Acute Cholecystitis after Operative Treatment of Compression Fracture in the Thoracolumbar Junction Area: A Report of Two Cases

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The causes of postoperative acute cholecystitis are mainly bloodstream infections, stasis of bile juice due to immobilization, or highly viscid bile caused by dehydration. Postoperative acute cholecystitis is not a rare complication after gastrointestinal or orthopedic operations. We describe patients in whom the etiology of postoperative acute cholecystitis was unclear. The two patients underwent kyphoplasty and percutaneous pedicle screw fixation to treat compression fractures in the thoracolumbar (T-L) junction. After reviewing these two cases, it was difficult to confirm whether postoperative cholecystitis was due to immobilization, dehydration, or infection. We assumed that the cause of acute cholecystitis was damage of the sympathetic or parasympathetic chain around the T-L junction.

Key Words: Fractures; Compression; Cholecystitis; Acute; Autonomic nervous system

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INTRODUCTION

The incidence of mortality due to postoperative acute cholecystitis was reported as 10% to 50%⁴⁾. When acute cholecystitis occurs, it rapidly progresses and fulminates, so if it is not managed quickly, it worsens like peritonitis or sepsis and becomes fatal. It is widely known that acute cholecystitis usually occurs after hip surgery due to bloodstream infection, stasis of bile juice due to immobilization, and highly viscid bile caused by dehydration⁶⁾. Another rare factor is sympathetic stimulation that leads to spasm, ischemia, and mural damage and states of decreased perfusion, leading to hypoxia of the gallbladder¹⁴⁾.

Recently, the development of acute cholecystitis after kyphoplasty for T12 compression fracture was reported²⁾. The authors insisted that infection occurred immediately postoperatively because the position of the gallbladder (GB), which is located in the T12 level on X-ray and was adjacent to the surgery field. However, it is not clear how cholecystitis occurred in this case.

We review two patients who developed acute cholecystitis after undergoing kyphoplasty and percutaneous pedicle screw fixation (PSF) for compression fracture at the T12 and L1 levels, and suggest that the possible cause of acute cholecystitis was damage of the sympathetic or parasympathetic chain in these cases.

CASE REPORTS

Case 1

A 74-year-old woman visited our hospital with severe back pain after falling from bed. The X-ray and magnetic resonance imaging (MRI) scan revealed acute compression fracture of the T12 vertebral body with kyphotic change. She had a history of diabetes mellitus, hyperlipidemia, depressive mood disorder, and osteoporosis. She did not have any history of abdominal operation. The anterior body of the T12 was compressed more during weight bearing than during non-weight bearing. Computed tomography (CT) showed that the anterior border of the vertebral body was more fragmented than the posterior border.

The patient underwent percutaneous kyphoplasty using the extrapedicular approach under local anesthesia. The trocar was inserted in the far lateral aspect of the vertebral body. The vertebral height recovered from 41% to 67% (Figs. 1, 2). Her back pain was relieved postoperatively, and compression of the T12 vertebral body did not worsen with weight-bearing posture. Immediately postoperatively, she had no signs of infection, such as abdominal pain and fever, and no laboratory abnormality. She ambulated very well with a brace and followed a good diet. However, her blood pressure was high up to 170/100, and she had dyspepsia and diarrhea on postoperative 1 day.

Fig. 1. Lateral radiograph of the thoracolumbar spine reveals an anterior wedge compression deformity of the T12 vertebral body.

Fig. 2. Lateral radiograph of the thoracolumbar spine showing cement in a T12 vertebral body.

On postoperative day 8, she developed a high fever (39°C) and abdominal pain in the right upper quadrant area. The white blood cell (WBC) count, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) level were within normal range.

Fig. 3. Upper abdomen ultrasonogram shows the thickened gallbladder wall and pericholecystic fluid without a gallstone.

Fig. 4. Fluoroscopy shows the vertebroplastic state of a compressed T12 body with the catheter for percutaneous transhepatic gallbladder drainage.

The total bilirubin level increased from 0.68 mg/dL to 2.5 mg/dL (range 0.10-1.50 mg/dL), alkaline phosphatase (ALP) level increased from 179 IU/L to 532 IU/L (range 136-361 IU/L), gamma-glutamyl transpeptidase (γ -GTP) level increased from 58 IU/L to 119 IU/L (range 15-101 IU/L), aspartate aminotransferase (AST) level increased from 8 U/L to 35 U/L (range 0-40 U/L), alanine aminotransferase (ALT) level increased from 4 U/L to 38 U/L (range 0-40 U/L), amylase level increased to 435 U/L (range 28-100 U/L), and lipase level increased to 955 U/L (range 13-60 U/L). On the abdominal CT scan and ultrasonogram (US), the GB was markedly dilated and its wall thickness was comparable to that observed in acute cholecystitis. However, GB stones were not found (Fig. 3). Percutaneous transhepatic GB drainage

Fig. 5. Lateral radiograph of the thoracolumbar spine reveals an anterior wedge compression deformity of the L1 vertebral body.

Fig. 6. X-ray shows hardware fixation of the L1 fracture with a percutaneous pedicle screw placed in T11, T12, L1, and L2.

