

Severe systemic hypotension during repair of leaking large meningocele

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Abstract

We report a case of a 4-month-old male infant with lumbar meningocele. During surgical repair there was severe systemic hypotension. Though the occurrence of intraoperative hypotension during meningocele repair has been suggested, the exact mechanism remains unknown. While fluid loss is believed to be the main cause of the systemic hypotension, it seems unlikely. We discuss the possible mechanism of the severe hypotension during the intraoperative period in our patient.

Key words Meningocele · Intraoperative complication · Systemic hypotension

Introduction

Meningocele is a dysraphic condition of the spine. These lesions result from failure of the neural tube to fully close during embryogenesis [1]. Meningocele has an incidence of 20 per 100 000 births [2] and may be seen anywhere along the spinal cord, giving rise to a variety of neurological defects in the lower extremities, bladder, and bowel. The defects are usually midline and are covered with a transparent membrane, allowing leakage of cerebrospinal fluid (CSF). This leakage of CSF can cause volume and electrolyte disturbances which need perioperative correction. While these complications can be corrected early, the intraoperative occurrence of systemic hypotension needs anesthetic vigilance.

Case history

A 4-month-old male infant, weighing 6 kg, was admitted to the neurosurgical ward with presenting complaints of a large swelling in the lower back since birth. He had been delivered as a full-term baby by normal vaginal delivery. He had no impairment of bowel or bladder and no weakness of the lower limbs. The size of his head was normal and there was no delay in developmental milestones. On local examination, there was a large cystic swelling in the lumbar region, measuring 13 × 12 cm. Magnetic resonance image showed a midline laminar and soft-tissue defect in the L2 region (Fig. 1). A diagnosis of lumbar meningocele was made and elective excision and repair was planned. His cardiovascular and respiratory systems were unremarkable. Results of all routine investigations were within normal limits.

Following adequate fasting, the patient was taken to the operating theater and routine electrocardiograph and pulse oximeter monitors were attached. General anesthesia was induced with sevoflurane and oxygen; intravenous access was gained on the dorsum of the left hand using a 24-G cannula. Intravenous fentanyl 2 µg·kg⁻¹ was administered, followed by rocuronium 1 mg·kg⁻¹ to facilitate tracheal intubation with a 4.5-mm endotracheal tube. Anesthesia was maintained with sevoflurane in a mixture of oxygen and nitrous oxide (1:2) to maintain an end-tidal carbon dioxide of 34 ± 2 mmHg. Then the patient was turned to the prone position. After this positioning, the airway was confirmed again. At the time when the sac of the meningocele was repaired, there was aspiration of about 150–175 ml of CSF and an associated fall in systolic blood pressure, from 100 mmHg to 40 mmHg. There was bradycardia and the heart rate dropped from 150 beats·min⁻¹ to 100 beats·min⁻¹. This was followed by tachycardia, the heart rate being 180 beats·min⁻¹. Even after proper fluid replacement with 100 ml crystalloid and 100 ml blood, the blood pressure did not improve.



Fig. 1. Magnetic resonance image, showing midline laminar and soft-tissue defect in the L1–L2 region

The total duration of this hypotensive episode lasted about 15 min. However, immediately after closure of the surgical wound and change of position from prone to supine, blood pressure came back to normal baseline values. The neuromuscular block was reversed with neostigmine and glycopyrrolate. The trachea was extubated and the patient was taken to the neurosurgical intensive care unit. During the next 24 h all vital signs were stable, and on the second postoperative day the patient was taken to the ward. At the end of 10 days, the patient was moving all four limbs and was discharged with neurologically intact status.

Discussion

A literature search revealed primarily surgical and medical complications related to meningocele. Some of these were: worsened neurological level, wound dehiscence, wound infections, CSF leak, postoperative ileus, symptomatic Chiari malformation, shunt infection, necrotizing enterocolitis, and problems related to kyphectomy [3]. Though the occurrence of intraoperative hypotension during meningocele repair has been suggested, the exact mechanism remains unknown. While systemic hypotension is usually attributed to fluid loss, this seems unlikely in patients with meningocele as the cavity of the meningocele is in communication with the subarachnoid space and not the systemic circulation.

In our case, the patient failed to respond to fluid and blood administration. This means that volume deficit was not the reason for the hypotension. It is likely that the sudden leakage of CSF may have led to the development of a pressure gradient between the vertebral column and the intracranial compartment. This could have led to herniation of the brain (coning) and stretching of the vagal nuclei in the floor of the fourth ventricle. The resultant bradycardia may have caused systemic hypotension as well, because, in the pediatric age group, blood pressure is dependent on heart rate. However, the hypotension continued even after the bradycardia. The tachycardia that followed a few minutes later could have been the result of a compensatory mechanism in response to the persistent hypotension. It is also possible that a downward shift of the brain, with consequent traction on pain-sensitive receptors in the dura mater from a reduced CSF supporting cushion produced the tachycardia. Therefore, the vigilant role of the anesthetist can again be emphasized. Knowledge of the causation of hemodynamic disturbances during meningocele repair is important. While the patient can sustain minor brain shifts, gross herniation may even produce asystole. Hence, proper monitoring and quick intervention with fluids, vasopressors, and atropine during perioperative events is essential.

References

1. Botto LD, Moor CA, Khoury MJ (1999) Medical progress: neural-tube defect. *N Engl J Med* 341:1509–1519
2. Stein SC, Feldman JG, Friedlander M (1982) Is myelomeningocele a disappearing disease? *Pediatrics* 69:511–514
3. Pang D (1995) Surgical complications of open spinal dysraphism. *Neurosurg Clin N Am* 6:243–257