Spinal Subdural Abscess Following Food Intoxication: A Case Report

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Abstract:

Introduction: Spinal subdural abscess (SSA) or empyema is a rare pathology and its exact incidence is unknown. *Staphylococcus aureus* (*S aureus*) is the most frequently responsible organism. The patients with SSA may have one or more predisposing immunosuppressive conditions. However, here we report a rare case of SSA following food intoxication without any significant comorbidities.

Case Report: A 42-year-old healthy man presenting with fever, severe low back pain (LBP), and trunk motion restriction was transferred to our hospital. He had been treated for an unknown fever after food intoxication in another hospital. Eighteen days earlier, he and his colleagues together ate raw horse meat and briefly boiled chicken breast. They all had food intoxication on the following day. Subsequently, our patient began to have a high fever and severe LBP. Laboratory data showed leukocytosis of 16,000/mm³. Also, the C-reactive protein was elevated to 26 mg/dL. The blood culture result was consistent with *S aureus*. Magnetic resonance imaging (MRI) showed focal epidural fluid collection that appeared contiguous with the subdural fluid collection through a dural defect in the axial plane on T2-weighted (T2W) images. An emergent surgery was performed. Frank pus was expressed from the epidural space as well as from the subdural space through the defect. The pus later grew *S aureus*. The patient was started on antibiotic therapy postoperatively. The patient completely recovered 1 month after surgery.

Conclusions: SSA following food intoxication is a very rare case. SSA can be identified with a small dural defect and the intrathecal fluid collection compressing the cauda equina in the axial plane on T2W magnetic resonance images. Having suspicion of epidural abscess and likewise subdural abscess and making an early diagnosis using MRI and an emergent surgery are important when the clinician notices a febrile patient with severe LBP and trunk motion stiffness. **Keywords:**

spinal subdural abscess, empyema, food intoxication

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Introduction

Spinal subdural abscess (SSA), also known as spinal subdural empyema, is a rare pathology, and its exact incidence is unknown. Ramos et al. stated that only 66 cases in adults have been reported in the English language literature by 2016¹). Another author described that only just a hundred cases have been reported, which are much less common than spinal epidural abscess²). In the majority of cases reported to date, *Staphylococcus aureus* is the most frequently implicated causative agent^{1,3,4}). The majority of the patients with SSA may have one or more predisposing conditions, such as diabetes mellitus, alcoholism, immunosuppressive drugs, chronic renal failure, malignant tumors, intravenous drug abuse, rheumatic heart valve disease, and tuberculosis⁵). In addition, a large group of patients had preexisting spinal disease, and all of these patients had surgery or injections performed¹). We describe here a case of SSA following food intoxication in a 42-year-old man with no significant comorbidities who presented with prolonged fever, severe low back pain (LBP), and difficulty in urination.

Case Presentation

A 42-year-old healthy man without any metabolic disease or spinal disorders presenting with prolonged fever and se-

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Figure 1. Preoperative sagittal MR images demonstrating focal collection that showed heterogeneous intensity in T1W (*left*) and high intensity in T2W (*right*) posteriorly compressing the thecal sac (*arrow*).

vere LBP was transferred to our hospital from another hospital. He had been treated for an unknown fever after food intoxication with fluid infusion and antibiotics, but in vain. Eighteen days earlier, he and his colleagues together ate raw horse meat and *tori-wasa* (briefly boiled chicken breast normally served cold and mostly raw as sashimi with wasabiseasoned soy sauce) at a Japanese-style bar. They all had food intoxication on the following day. Subsequently, only our patient began to have a high fever and severe LBP.

The patient complained of intolerable LBP as well as numbness and dullness in both thighs and in the sacral area. He also reported difficulties in urination. Furthermore, he had a slight headache that suggested a meningeal sign, but he did not have symptoms such as seizure, vomitus, neck rigidity, or consciousness loss. Manual muscle testing of the lower extremity was intact. Trunk motion was severely restricted with stiffness. He also did not have any distant infectious lesion such as dental caries, paranasal sinusitis, or otitis media, which could have spread hematogenously. He neither had a history of lumbar puncture nor spinal injection. Laboratory data showed leukocytosis of 16,000/mm³ with a left shift. The C-reactive protein (CRP) was 26 mg/ dL. The blood culture result was consistent with *S aureus*.

