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Original Article

Cauda equina syndrome: an uncommon symptom of aortic diseases

Fuliang He[1], Tong Xing[2], Fang Yu[3], Hongchuan Li[3], Xiutong Fang[3], Hongxing Song[3]

[1]Department of Interventional Therapy, Beijing Shijitan Hospital, Capital Medical University, Beijing, China; [2]Department of Vascular Surgery, Beijing Chaoyang Hospital, Capital Medical University, Beijing, China; [3]Department of Orthopedics, Beijing Shijitan Hospital, Capital Medical University, Beijing, China

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Abstract: Background: In order to help diagnose and deal with the fetal aortic diseases in time, we retrospectively reviewed 8 patients who presented with cauda equina syndrome (CES) but actually suffered from low spinal nerve ischemia due to aortic diseases. Material and Methods: 8 patients were initially diagnosed as CES. 7 patients were confirmed with aortic diseases. 1 patient was confirmed with aortic saddle embolism post emergent laminectomy. Relief of CES symptoms was evaluated during preoperation and follow-up period. Results: 1 patient was diagnosed as aortic dissection and 5 patients as AAA. These 6 patients underwent endovascular aortic repair (EVAR). The CES was relieved in 5-10 d post procedure. The 7th patient was diagnosed with acute abdominal aortic occlusion and then underwent catheter directed thrombolysis with recombinant tissue plasminogen activator (rTPA) for 20 h and CES disappeared. The JOA scores of the 7 patients were recovered from preoperative 15.14±1.21 to 21.00±2.16 within 5-10 d (P<0.01), and evaluated to be 24.12±1.34, 25.88±1.21 and 26.29±1.11 at 3 m-, 6 m- and 12 m-follow-up point, respectively. The 8th patient was initially diagnosed as lumbar spinal stenosis and lumbar disc herniation. The patient underwent emergent vertebral canal decompression and presented with serious CES symptoms. CTA confirmed that the patient had been suffered from aortic saddle embolism (ASE). Conclusion: CES caused by abdominal aortic diseases is a special event with fetal consequences if it is not recognized and treated promptly. Orthopedists and neurosurgeons should pay attentions particularly to this issue to preserve the cauda equina functions to their maximums.

Keywords: Cauda equina syndrome, aortic diseases, misdiagnosis

Introduction

Cauda equina syndrome (CES) is a common feature resulting from dysfunction of the sacral and lumbar nerve roots within the Cauda Equina. It could lead to bladder and bowel disturbances, impairment of sexual functions, perianal or saddle numbness and neurological symptoms in lower limbs [1, 2]. The symptoms of CES vary differently depending on the location of the injury in cauda equine. The most essential reason is the compression of spinal nerve roots. Nucleus of pulposus herniation at L3-S1 levels has been always observed clinically. Other causes of CES are as follows: aortic diseases; congenital diseases including spinal dysraphism, dwarfing syndromes and congenital tumors; Iatrogenic symptoms secondary to surgery or epidural anesthesia; spinal tumor; infective diseases and epidural/subdural hematoma [3].

Aortic diseases including abdominal aortic aneurysm (AAA), aortic dissection and even aortic saddle embolism (ASE) lead to a high mortality. Misdiagnosis of such kinds of diseases could be fetal. Aortic diseases mostly present with abdominal and back pain, lower limb ischemia, visceral ischemia and other symptoms. According to the literatures, the initial symptoms of aortic diseases that are similar to those of CES are rare [4, 5]. We retrospectively reviewed 8 patients who were initially diagnosed with CES in orthopedics department, but actually suffered from low spinal nerve ischemia due to aortic diseases. Prompt understanding of such disorders is of great importance and may help to improve the outcomes of the patients in future.

