condition presented with mildly painful muscle stiffness and cramps. Percussion of the muscle belly produced a characteristic mounding of the muscle. Involuntary rolling contractions of the muscle known as ‘rippling’ could also be seen. The propagation was estimated to be 0.6 m/s, approximately 10 times slower than the propagation velocity of the muscle fibre action potential. The phenomenon is thought to be due to local contraction of a portion of the muscle fibre causing stretching of the neighbouring sarcomere and thus a mechanical stimulus to contract. Percussion induced rapid muscle contraction (PIRC) may also occur. The local mounding, PIRC and rippling phenomena are all electrically silent in EMG, differentiating this condition from myotonias.

It was not until 2001 that the first caveolin-3 mutation was found to be the cause of autosomal dominant RMD in five previously described families. Of the 10 mutations now identified in the caveolin-3 gene, seven have been associated with the RMD phenotype. Most of the mutations identified are also associated with other phenotypes. Indeed the Arg26Gln mutation seen in our patient has been reported to be found in all of the four different phenotypic groups. Our case similarly highlights this phenotypic variability with the overlapping features of local mounding and PIRC as well as distal wasting.

Rippling muscle disease is a rare, relatively mild, non-progressive condition. For these reasons, few therapeutic trials have been published. Only case reports report the benefit of dantrolene and calcium channel antagonists in two patients.

References


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Acute paraplegia following chiropractic therapy

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Abstract

We report a 44-year-old man suffering complete paraplegia due to paraspinal and epidural abscess, following chiropractic therapy for severe back pain and whose diagnosis was delayed. He received an immediate laminectomy from T3 through T6 to decompress the full extent of the abscess and appropriate antibiotic therapy for 4 weeks postoperatively for the identified microorganism (Staphylococcus
1. Introduction

Combined paraspinal and spinal epidural abscess (SEA) is a rare but potentially devastating condition. Early diagnosis and management are mandatory before deterioration of neurologic function occurs. Herein, we describe an uncommon case of acute paraplegia following chiropractic therapy for severe back pain due to paraspinal and epidural abscess, and we also review the literature.

2. Case report

A 44-year-old man who was previously in good health had complained of right subscapular pain over the previous 2 weeks. For this pain, he had received several chiropractic treatments. Within the previous week, he had come to our emergency room four times due to persistent intractable pain in the scapular area. There was no local heat, skin change or swelling in the scapular area. There was no neurological deficit, and the laboratory data revealed only mild leucocytosis with a shift to the left (white blood cell count, 10,400/mm³ with 73.3% neutrophils and 0% band forms) with an elevated erythrocyte sedimentation rate (ESR) of 32 mm/h (hospital normal range, 0–15). The plain thoracic-lumbar spine X-ray was normal. Conservative treatment was recommended at that time. Unfortunately, he presented to the emergency room with complete acute paraplegia and abdominal distension 3 h after a chiropractic session. Physical examination was notable for total sensory loss below T5, the complete absence of power and reflexes of the lower extremities, a dilated bladder, and priapism. Magnetic resonance imaging (MRI) revealed an area of longitudinally fusiform, mixed high and intermediate signal intensity on both T1-weighted (left) and T2-weighted (right) images from T3 to T6 of the posterior epidural space. The underlying cord was markedly compressed.

Fig. 1. Magnetic resonance imaging revealed an area of longitudinally fusiform (between arrows) mixed high and intermediate intensity on T1-weighted (left) and T2-weighted (right) images from T3 to T6 of the posterior epidural space. The underlying cord was markedly compressed.
T1- and T2-weighted images and the underlying cord was markedly compressed. There was also abnormal intermediate T1-weighted and high T2-weighted intensity in the T3–T6 right paraspinal muscles (Figs. 1–3). Under the suspicion of acute paraspinal and epidural haematoma, he immediately underwent an extended laminectomy from T3 through T6. Thickened dura with a diffuse suppurative coating and multiple yellowish abscesses mixed with fat in the epidural space from level T3 through T6 were found. The interspinous ligament between T4 and T5 was torn, which communicated at the T5 level with the right paraspinal muscles. Extensive debridement and abscess aspiration were performed. Cultures of pus obtained at surgery were positive for Staphylococcus aureus. The patient received a 4-week course of postoperative antibiotics (oxacillin). After a 3-month rehabilitation program, he had recovered bladder function with moderate residual left lower extremity paresis.