Magnetic resonance imaging (MRI) without contrast revealed focal collection that exhibited heterogeneous intensity on T1-weighted (T1W) images and high intensity in T2weighted (T2W) images, posteriorly compressing the thecal sac at L3-L4 (Fig. 1) in the sagittal plane. Epidural fat tissue coexisting with epidural fluid collection was observed on the focus. In the axial plane of T2W image, epidural fluid collection appeared contiguous with the subdural fluid collection through the small dural defect (Fig. 2). This fluid collection compressing the cauda equina in and out of the thecal sac indicated not only the epidural but also the subdural abscess. MRI with gadolinium contrast was not performed because MRI without contrast provided us with sufficient information that our patient had both the epidural and subdural abscess of the spine, and an emergent surgery was necessary.

A lumbar puncture was not carried out because there would be a risk in contaminating the previously sterile subarachnoid space.

Operation

An emergency L3-L4 wide laminoplasty was performed on the day of admission. After removing the ligamentous flavum, a substantial amount of frank pus expressed from the epidural space. The pus later grew *S aureus*. An adhesive inflammatory scar was carefully removed from the dura. The dura appeared markedly discolored and sclerotic. A small fissure, described as defect, was observed on the dura. No cerebrospinal fluid was encountered, and an intact arachnoid membrane appeared through the defect. A considerable amount of pus expressed from the subdural space when the surgeon gently palpated the dura to irrigate and clean out the pus. After copious irrigation, the defect was sutured closed. A drainage was placed in the epidural space. Then, the wound was closed in layers.

Postoperative Course

The patient was started on intravenous administration of cefotiam hydrochloride (1 g twice daily) for 14 days postoperatively then oral administration of trimethoprimsulfamethoxazole (two tablets, twice daily) for the subsequent 11 days, and his symptoms subsided. We confirmed that the CRP was negative 1 month after surgery. There has been no recurrence.

Meanwhile, at day 14 postoperatively, the MRI showed a cystic lesion, what is called as an empty thecal sac sign⁶⁾ at the L5-S1 level (Fig. 3). This was postulated as adhesive arachnoiditis⁷⁾ or residual subdural abscess. Laboratory data indicated that the patient was improving and he did not exhibit any symptoms. This intradural cystic lesion disappeared from the MRI taken 6 weeks postoperatively (Fig. 4).

Discussion

Prior to the present time, the incidence of SSA was reported as rare^{1,8-10)} and only just a hundred cases have been reported, which are much less common than spinal epidural abscess²⁾. Bartels et al. persuasively stated the reasons why there have not been many reports of SSA in comparison with cranial subdural abscess. They ascribed it to the absence of sinuses in the spine, the width of the epidural spinal space acting as a filter, and the centripetal direction of the spinal blood flow in contrast with the predominantly



Figure 2. Preoperative axial MR images demonstrating a fluid collection that exhibited low intensity in T1W (*left*) and high intensity in T2W (*right*). Epidural fat tissue coexisting with epidural abscess was observed in T1W image (*solid arrow*). Epidural fluid collection appeared contiguous with the subdural fluid collection through the dural defect (*dashed arrow*).



Figure 3. At day 14 postoperatively, the T2W MRI (*left* and *right*) showed an empty thecal sac sign at the L5-S1 level (*arrow*). This was postulated as adhesive arachnoiditis or residual subdural abscess.



Figure 4. The intradural cystic lesion disappeared from the MRI taken 6 weeks postoperatively.

centrifugally oriented bloodstream in the head⁸⁾.