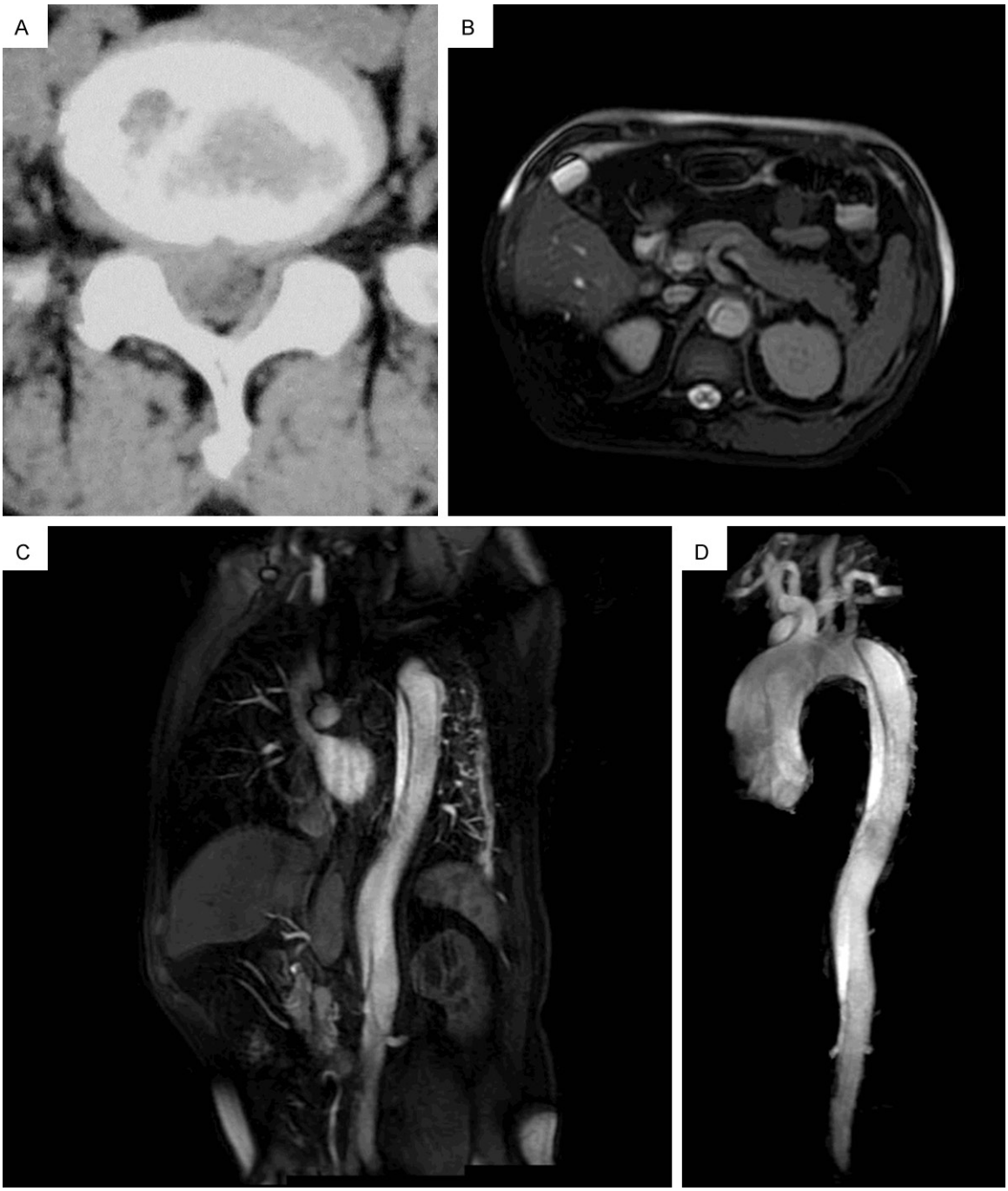

Figure 1. 40-year-old male patient diagnosed as aortic dissection. A: The transverse image of lumbar CT scan showed L4/5 lumbar disc herniation. B, C: The transverse and coronal images of aorta MRA revealed an aortic dissection affecting the feeding arteries of cauda equina. D: The 3-D reconstruction of aorta MRA showed a Type III aortic dissection.

