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A surprising cause of paresis following scoliosis correction

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Abstract Paralysis following scoliosis correction is a catastrophic situation. We report an unusual metabolic cause of neurological deficit after anterior thoracic release. A 15-year-old female developed proximal leg paralysis 1 day after surgery. Investigations disclosed severe serum hypokalaemia (2.8 mmol/l). After intravenous potassium substitution the neurological status completely normalized within a few hours. We assume that the condition was a mani-

festation of hypokalaemic paralysis since no further abnormalities could be disclosed. Spinal surgeons should bear in mind hypokalaemia as a benign and easily correctable cause of paresis following surgical scoliosis correction.

Keywords Hypokalaemia · Scoliosis · Paresis · Spine · Deformity

Introduction

The most feared complication in spinal surgery is paraplegia. In a large reported series the risk of a cord lesion in spinal deformity surgery has been found to be 0.5–1.7%. Risk signs noted are congenital deformity, kyphosis, postradiation deformity and larger (more than 100°) curves. The lesions are caused by cord stretching in instrumented patients or direct trauma [12].

We report an unusual metabolic complication which caused transient paresis of the proximal leg muscles after scoliosis surgery.

Case report

A 15-year-old Turkish girl (B.U., 162 cm, 42 kg) was admitted for surgical correction of severe thoracic scoliosis. The patient presented with a marfanoid habitus and a thoracic curve of 116° (Cobb). Preoperative neurological examination was unremarkable and the MRI of the spinal cord revealed no cord abnormalities. Halogravity traction and anterior release, followed by further traction and finally posterior instrumentation were scheduled. After a 5-week period of traction, anterior release Th4–L1 was performed uneventfully in controlled hypotension. The right segmental arteries Th5–Th12 were ligated. Autologous iliac bone and rib grafts

were placed in the disc spaces on the concave side of the curve. The cell saver technique was used (400 ml autotransfusion), central venous pressure was monitored (6–11 mm H₂O) and fluid intake was adjusted accordingly. The Stagnara wake-up test and postoperative function were normal, with forceful dorsi- and plantarflexion bilaterally.

During the following day the patient developed symmetrical paresis of the hip and knee flexors and extensors (0/5). The dorsi- and plantarflexion of the feet were still forceful (4/5) and sensibility was not disturbed in either leg. Patellar tendon jerks and Achilles tendon reflexes were absent. There were no evident pyramidal signs. Rectal tone was normal, as was examination of the upper extremities. The neurologist additionally disclosed a decreased T5–10 sensibility on the right hemithorax and suspected a traumatic cord lesion. A CT scan of the thoracic spine was performed, and intraspinal haematoma and dislocation of intersomatic rib grafts were ruled out. A psychological exploration of the patient revealed no suspicion of simulation. At that time a routine blood check showed decreased potassium (2.8 mmol/l) and haemoglobin (8.4 mg/%) levels. Other parameters including serum pH (7.36) and sodium (138 mmol/l) were within normal limits. There were no electrocardiographic changes present.

Intravenous potassium substitution (20 mEq/h) was started under continuous ECG monitoring. Red blood cells (2 units, 350 ml) were transfused to correct anaemia. A 24-h urine measurement and electromyography were initiated to determine the aetiology of the hypokalaemia, but the paresis resolved before the evaluations were finished. Secondary hypokalaemia caused by a transcellular shift due to metabolic acidosis was ruled out by arterial blood gases (serum pH 7.36). Since the patient was normothermic, increased

Fig. 1a, b Anteroposterior profile (a) and sagittal profile (b) radiographs at presentation, after anterior release Th4–L1 and after posterior instrumentation

potassium loss through the sweat was also ruled out. Paresis of the hip and knee muscles disappeared completely within the following 6 h. The potassium level had been corrected to 3.6 mmol/l and haemoglobin had been increased to 10.2 mg%. The cause of the resolved paraparesis was still unclear.

During the next day bowel stimulation was performed using Neostigmin (3 mg/24 h) and oral laxatives. The patient developed proximal leg muscle weakness again and the potassium level had dropped to 3.2 mmol/l. Careful potassium substitution was continued and symptoms disappeared again within a few hours.

