fishbone being the underlying cause in any case of unexplained back pain or mediastinum

Table 1

Routine blood testing results of the patient at different times*.

	WBC, /mm^3	CRP, mg/L	PCT, ng/L	ESR, mm/h
1st time	14,660	NA	3.97	NA
2nd time	16,430	88.3	1.88	NA
3rd time	7280	38.8	0.23	NA
4th time	4960	20.97	0.23	44
5th time	4840	15.65	NA	36

CRP = C-reactive protein, ESR = erythrocyte sedimentation rate, NA = not available, PCT = procalcitonin, WBC = white blood cell.
* Routine blood testing was taken on September 1, 2015, September 4, 2015, October 5, 2015, November 6, 2015, and December 16, 2015, respectively.

inflammatory process so that such condition can be diagnosed and treated in a timely manner.

In the literature, a number of complications related to fishbone were ever reported, including vocal cord paresis,[5] hepatic abscess,[6] aortic-oesophageal fistula, subclavian oesophageal fistula and carotid rupture,[5] subclavian artery pseudoaneurysm,[7] aortic pseudo-aneurys,[8] false esohageal hiatus hernia,[9] and pharyngeal perforation.[10,11] In addition, fish bone injury may also cause retropharyngeal abscess, which may further lead to cervical SEA,[12–15] or even brain abscess[14] (Table 2). SEA is an infection that forms in the space between the bones of the spine and the lining membrane of the spinal cord.[16] In this study, a thoracic SEA case was presented with a clearly different mechanism from that of cervical SEA. The esophagus is located in the septum. Mediastinitis is often due to the perforation of esophagus, trachea, or bronchus. In this case, it was believed that a fishbone perforated the esophagus, led to a local infection of esophagus (alimentary tract), which was followed by a bout of bacteremia. Thereafter, mediastinitis was developed with bacteria entering the mediastinum through hematogenous spreading or direct extension from the infection of esophagus. Consequently, a few abscesses were formed in the mediastinum. The infection further spread to thoracic spinal epidural space and, eventually, formed SEA.[17–19] As a rare clinical condition, its low incidence limits many physicians exposure to this pathology. Hence, physicians should pay extra attention on patients with fishbone-related throat injury, and be aware of SAE induced by such injury.

Blood or/and abscess cultural test is crucial to identify the causative organisms. It was reported that several causative organisms may cause SEA, including *Staphylococcus aureus*, Gram-negative bacteria, coagulase-negative bacteria, staphylococcus, *Escherichia coli*, *Pseudomonas aeruginosa*, streptococcus species, and *Mycobacterium* spp.[2,20,21] Among them, *S. aureus* is the leading agent.[22] Notably, the sensitivity of blood cultural test may decline with pre-use of antibiotics. As such, blood/abscess specimen should be collected for cultural test before antimicrobial therapy. Once the causative pathogen is identified, antibiotic treatment should be adjusted accordingly.[23] In this case, antibiotics of broad-spectrum had been used on the time of admission without cultivating the causative microorganism from blood and abscess, which, quite likely, relates to the negative results of the subsequently cultural tests.

Diagnosing SEA can be challenging for clinicians. A large number of SEA patients may either be misdiagnosed at the time of presentation or suffer from a significantly diagnostic delay. The misdiagnosis can occur for up to 74% of patients, which leads to poorer outcome.[18] Diagnosis of SEA should be based on clinical presentation (back pain, motor weakness, fever, sensory abnormalities and bladder, bower incontinence, etc) with support from laboratory examination and imaging data.[21] MRI is crucial to establish the diagnosis and monitor the disease progression.[20,24] Especially, gadolinium-enhanced MRI is the most sensitive imaging method,[22] which should be taken as soon as SEA is suspected.[19] Relatively, CT scan is less specific and cannot distinguish SEA from other lesions that compress the