Material and methods

8 patients (40-79 years old, mean age 66.13) were initially diagnosed as CES in orthopedics department. The patients presented with CES symptoms including urinary retention, incontinence, sensory disorders, pains or weakness in lower limbs. The course of the disease was lasted from 3 h to 6 m. Lumber MR or CT was performed to each patient. 5 patients were diagnosed with lumbar disc herniation, 1 with spondylolisthesis and 2 with lumbar stenosis.

The JOA (Japanese Orthopedic Association) scores were used to evaluate the CES degrees. An abdominal MRI, MRA or CTA were performed

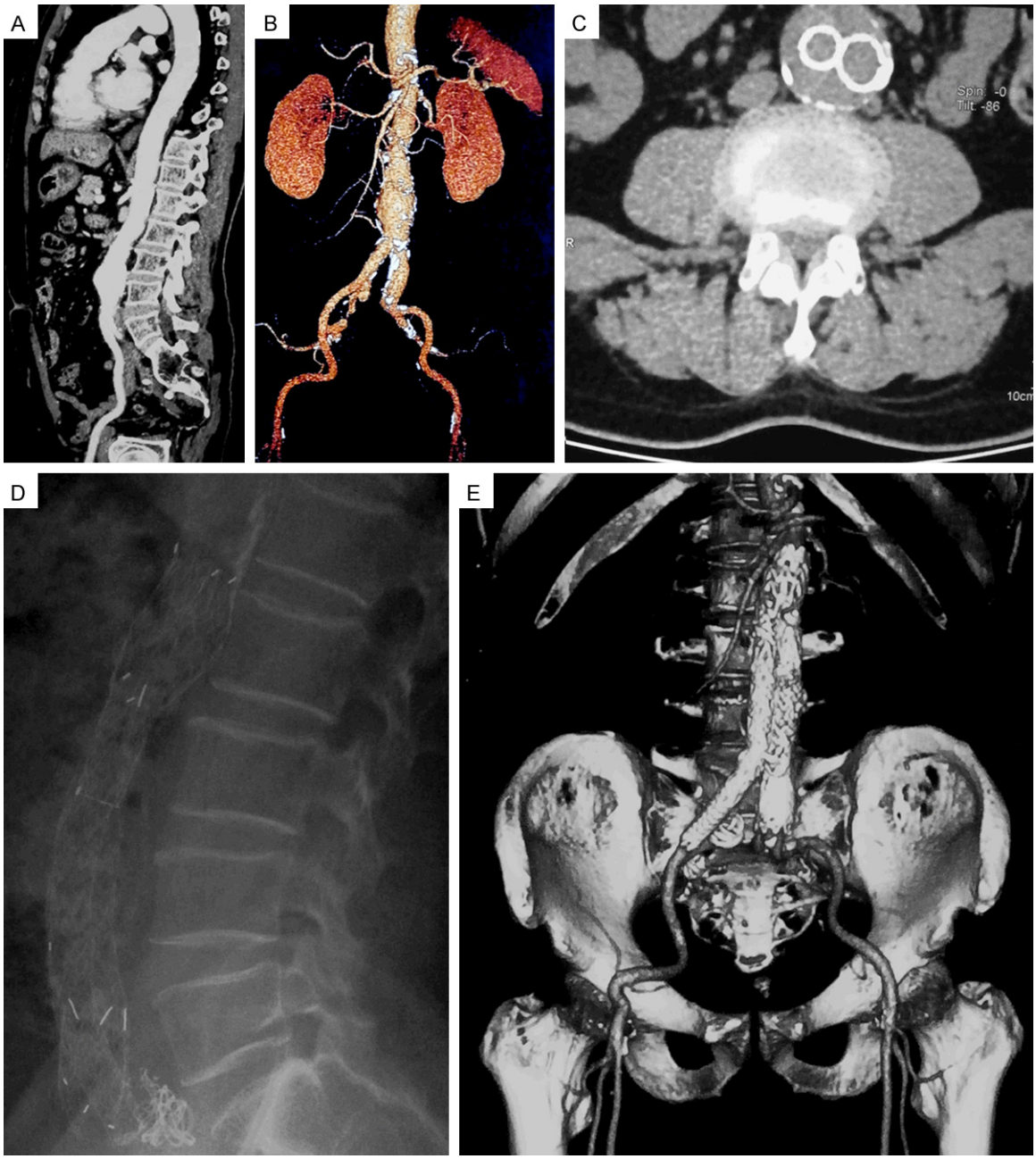

Figure 2. 74-year-old male patient diagnosed as AAA. A, B: The sagittal image and 3-D reconstruction of preoperative CTA showed the AAA in the cauda equina region. C: Transverse image of lumbar CT showed the iliac branch stents and the slight lumbar stenosis at 12 m-follow-up after EVAR. D, E: The Lateral X-ray and 3-D reconstruction at 12 m follow-up after EVAR.