The following course of treatment was unremarkable and included continued halo traction for three additional weeks, posterior correction and instrumentation, and remobilization of the patient (Fig. 1a, b). The serum potassium level was closely monitored and was maintained above 4.0 mmol/l by oral supplementation.

In our retrospective analysis we could not detect any unusual predisposing factors nor former episodes of transient paresis in the

patient or family history. So we interpret this dramatic course as a recurrent manifestation of hypokalaemic paresis unrelated to the surgery. Recently the patient presented in the outpatient clinic for the 1-year follow-up. Neurological examination was unremarkable. No further episodes of sudden proximal leg paralysis had occurred.

Discussion

Neurological deficits following surgical scoliosis correction can be catastrophic. Multiple risk factors related to the aetiology and pattern of deformity as well as direct trauma by the surgical procedure are known causes [12]. An unusual case of severe paresis following scoliosis surgery unrelated to the deformity and technique performed is reported. The metabolic disorder described raised diagnostic confusion and, to our knowledge, has not been reported previously as a complication of scoliosis surgery. As in our case, a neurological deficit was found following an uneventful scoliosis operation and misinterpreted as a sign of a cord lesion. Careful postoperative observation is recommended especially with instrumentation using the distraction principle for deformity correction [6].

Interpretation of our patient's neurological symptoms was difficult. Preserved normal sensitivity and forceful foot dorsi- and plantarflexion were present, in contrast to symmetrical plegia of the hip and knee flexors and extensors. Distraction or direct damage to the spinal canal had not occurred during the procedure. To rule out an intraspinal haematoma or dislocation of an intervertebral graft in the canal a CT of the operation site was performed and showed no pathology. MRI could have disclosed an intraspinal pathology such as medullary infarction but was not practicable immediately because of continued halo traction.

As the low potassium level was the only pathological finding, substitution was performed immediately and symptoms disappeared rapidly. The diagnosis was therefore established empirically. The sudden drop in the serum potassium level was surprising since the preceding value 6 h before was normal (3.8 mmol/l). The intraoperative blood loss (850 ml) was not alarming, nor was the postoperative fluid loss through the chest drainage (340 ml/24 h). There was no gastrointestinal potassium loss since no nasogastric tube was placed and the patient had not vomited or suffered diarrhea before the onset of paresis. Nevertheless, extrarenal potassium loss is the most likely cause of hypokalaemia. In our patient additional potassium shift into the muscle may have caused the neurological impairment.

Only sporadic cases of acute-onset hypokalaemic paraparesis have been described in the literature, and none following surgery [4]. Attacks have been induced by injection of insulin, epinephrine, fluorohydrocortisone and glucose, and by ingestion of a meal high in carbohydrates. Epidemic onset has been recognized following severe

gastroenteritis in Indian and Australian aboriginal communities [2, 11]. The extent of hypokalaemic paralysis varies from slight weakness of the legs to complete paralysis of the trunk and limbs. The respiratory muscles are usually spared even in severe attacks. Tendon reflexes and electrical reactions of the muscles are diminished in proportion to the degree of weakness. Cutaneous sensation is not disturbed. The duration varies from a few hours to 24 or 48 h. Usually, during asymptomatic intervals the patients are strong and the potassium content of the serum is normal [8].

Secondary hypokalaemic paralysis has been recognized in patients suffering from diabetes, renal tubular acidosis, thyrotoxicosis and self-induced water intoxication [1, 3, 5, 7, 10]. Our patient proved to have normal kidney and thyroid function as well as unremarkable blood sugar serum levels. Rudolf et al. reported a case of

hypokalaemic paralysis in a patient with chronic laxative abuse [9]. The potassium level was 1.7 mmol/l. In our patient the potassium depletion was not as severe, but the drop was acute. This may explain why the symptoms became apparent much more quickly in our case.

An increase in haemoglobin levels may also have contributed to improvement of the paresis since intramedullary pathology could not be ruled out by MRI. The RBC were transfused in parallel with the serum potassium correction and a final differentiation of both measures remains impossible.

In conclusion, spine surgeons should bear in mind hypokalaemia as a benign and easily correctable cause of paresis following surgical deformity correction. Therefore, serum potassium levels should be closely monitored and supplements should be given before symptoms of hypokalaemia become clinically apparent.

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