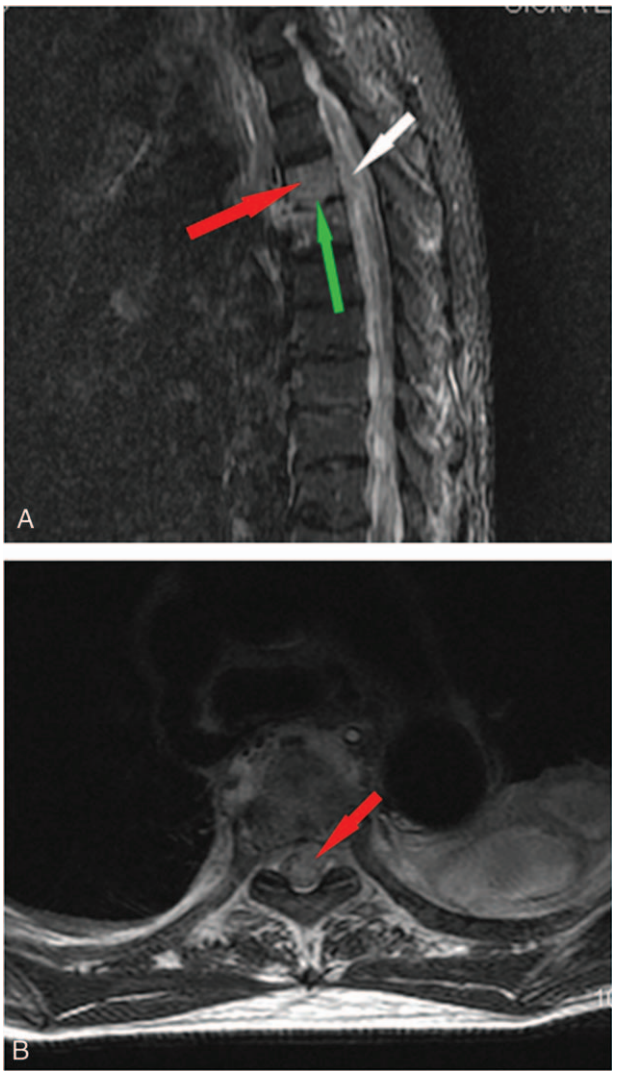

Figure 3. (A) Sagittal T2-weighted MRI image (taken on September 10, 2015) with fat saturation showing epidural abscess at the level of T4 to T5, spondylitis of T4 and T5 (red arrow) with T4-5 discitis (green arrow) and spinal cord compression (white arrow). (B) Axial T2-weighted MRI image showing epidural abscess with spinal cord compression (red arrow).

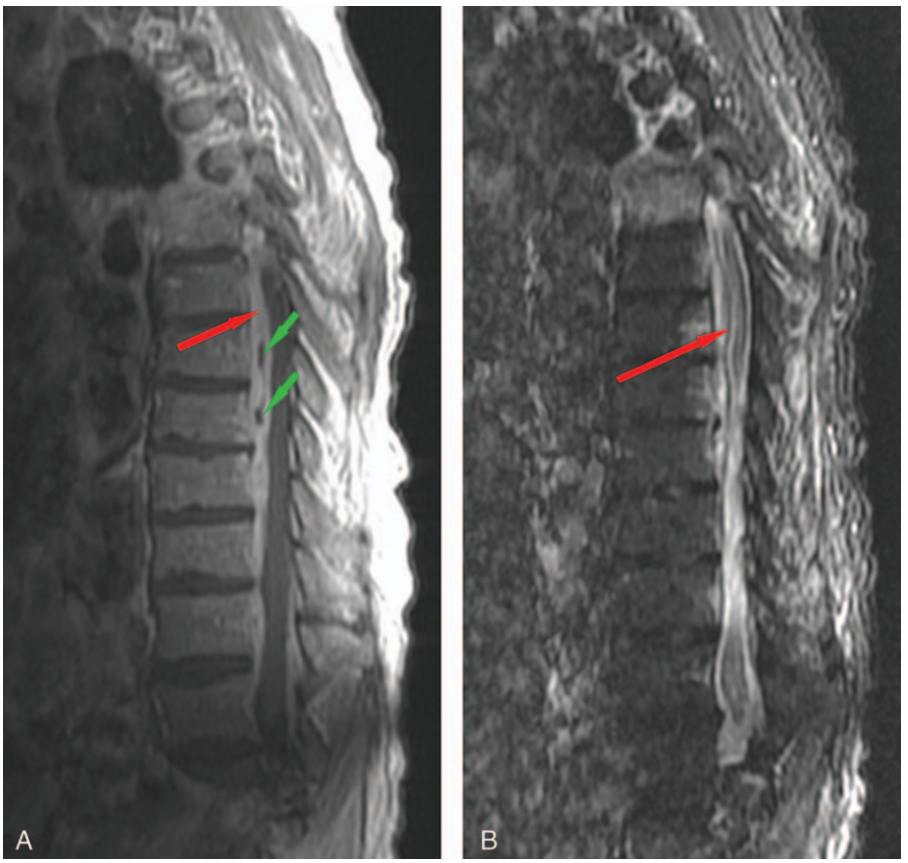

Figure 4. (A) Sagittal postcontrast T1-weighted MRI image (taken on November 2, 2015) showing the extensively enhanced granulation tissue in the epidural region (red arrow) with nonenhancing area representing epidural abscess collection at the level of T4-5 and below (green arrows), as well as the corresponding spinal cord being compressed. (B) Sagittal T2-weighted MRI image with fat saturation showing the epidural hyperintensity (red arrow) in the corresponding region.

thecal sac.[17] In addition, laboratory tests are worth evaluating, such as white blood cell (WBC) count, CRP, and ESR.[21] As for this case, the patient presented with back pain, paraplegic, and fever, with positive results of ESR, CRP, procalcitonin (PCT), and WBC. On the basis of the clinical presentation and imaging findings, a diagnosis of SEA was made, but a much delayed one. This is mainly due to incomplete patient history taking and difficulties in imaging evidence identification.