on these patients. 7 patients presenting with CES were confirmed with aortic diseases, and then transferred to vascular centers for further treatments including endovascular aneurysm repair or catheter directed thrombolysis. 1 patient who was initially diagnosed as lumbar stenosis underwent emergent laminectomy. However, the CES was confirmed later to be caused by ASE. He underwent femoral artery embolectomy. The patients underwent a 12-21-m follow-up. The recovery of CES was evaluated before operation and at one-, three- and six-m post treatment.

Statistical analysis

SPSS 17.0 for windows was utilized for the data processing and statistical analysis. The Paired Sample t test was carried out for the measured

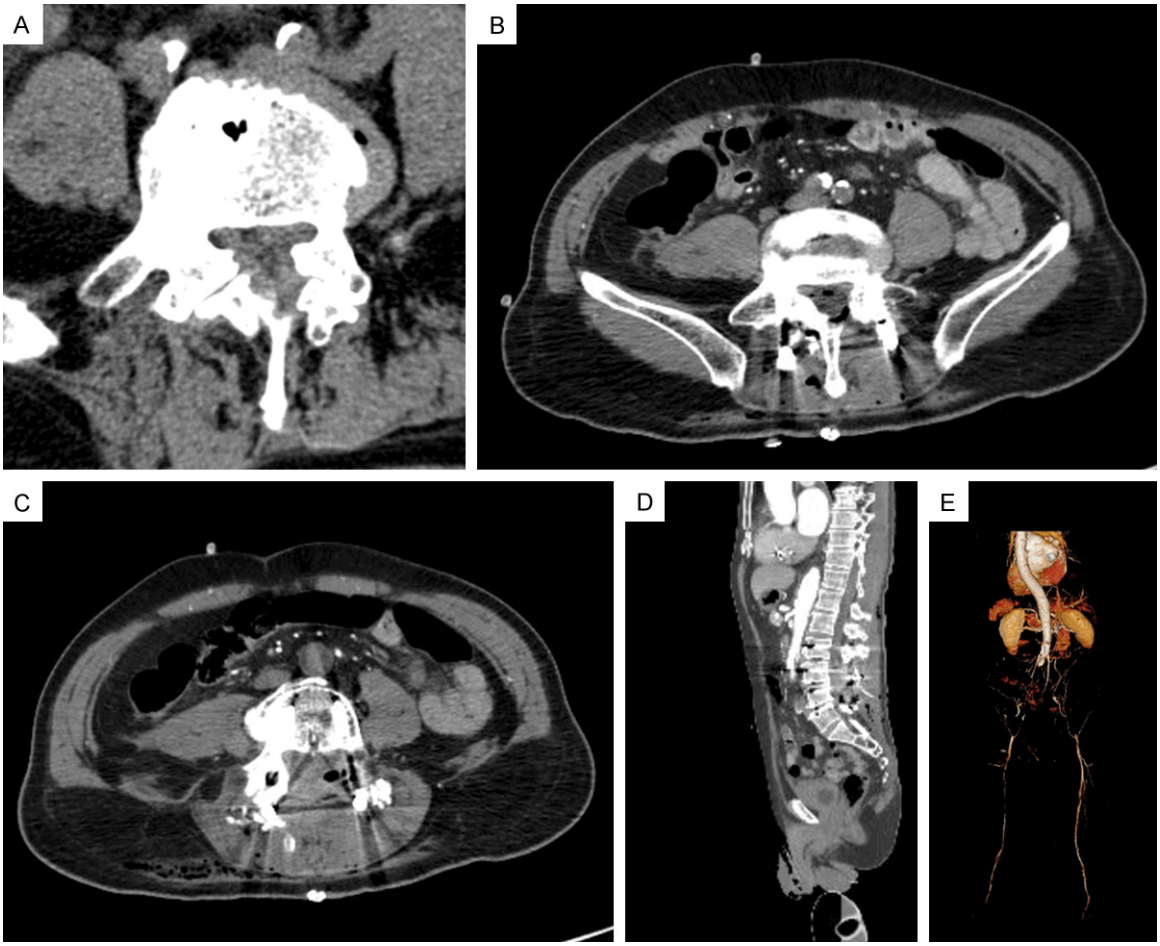

Figure 3. 79-year-old male patient diagnosed as ASE. A: Lumbar CT scan showed L4/5 severe spinal stenosis. B-D: The emergent CTA showed no blood supply in abdominal aorta and bilateral iliac arteries after laminectomy. E: The 3-D reconstruction of CTA showed the severe embolization of abdominal aorta and bilateral iliac arteries.

data. The count data were summarized as frequencies and continuous variables as $\bar{x}\pm S$. A *P*-value of <0.05 was considered to be statistically significant.

Results