(PTGBD) was performed emergently (Fig. 4), and we prescribed antibiotics to prevent infection and medication to control her pain.

After the procedure, her abdominal pain was improved and fever subsided. In the culture study, bacteria were not identified in the bile juice. The PTGBD catheter was removed after 6 weeks, and she was discharged.

Fig. 7. Upper abdomen ultrasonogram shows the thickened gallbladder wall and pericholecystic fluid without a gallstone.

Case 2

A 62-year-old man experienced back pain after a fall. He was diagnosed as having a burst fracture of the L1 vertebral body with posterior interspinous ligament injury based on MRI findings. The cortical bone of the anterior and posterior vertebral body was fractured based on CT findings. The patient had a history of colon cancer and depressive mood disorder. He received chemotherapy after operative treatment for colon cancer and was determined to have fully recovered 2 years ago. He did not have any abdominal symptoms, such as pain, dietary problems, and gastrointestinal issues. He underwent PSF, which was performed under general anesthesia, at the T11-T12-L1-L2 level bilaterally. The trocar was inserted through the L1 pedicle bilaterally. During the operation, the trocars were inserted beside the L1 vertebral body because of the insufficient medial angle. So we removed the trocars and repositioned. The vertebral height recovered from 54% to 80% (Figs. 5, 6). He was prescribed absolute bed rest (ABR) for 7 days postoperatively, and then ambulation was permitted with a brace. Although his pain improved, he complained of dizziness, palpitation, and dyspepsia starting from postoperative day 2. We prescribed medications for these symptoms.

On postoperative day 12, he developed right upper quadrant area pain, nausea, and vomiting with a fever of 38.5°C. The abdomen was tender in the right upper quadrant and showed positive Murphy's sign. Additionally, the CT scan showed distension of the GB with mild wall thickening and pericholecystic fatty standing without any radio-opaque stones in the GB and biliary

Fig. 8. Fluoroscopy shows the catheter for percutaneous transhepatic gallbladder drainage.

tree, and the US showed distension of the GB with sludge but no GB stones (Fig. 7). The WBC count was increased at $15,200/\text{mm}^3$ (range $4,000\text{--}10,000/\text{mm}^3$), CRP level was increased at 8.21 mg/dL (range $0\text{--}0.8\text{ mg/dL}$), total bilirubin level increased from 0.52 mg/dL to 2.72 mg/dL (range $0.1\text{--}1.5\text{ mg/dL}$), AST level increased from 34 IU/L to 48 IU/L (range $0\text{--}40\text{ IU/L}$), and ALT increased from 40 IU/L to 55 IU/L (range $0\text{--}40\text{ IU/L}$). He was diagnosed as having acute cholecystitis so we performed PTGBD emergently (Fig. 8), and prescribed antibiotics to prevent infection and medication to control his pain.

In the culture study, bacteria were not identified in the bile juice. The patient showed improvement in his symptoms 2 weeks later, and the PTGBD catheter was removed within 8 weeks.

DISCUSSION

Compression fracture is a common disease in elderly patients^{10,13}. Waiting for self-fusion through bed rest, percutaneous vertebroplasty, and kyphoplasty are treatment options for compression fracture¹¹. Recently, percutaneous PSF has been used in cases of unstable vertebral fracture^{5,9,15}. Patients with unstable vertebral fracture who undergo surgery rarely develop postoperative acute cholecystitis. Only a few cases have been reported in the literatures^{1,19,14}, particularly with fracture in the T-L junction².

It is widely known that operative treatments of the gastrointestinal tract, such as total gastrectomy, subtotal gastrectomy, and esophagectomy, generally cause acute cholecystitis^{1,14}. Orthopedic surgery accounts for up to 16.5% of cases of postoperative acute cholecystitis. The mechanisms of postoperative acute cholecystitis are uncertain, but some such as bile stasis due to fasting, anesthesia, dehydration, fever, and use of narcotics for pain relief have been considered^{8,14}. Common causes of postoperative acute cholecystitis are bloodstream infections,

stasis of bile juice caused by immobilization, or highly viscous bile due to dehydration⁶. Patients with long-term immobilization due to compression fracture usually lose their appetite and become dehydrated. However, immobilization itself cannot explain the mechanism entirely because non-operative patients with immobilization do not develop acute cholecystitis. Bloodstream infections can better explain the mechanism because the type of postoperative cholecystitis is mainly acalculous cholecystitis. An et al.² suggested that postoperative acute cholecystitis occurred because of closure of the GB to the site of kyphoplasty. However, the definite causes are unknown, and the authors only suggested infection around the surgical site as the cause of postoperative acute cholecystitis. Likewise, acute cholecystitis developed postoperatively in the T-L junction area in our patients. However, we could not find any sign of infection around the surgical site. Therefore, we suggest that acute cholecystitis can develop because of sympathetic chain stimulation that leads to spasm, ischemia, and mural damage of the GB¹⁴.

