Painful muscle spasm reversed by magnesium sulphate

A case report

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Summary

A 25-year-old paraplegic man who had sustained a T3/T4 vertebral compression fracture 3 years previously presented with severe, painful spasm of the left hamstring muscle group of 2 hours' duration. This spasm produced extreme knee flexion of a degree which held the left foot posterior to the right buttock. An intravenous injection of 2 g magnesium sulphate produced immediate relief. Possible mechanisms of action of magnesium are discussed.

Case report

A 25-year-old man who had been paraplegic for 3 years after a traumatic T3/T4 vertebral compression fracture presented at Johannesburg Hospital with a history of several attacks of muscular spasm of the lower limbs. To prevent flexion contracture of the knees and hips, plaster of Paris casts had been applied to maintain the joints in a neutral position. According to the patient, previous attacks of spasm had been treated with spinal or epidural blockade.

On this occasion the patient had developed a painful muscular spasm of the left lower limb severe enough to fracture the plaster cast. The leg was fully flexed at the knee and the left foot lay posterior to the right buttock. No other limb was involved in the spasm. Voluntary motor function in the upper limbs was normal. Ventilation was not impaired.

The immediate relief of pain was essential, bearing in mind that previous attacks had occurred and that further recurrences were possible when the effects of therapy wore off. Spinal anaesthesia seemed inappropriate since frequent repetition of the block would probably be necessary. In addition, some authors consider obvious disease of the central nervous system to be a relative contraindication to spinal or epidural anaesthesia although we accept that a good argument could be put forward for the use of regional blockade in the situation under discussion.

It was therefore decided to treat this patient with intravenous magnesium sulphate. Slow intravenous injection of magnesium sulphate 2 g produced a dramatic response. Within 30 seconds the pain was relieved and the left leg, which had previously been fixed in flexion, became flaccid and could be placed in the normal anatomical position.

Spasms returned after 4 hours, and a further magnesium sulphate injection was given with similar effect. Four-hourly intravenous injections of magnesium sulphate 2 g were therefore ordered and treatment continued over a 72-hour period. Serum magnesium samples were taken 2 hours after each dose. On no occasion was the serum magnesium level elevated (normal range 0.75-1.1 mmol/l). On occasions, pain and hypertonus recurred before the end of the 4-hourly dosage interval but at no stage was any muscle weakness evident in the upper limbs, nor was respiration embarrassed.

After 72 hours of this regimen, the patient was transferred to a specialist neurosurgical unit for definitive management.

Discussion

Magnesium is the fourth most abundant cation in the body and, after potassium, the second most important intracellular cation. It acts as a co-factor for any enzymatic reaction requiring adenosine triphosphate and plays a key role in the regulation of cardiac excitability, skeletal muscle function and neuromuscular transmission.

Infusions of magnesium salts have a well-established role in the treatment of hypomagnesaemia and in the management of pre-eclampsia, and have been suggested in the management of tetanus. Consequently appropriate and safe dosage regimens are well documented.

When the patient presented, the cause of his pain and spasticity was not known with certainty but the presence of pain would suggest that the autonomic nerve supply was intact. Hypomagnesaemia may cause frank tetany, but he did not seem to have any of the common causes of magnesium deficiency. Furthermore, the distribution of the spasm was unlike that normally associated with tetany.

The decision to use magnesium was based on the knowledge that magnesium inhibits both the release of acetylcholine and depolarization at the motor end-plate. However, the magnesium levels required to achieve the desired result in this patient were insufficient to interfere with neuromuscular function in normally innervated muscle. This poses the question why the spastic muscles appeared exquisitely sensitive to the effects of magnesium.

There may be several contributory factors. Spasm is due to the uncontrolled discharge of the involved anterior horn cells. Nerve terminals in the affected muscle groups might release smaller amounts of acetylcholine per neuronal discharge, the spasm being maintained by the high discharge rate. Alternatively, the muscle may have been less sensitive to the effects of released transmitter substance. The latter explanation seems unlikely as inactive muscle usually becomes hypersensitive to acetylcholine. In either case, the inhibitory effects of magnesium would be amplified. Penetration of magnesium ions into the sarcoplasm would allow competition for calcium-
binding sites on troponin C and would interfere with calcium release from the sarcoplasmic reticulum.11

A further possibility involves the intact autonomic supply in this patient. It is known that adrenergic discharge increases the contractile response in striated muscle,12 and thus may have contributed to the sustained contraction from which our patient suffered. Magnesium inhibits the release of catecholamines from adrenergic terminals.13 The effectiveness of the magnesium infusion may have been the result of a combination of the various effects described above. Another possible explanation is provided by the work of Gordon,14 who showed that in chronically denervated muscle magnesium decreases the severity of contracture following acetylcholine-induced depolarization, possibly by interfering with calcium influx.

A continuous infusion might have provided better control of symptoms than the intermittent regimen employed in this case. Magnesium sulphate infusions may be valuable as a simple, effective means of controlling painful muscle spasms in the paraplegic patient.

REFERENCES

Fetale dwarsligging veroorsaak deur 'n reuse-leiomioom in die laer segment van die uterus

'n Gevalbespreking

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Summary

A 20-year-old primigravida presented at 39 weeks' gestation with the fetus lying in the transverse position. Ultrasound examination indicated an anterior placenta praevia grade I - III. During a vertical lower uterine segment caesarean section, a giant intramural leiomyoma of approximately 25 x 25 cm was found. The uterine incision was extended into the upper segment and a healthy male fetus of 2568 g delivered. The placenta was situated anteriorly but did not extend into the lower uterine segment. A myomectomy had to be performed to enable closure of the uterine incision.

The association of a transverse lie with anterior implantation of the placenta may result in a false ultrasound diagnosis because the lower anterior portion of the uterine wall and the attached portion of the placenta simulate a placenta praevia. In our case, an unsuspected leiomyoma in the lower segment contributed to this.

Furthermore, this case illustrates the advantage of using a lower uterine segment vertical incision when performing a caesarean section for transverse lie. The incision can then readily be extended into the upper uterine segment when necessary.

Gevalbespreking

'n Twintigjarige primigravida is opgevolg deur die voorgeboorte- en geboorte- kliniek van die Tygerberg-hospiital vanaf 35 weke swanger­skapsduur. Die voorgeboortelike verloop was ongekompliseerd tot 39 weke swanger­skapsduur, toe sy tekens van ligte pre­eklampsie ontwikkel het. Haar bloeddruk het verhoog van 120/70 mmHg tot 140/90 mmHg en 'n spoor proteien het in die urien verskyn. Verder is met buikundersoek 'n dwarsligging van die fetus gediagnostic. 'n Ultraklankondersoek het ook