Seven patients of them were transferred to vascular surgery department or interventional therapy department. 1 patient was diagnosed as aortic dissection (**Figure 1A-D**) and 5 patients as AAA (**Figure 2A**, **2B**). All of these 6 patients underwent endovascular aortic repair (EVAR) (**Figure 2C-E**). The time to operation from presentation was within 72 h. No intraoperative complications were observed. The CES was relieved in 5-10 d post procedure. The patients were prescribed lifelong anticoagulation in case of thrombotic events in future. The 7th patient was diagnosed with acute abdominal aortic occlusion. Catheter directed thrombolysis was performed on him with recombinant tissue plasminogen activator (rTPA) for 20 h. The abdominal aorta was resumed patency. Then the symptoms of CES were disappeared. A lifelong antiplatelet therapy was prescribed after the procedures. The JOA scores of the 7 patients were recovered from preoperative 15.20±4.05 15.14±1.21 to 21.00±2.16 within 5-10 d after the procedures (P<0.01). The mean value of the JOA score was 24.12±1.34, 25.88±1.21 and 26.29±1.11 at 3 m-, 6 m- and 12 m-follow-up point, respectively.

The 8th patient with an over-10-year history of atrial fibrillation (AF) was initially diagnosed as lumbar stenosis with a JOA score of 14. The patient underwent emergent vertebral canal decompression after 11 h of the symptom presentation. However, the symptoms were not relieved after the operation. He presented with even more symptoms including lower limb pain,