To date, there is no consensus or guidelines for the management of SEA. Clinical recommendations are based mostly on results of studies and on expert opinions.[25] An emergent surgical decompression was recommended as early as the diagnosis was made, regardless of neurological defects.[26] Likewise, others regarded neurological defects as the major determinants for treatment options. Patients who have only back pain without other neurological sequelae or spinal instability are

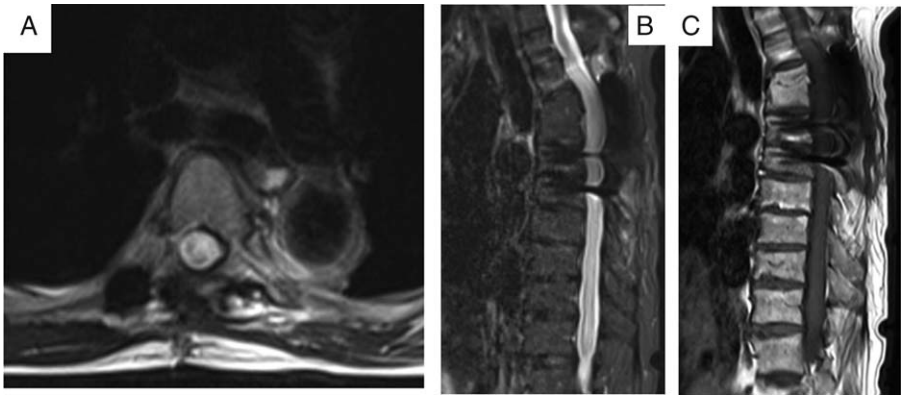

Figure 5. Follow-up MRI images of thoracic spine 6 months postoperatively (taken on May 10, 2016). Axial T2-weighted image (A) and sagittal T2-weighted images with (B) and without (C) fat saturation showing complete resolution of epidural abscess with fusion of T4-T5 (image torsion is due to metal artifact).

Table 2

The microbiological, clinical, therapeutical characteristics, and outcome of epidural abscess cases due to fish bone.

Year of study [Reference]	Patient	Microbiology	Clinical manifestation	Spine level	Treatment	Outcome
The present study	62-year-old woman in China	Not detected	Back pain, motor weakness, fever, sensory abnormalities, and bladder/bower incontinence	T4-T5	Antibiotic therapy, surgical drainage, and decompression	Paraplegia and bower incontinence remained 6 mo later
1990 [12]	12-year-old girl in Liberia	Not examined	Dyspnea, dysphagia, fever, and neck pain	C1-C2	Fishbone was removed through a neck incision	Recovered
1997[13]	53-year-old woman in Taiwan	*Streptococcus viridians* (abscess and blood culture)	Fever, chills, nuchal pain, respiratory distress, and severe weakness of four limbs	C1-C2	Antibiotic therapy, surgical drainage	Quadriplegia remained on the 96th day.
2007[14]	52-year-old woman in India	Not examined	Headache, throat foreign body sensation, neck pain, and fever	C2-C3	Not mentioned	Died
2012[15]	72-year-old man in Korea	*Eikenella corrodens* (abscess culture)	Dyspnea, neck and shoulder pain, weakness on right side	C3-C4	Antibiotic therapy, surgical decompression, fishbone removed	Right hemiparesis and left hypesthesia remained 1 y later

suggested to receive medical or conservative management with close monitoring for worsening neurological deficits.[21] The upper time limit for considering surgical intervention was thought to be 36 to 72 hours from the onset of neurological sequelae.[25] Medical management could be considered with initiation of intravenous use of broad-spectrum antibiotics for the patient, either being a poor surgical candidate due to debilitating medical illness, or with an extensive multilevel abscess or pan spinal disease that precludes adequate drainage, or having complete paralysis of 48 to 72 hours duration.[17,19] On the contrary, for those without neurological deficit or high surgical risk, CT-guided needle aspiration combined with antimicrobial therapy may be an option if they do not respond satisfactorily to antimicrobial treatment alone.[23] Obviously, there are some doubts about treatment options for this patient. Surgical intervention should have been considered within 72 hours from the onset of neurological sequelae. In addition, antibiotic therapy should have been carried out till the disappearance of abscess. Neurologic prognosis depends upon prompt diagnosis and duration of neurological deficits before surgery.[23] It is therefore not surprising of a poor neurologic prognosis for this patient.

4. Conclusion

A rare case of thoracic SEA caused by fishbone perforation was described in this study. It is quite unfortunate for this patient to have a poor prognosis due to a delayed diagnosis and improper management. A number of lessons can be learnt from this case. Physicians should pay extra attention on patients with fishbone-related throat injury, and be aware of SAE induced by such injury. Second, blood/abscess specimen should be collected for cultural test before antimicrobial therapy. Third, gadolinium-enhanced MRI is recommended to be taken as soon as SEA is suspected. Finally, proper treatment option should be taken in a timely manner.

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