After review of our two cases, it was difficult to confirm whether postoperative cholecystitis was due to immobilization, dehydration, or bloodstream infection. Our patients showed improvements in the visual analog scale score 8 to 3 postoperatively and had no signs of surgical site infection. Therefore they could ambulate and diet well. In case 1, the patient did not develop cholecystitis even though she was on bed rest for 2 months waiting for self-fusion. If stasis of bile juice due to hypomobility was key in the development of cholecystitis, this complication would have occurred within 8 weeks before kyphoplasty. Therefore, it is difficult to consider hypomobility as the key factor of cholecystitis because the patient developed cholecystitis after kyphoplasty. Moreover, infection cannot be the cause because there were no signs of infection at the surgical site or according to the postoperative laboratory findings, as the WBC count, ESR, and CRP level were within normal range. In case 2, the patient did not show any signs of surgical site infection and only complained of abdominal pain. Although he maintained ABR and underwent percutaneous PSF under general anesthesia 1 day later, he suddenly developed cholecystitis on postoperative day 2. These two cases showed no common causes of acute cholecystitis such as fasting, anesthesia, dehydration, fever, narcotic use, or bloodstream infection, as reported in previous studies.

In our cases, there were no common factors of postoperative acute cholecystitis, but our patients complained of symptoms of sympathetic stimulation. We suggest that the cause of cholecystitis was cholestasis due to damage or change of the sympathetic or parasympathetic chain¹². We tried to determine why a sympathetic chain problem developed in our patients. In the postoperative period, case 1 had dizziness, dyspepsia, and diarrhea and her blood pressure was high, and case 2 had dizziness, dyspepsia, and palpitation. The patients did not complain of these symptoms before the operation. Their post-operative

laboratory test results, including the AST from 8 U/L to 35 U/L in case 1, from 34 U/L to 48 U/L in case 2 (range 0-40 U/L), ALT from 4 U/L to 38 U/L in case 1, from 40 U/L to 55 U/L in case 2 (range 0-40 U/L), bilirubin from 0.68 mg/dL to 2.5 mg/dL in case 1, from 0.52 mg/dL to 2.72 mg/dL (range 0.10 mg/dL-0.15 mg/dL), ALP from 179 IU/L to 532 IU/L (range 136 IU-361 IU/L), and γ GTP levels, were slightly increased from 58 IU/L to 119 IU/L (range 15-101 IU/L). These laboratory findings were suggestive of cholestasis¹⁸⁾.

The sympathetic chain of the spine comprises paravertebral ganglia distributed from the upper cervical spine to coccyx, and it lies over the anterolateral surface of the vertebral body. Especially, the GB is innervated by two nervous systems. One system is the sympathetic nerve from the celiac plexus, and the other is the parasympathetic nerve from the vagus nerve. At the T8 and T9 levels, the preganglionic sympathetic chain receives afferent sensory information from the GB. The smooth muscles of the GB and bile duct are innervated by parasympathetic fibers, but they inhibit function of the sphincter muscles. Parasympathetic function of the GB causes contraction of the GB muscle and secretion of bile juice into the cystic duct⁷⁾. Sympathetic nerves originate in the vertebral column, beginning in the first thoracic segment of the spinal cord, extending upward until the second or third lumbar segment. The celiac plexus is a complex network of nerves located around L1. Sympathetic function of the GB causes relaxation and decreases GB secretion¹⁷⁾.

In both patients described herein, a trocar was inserted into the vertebral body in the T-L junction, and distraction was performed using a balloon device in kyphoplasty. Additionally, pedicle screw and rod compression was performed in PSF. We think that there is a possibility that the sympathetic or parasympathetic chain was injured when we were trying to find the proper point for trocar insertion. Case 1 underwent percutaneous kyphoplasty using the extrapedicular approach. In case 2, the trocar was inserted into the L1 vertebral body at bedside because of the insufficient medial angle. The location of trocar insertion could have caused damage to the sympathetic chain in both cases. We also think that the sympathetic problems that the patients complained of were due to direct injury of the sympathetic chain. However, the surgical procedures performed enabled reduction of the vertebral body. Saliou et al.¹⁶⁾ suggested that hyperalgesia in kyphoplasty is related to balloon inflation that can cause paravertebral soft-tissue distraction. We hypothesized that this reduction stimulated the celiac plexus and activated the sympathetic nerve fiber, which can disrupt bile juice secretion. As a result, bile juice accumulated and caused cholecystitis^{7,17)}. Further, this implies that cholecystitis was due to failure of bile juice secretion rather than obstruction because the CT scan and US did not show GB stones in both of our patients.

CONCLUSION

It is difficult to definitively diagnose postoperative cholecystitis due to sympathetic chain activation because some cases of postoperative cholecystitis develop in patients who undergo different types of spinal surgery performed at other sites. The exact cause of postoperative acute cholecystitis cannot be precisely confirmed; however, we assume that the sympathetic chain was injured by the kyphoplasty needle during massive reduction of the vertebral body height. We think it is possible to cause acute cholecystitis during kyphoplasty. Therefore, spinal surgeons must be careful when inserting the trocar into the T-L junction area and performing massive reduction of the vertebral body height. Especially, it is important to be aware of the possibility of acute cholecystitis when sympathetic symptoms develop after performing a surgical procedure in the T-L junction area.

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