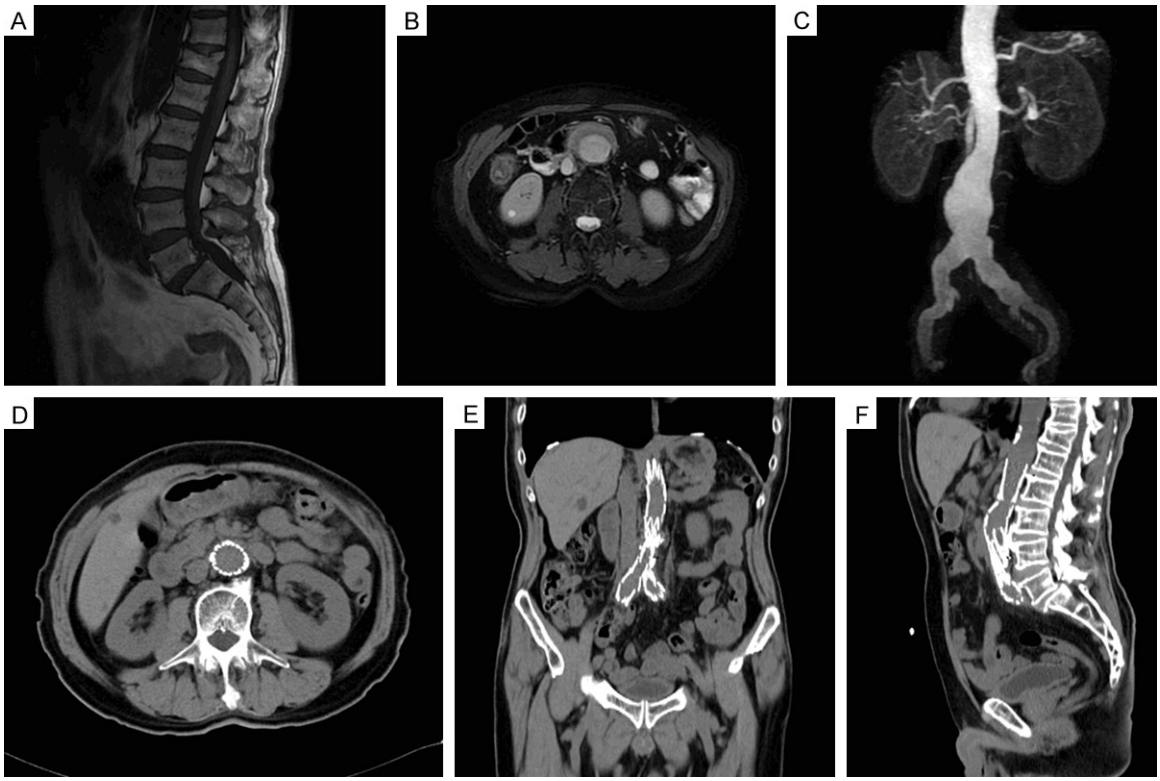

Figure 4. 72-year-old male patient diagnosed as AAA. A: The sagittal image of lumbar MRI showed lumbar spondylolisthesis of L5, meanwhile, a dilated abdominal aorta was observed. B, C: An MRA of abdominal aorta confirmed the AAA with thrombus in lumen affecting the cauda equina. D-F: The Abdominal CT showed the AAA was well handled at 12 m-follow-up after EVAR.

pallor and pulselessness that was the characteristics of acute lower limb ischemia. Ultrasound and CTA confirmed that the patient was suffered from the acute ASE (**Figure 3A-E**). Then he underwent the femoral artery embolectomy under local anesthesia 30 h after the initial symptoms appeared. The patient died 8 h after the surgery due to cardiac arrest. It was supposed that the patient's heart event was related to ischemic metabolites that entered the circulatory system after the blood supply of the lower limbs was resumed.

Typical case

A 78-year-old male patient presented with frequent micturition and sensory disorders of lower limbs for 10 d. He had a 2-year history of abdominal pain and an over 20-year history of hypertension. The clinical examination revealed an abdominal pulsatile mass. No numbness or motor deficits of bilateral lower limbs was found. The bilateral dorsal pedal arteries were palpable. The JOA score was 17. The MRI showed slight hernia of L3/L4 and L5/S1 intervertebral discs. Meanwhile, a dilated abdominal aorta was observed (**Figure 4A**). MRA and CTA were performed. The abdominal aneurysm and bilateral iliac aneurysms were detected (**Figure 4B**, **4C**). It was concluded that the patient's symptoms were caused by cauda equina ischemia due to feeding artery embolism. The patient was transferred to vascular center and underwent EVAR 40 h later. The cauda equina syndrome was relieved in 4 d post operation with a JOA score of 23. The patient is still under the following up. The neurological symptoms have not been recurred within a 1-year period with a JOA score of 25 (**Figure 4D-F**).