The most common causative agent is *S aureus*^{1,4)}. Morbidity and mortality are very high in this pathology as well as spinal epidural abscess^{4,11)}. There are normally some predisposing factors such as diabetes mellitus, alcoholism, immunosuppressive drugs, malignant tumor, chronic renal failure, intravenous drug abuse, rheumatic heart valve disease, and tuberculosis⁵⁾. Also, secondary infection after spinal surgery or epidural injection may be potential causes for the development of SSA^{10,11)}. The mechanism of infection for SSA is considered as follows: (1) hematogenous spread from a distant site, (2) direct spread from an adjacent infection, (3) iatrogenic sources, and (4) unknown¹⁾. Despite of the previous reports, to the best of our knowledge, there has been no report concerning SSA after food intoxication in a healthy man. Our patient had no history of disease noted above, former spinal surgery, epidural injection, or any infectious lesion that could have spread hematogenously from a distant site. He experienced food intoxication after eating raw horse meat and *tori-wasa* with his colleagues. Then, several days after ingestion, he started to have severe LBP and a high fever over 39 °C. In the present case, the source of the infection was conjectured as *S aureus* infection subsequent to food intoxication. The result of his blood culture corresponded to that of the abscess obtained during surgery. We considered that he might have had a spinal epidural abscess first. Then, the anatomical barrier of the dura may have been disrupted by direct spread of the adjacent epidural abscess, resulting in the inflammatory infiltration of the abscess into the subdural space. In the present case, there was enough time for SSA development after the onset of food intoxication.

A lumbar puncture for cerebrospinal fluid test should not be planned because there would be a risk contaminating the previously sterile subarachnoid space¹²⁾.

MRI is considered the essential imaging modality of choice for the evaluation of SSA as well as epidural abscess¹¹). It is advocated that the epidural fat is typically preserved on axial and sagittal images in patients with SSA¹³. However, it is not always possible to distinguish a subdural abscess from an epidural abscess in a sagittal plane because both the epidural and subdural abscess of the spine could coexist. On the other hand, the location of spinal epidural or subdural abscess is well assessed in the axial plane of T2W images. As the dural layer of the meninges is thick and visible by T2W images, SSA can be identified with a small defect of the dura and the subdural fluid collection suggesting that the abscess appears contiguous with the epidural fluid collection through the defect (Fig. 2). In this instance, only a slight deviation of the dura toward the cauda equina is observed, whereas the deviation of the dura compressing the cauda equina is apparently recognized in the epidural abscess¹⁴⁾. Additionally, MRI with contrast may confirm the location of the abscess¹¹⁾. In our case, MRI with gadolinium contrast was not performed because MRI without contrast provided us with sufficient information that our patient had both the epidural and subdural abscess of the spine, and an emergent surgery was necessary.

At day 14, postoperatively, the MRI showed an empty thecal sac sign at L5-S1 level (Fig. 3). This cystic lesion might have been an arachnoiditis or a residual SSA. If it was the latter, our irrigation might not have been sufficient. However, laboratory data indicated that the patient was improving and he did not exhibit any symptoms. This cystic lesion spontaneously disappeared from the MRI at 6 weeks postoperatively (Fig. 4).

As advocated in the past, early diagnosis and the definitive treatments of the SSA, as well as the spinal epidural abscess, including surgical decompression of the neural structures, copious irrigation, and administration of the appropriate antibiotic agents are essential^{1,3,4,8,9,15}. A symptom of intolerable LBP with trunk motion restriction and high fever indicates the existence of infection in the spine. It was consoling to think that our patient had completely recovered despite of delayed diagnosis. Generally, delay of treatment normally deteriorates patients' neurological function and may lead to a lethal consequence⁴. Having the suspicion and an early recognition of not only the epidural abscess but also the SSA is important.

Conclusion

We presented here a very rare case of SSA following food intoxication caused by *S aureus* in a healthy man. SSA can be identified with a small dural defect and the intrathecal fluid collection compressing the cauda equina in the axial plane on T2W magnetic resonance images. On the grounds that morbidity and mortality are high in SSA, having suspicion of epidural abscess and likewise subdural abscess and making an early diagnosis using MRI and an emergent surgery are important when the clinician notices a febrile patient with severe LBP and trunk motion stiffness.

Conflicts of Interest: The authors declare that there are no relevant conflicts of interest.

Author Contributions: Tatsuto Takeuchi wrote and prepared the manuscript, and all of the authors participated in the study design. All authors have read, reviewed, and approved the article.

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