3. Discussion

Combined paraspinal muscle and SEA is an uncommon entity, but its clinical importance overshadows its rarity. Risk factors for abscess formation include immunocompromised states such as diabetes mellitus, alcoholism, cancer, intravenous drug abuse, chronic renal failure, liver disease and acquired immunodeficiency syndrome, as well as spinal procedures including epidural anaesthesia and spinal trauma or surgery. However, the condition has been reported in patients with no predisposing risk factors. In our patient, the cause of the abscess was unclear. But repeated blunt trauma due to chiropractic treatment was possibly responsible. Forceful massage with a hard tool, such as a wooden bar, to the back may cause damage to skin and soft tissue which will result in the formation of a haematoma; this serves as a rich nutrient source for infection. This subsequently induces bacterial invasion into the circulatory system, the development of septicaemia and ultimately abscess formation. In this case, we do not want to over-emphasise the relationship between forceful massage and abscess formation in the back. However, it may play a part in the aetiology of abscess formation.

Spinal epidural abscess usually results from haematogenous dissemination from distant foci including the skin, and respiratory and urinary tracts. Direct spread of microorganisms from adjacent structures is less frequent and usually follows a spinal procedure or epidural anaesthesia. In our patient, the definitive infection focus was not identified. The most probable initial infection source was the paraspinal soft tissue although the cause is still to be determined. Either direct spread of bacteria or haematogenous dissemination from the paraspinal muscle to the epidural space may explain development of the epidural abscess.

Staphylococcus aureus is the most common causative pathogen. The high incidence may result from the tendency of S. aureus to produce abscesses, its ubiquitous nature, and its ability to infect healthy as well as immunocompromised hosts. As in our case, the commonest site of infection is the thoracic region posterior to the spinal cord. The reasons for this are largely anatomical: the epidural space is wider at the thoracolumbar level and there is also a more extensive extradural venous plexus. In addition, the ventral aspect of the dura is tightly adherent to the posterior longitudinal ligament.
Presentations of SEA are nonspecific and can range from back pain to sepsis. Local tenderness with or without neurologic deficit is the most consistent symptom and occurs in virtually all patients during their illness. Fever is not mandatory for a diagnosis of SEA. In our case, the patient had come to our emergency room four times due to intractable back pain before complete paraplegia developed. There were no obvious toxic or neurologic signs identified. This makes diagnosis difficult; furthermore, because of its rarity, many physicians will not encounter such a case during their careers. At the time of presentation, the only abnormal laboratory findings were mild leucocytosis (WBC, 10 400/mm$^3$) and an elevated ESR (32 mm/h). These findings imply that a patient with intractable localised back pain and raised inflammatory markers may need an urgent MRI to rule out the possibility of abscess formation. Only increased awareness and rapid management of SEA will improve the outcome.

Magnetic resonance imaging is the radiological investigation of choice for SAE. The epidural mass may be hypointense, isointense, or slightly hyperintense on T1-weighted imaging and is nonhomogenous hyperintense on T2-weighted imaging. Low signal intensity represents a collection of liquid pus. In our case, the patient’s spinal MRI showed mixed high and intermediate signals on T1 and T2-weighted images in the epidural and paraspinal areas. Acute haematoma was misdiagnosed preoperatively. This misdiagnosis may be explained by the effect of a mixture of epidural fat, granulation tissue and frank pus. This finding is consistent with a previous report that even with MRI, a preoperative diagnosis of infection is difficult. In this situation, the diagnostic study of choice is MRI with gadolinium contrast.

The treatment of choice for SEA is a laminectomy of adequate length to decompress the full extent of the abscess, copious irrigation, and immediate antibiotic therapy for 4-6 weeks according to the identified microorganisms. The preoperative neurological condition and the degree of spinal canal compression are the major determinants for the outcome of SEA. Most patients with incomplete paresis or minor neurological impairment improve following surgery. A lesser degree of improvement is seen with increasing severity of clinical presentation. The preoperative neurologic condition of our patient was bowel and bladder dysfunction and complete paraplegia. He underwent an extended laminectomy from T3 through to T6 and received antibiotic (oxacillin) therapy postoperatively for 4 weeks. After a rehabilitation program of 3 months, he had recovered bladder function, but developed moderate residual left lower extremity paresis. This outcome may imply that mechanical compression alone is not the sole factor causing neurological deterioration. The explanation for the irreversible neurological deficits probably involves a vascular mechanism such as venous compression, thrombosis, and/or infarction due to focal bacterial embolism of the neural tissue. As our patient had an unfavourable out come, we want to emphasise the importance of early diagnosis and management before deterioration and neurologic deficit occurs.

We conclude that patients with localised back pain and raised inflammatory markers require urgent spinal gadolinium-enhanced MRI to rule out the possibility of SEA. We also want to emphasise the risk of acute paraplegia after forceful massage to the back for intractable back pain, particularly in the light of expanding use of chiropractic therapy for musculoskeletal disorders in Asian populations.

References