Discussion

Causes of cauda equina syndrome

CES is usually resulted from compression of the sacral and lumbar nerve roots. Recently, it has been reported that CES could be caused by spinal cord and nerve root ischemia due to aortic diseases. EL-Osta et al [5] investigated an unusual type of spinal cord infarction whose

symptoms mimicking CES were caused by embolus dropping from an AAA. After the conservative treatment none of the patients were fully recovered. One patient was deteriorated and died immediately. Sydney Wong et al [6] reported a patient who initially presented with CES experienced acute aorta occlusion that was actually caused by thrombosis of an AAA. The patient underwent bifemoral bypass, and was recovered fully 7 h after the surgery. These studies indicated that CES could be caused by embolism of the feeding artery. The "ischemic CES" may present the initial symptoms of aorta diseases. In the present work 6 patients out of 8 underwent EVAR after the diagnosis. They were recovered fully within 5-10 d. However, 1 patient died eventually without the timely diagnosis and treatment. This implies that the patients with ischemic CES should undergo the treatment as soon as possible. Any delayed patency of the feeding arteries may lead to gross disability for such patients. The Ischemic CES caused by aortic diseases is rare but clinically important. It requires particular attentions.

Blood supply of cauda equina

The artery of Adamkiewicz, also known as the great anterior radiculomedullary artery (GRA), is formed from the inferior intercostal or upper lumbar arteries, and grows between the T5 and L3 vertebra [7]. It contributes significantly to anastomoses with the iliolumbar artery, the median and the lateral sacral arteries [8]. As distal cord is supplied by these arteries, embolization of these vessels could cause ischemic CES. All of the patients in the present work were suffered from abdominal aortic diseases that would affect the arteries. Thoracic aorta diseases presenting as CES haven't been observed.

As believed in the past time, the thoracic cord at T4-T8 level is very sensitive to hypoperfusion [9]. However, recently this point of view has been proved to be controversial. An autopsy study of 66 patients suffered from ischemic myelopathy secondary to heart arrest or severe systemic hypotension showed that the thoracic cord infarction occurred only in 5 patients (7.6%), while, lumbosacral cord infarction presented in 63 patients (95.5%) [10]. Therefore, lumbosacral cord including cauda equina might be even more sensitive to the reduced blood flow.

AAA, ASE and CES

AAA means over 50% increase in the diameter of aorta. It affects eight percent of people over the age of 65 [11]. AAA is often called a "silent killer" because it usually has not any obvious symptoms. The symptoms of AAA include: constant or discontinuous abdominal pain, lower back pain and pulsing abdominal mass. A collection of mural thrombosis is often observed in the walls of AAA. Dropping clots would result in distal organs ischemia that is usually appeared in one single lower limb [12]. CES is rarely reported to be caused by dropping off thrombus in AAA. In the present work 3 patients with AAA presented with CES. The symptoms of 2 patients were localized in bilateral lower limbs without a sign of limb ischemia. The reason might be that the dropping clots could reduce the whole blood flow of the cauda equina feeding anastomoses instead of one single artery. The most popular treatment of AAA is EVAR [13] with a covered stent landing over the dilated abdominal aortic artery wall. All patients with AAA in the present work underwent this procedure. Relief of the symptoms might be due to the disappearance of clots dropping from the aneurysm wall.

ASE is a rare but lethal aortic disease. The embolus terminates in order to "straddle" the aortic bifurcation, causing bilateral lower extremity arterial ischemia with a high mortality rate [14]. Despite of its rareness ASE has been reported to have spinal cord infarction symptoms at its initial presence. Andrew S Olearchyk [15] reported a woman at the age of 85 presented with sudden paraplegia. She was confirmed with ASE and underwent femoral artery embolectomy, and then was fully ambulatory within a week after the surgery. The ASE progresses very fast within several hours. In contrast, the CES caused by lumbar disc herniation presents as a repeated and chronic disease [16]. If the CES progresses too fast, more and especial attentions should be paid to the aortic diseases including ASE.

Patients with emergent CES have been mainly admitted to orthopedics department with an initial diagnosis of degenerative diseases such as lumber disc hernia or lumbar spinal stenosis. During dealing with these patients, orthopedists should pay more attentions to ischemic CES, especially when the patients have symptoms like abdominal discomfort etc. We would

like to provide the following advices: 1. When the symptoms of CES progresses too fast, the CES caused by vascular diseases should be taken into consideration; 2. Abdominal examinations including checking pulsatile mass abdominal, a typical mark of abdominal aortic aneurysm and auscultation of vascular bruit, should be taken; 3. Ischemic CES should not be excluded if dorsal pedal arteries are palpable; 4. Abdominal aorta diseases should not be excluded when the results of lumber CT/MRI cannot match the symptoms of CES. 5. Once the patient is diagnosed as ischemic CES, the treatments of aortic diseases should be performed as soon as possible to restore the functions innervated by cauda equina. 6. During dealing with patients with CES, vascular surgeons should be invited for the diagnosis when abdominal aorta diseases are suspected by orthopedists and neurosurgeons.

Conclusion

CES can be caused by abdominal aortic diseases including AAA, abdominal aortic dissection and ASE that reduce the blood supply of the spinal cord and nerve root. Ischemic CES is a special event with a fatal consequence if it is not recognized and treated promptly. Orthopedists and neurosurgeons should pay especial attentions to this issue and restore the blood supply as soon as possible to preserve the cauda equina functions to their maximums.

Disclosure of conflict of interest

None.

Address correspondence to: Dr. Hongxing Song, Department of Orthopedics, Beijing Shijitan Hospital, Capital Medical University, 10 Tieyi Road, Haidian District, Beijing 100038, China. Tel: +86-10-63926159; E-mail: shx2@medmail.com.cn

References

[1] Todd NV. Cauda equina syndrome: findings on perineal examination. Br J Neurosurg 2013; 27: 852.

[2] Abuzinadah A, Almalik Y, Shabani-Rad MT, Ho CH, George D, Alant J and Zochodne D. Cauda equina syndrome secondary to intravascular lymphoma. Neurol Clin Pract 2012; 2: 158-161.

[3] Kavanagh M and Walker J. Assessing and managing patients with cauda equina syndrome. Br J Nurs 2013; 22: 134-137.

[4] Qadura M, Pervaiz F, Harlock JA, Al-Azzoni A, Farrokhyar F, Kahnamoui K, Szalay DA and Rapanos T. Mortality and reintervention following elective abdominal aortic aneurysm repair. J Vasc Surg 2013; 57: 1676-1683.

[5] El-Osta B, Ghoz A, Singh VK, Saed E and Abdunabi M. Spontaneous spinal cord infarction secondary to embolism from an aortic aneurysm mimicking as cauda equina due to disc prolapse: a case report. Cases J 2009; 2: 7460.

[6] Wong SS, Roche-Nagle G and Oreopoulos G. Acute thrombosis of an abdominal aortic aneurysm presenting as cauda equina syndrome. J Vasc Surg 2013; 57: 218-220.

[7] Dandu VH, Bradley L, Hoffman P and Hinduja A. Spinal cord infarction associated with use of phentermine. Acta Neurol Belg 2014; 114: 157-158.

[8] New PW and McFarlane CL. Survival following spinal cord infarction. Spinal Cord 2013; 51: 453-456.

[9] Rossi D, Goodwin D and Cruzzavala J. Spinal cord infarction following coronary artery bypass grafting surgery. W V Med J 2008; 104: 24-25.

[10] Duggal N and Lach B. Selective vulnerability of the lumbosacral spinal cord after cardiac arrest and hypotension. Stroke 2002; 33: 116-121.

[11] Robinson D, Mees B, Verhagen H and Chuen J. Aortic aneurysms-screening, surveillance and referral. Aust Fam Physician 2013; 42: 364-369.

[12] Stather PW, Sidloff D, Dattani N, Choke E, Bown MJ and Sayers RD. Systematic review and meta-analysis of the early and late outcomes of open and endovascular repair of abdominal aortic aneurysm. Br J Surg 2013; 100: 863-872.

[13] England A and Mc Williams R. Endovascular aortic aneurysm repair (EVAR). Ulster Med J 2013; 82: 3-10.

[14] Mohan UR, Mangat JS, Sutaria N and Franklin RC. Saddle arterial embolus in a patient with Down syndrome. Pediatr Cardiol 2006; 27: 117-121.

[15] Olearchyk AS. Saddle embolism of the aorta with sudden paraplegia. Can J Surg 2004; 47: 472-473.

[16] Fairbank J and Mallen C. Cauda equina syndrome: implications for primary care. Br J Gen Pract 2014; 64: 67-68.