Odontoidectomy in the treatment of neurogenic hypertension

Case illustration

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Study data have indicated a possible connection between essential hypertension and neurovascular compression of the brainstem.1–4 To support this mechanical compression theory, we report on a patient with medically refractory hypertension due to odontoid and basilar artery (BA) compression of the anterior medulla oblongata.

This patient had been well until she was 23 years old, when she developed an occipital headache. At that time her essential hypertension, which had been diagnosed 1 year previously, became medically refractory. Her blood pressure ranged from 170/100 to 260/120 mm Hg, despite efforts to treat it with urapidil, carvedilol, amlopidine, rilmenidine, minoxidil, enalapril, hypothyazid, amiloride, and Lasix. The extensive protocol and a neurophysiological examination revealed nothing abnormal.

Magnetic resonance (MR) images of the craniocervical region demonstrated an anterior BA invagination due to the assimilation of the atlas with the skull. The ventral aspect of the medulla oblongata was compressed by a soft-tissue mass that surrounded the odontoid. This tissue mass caused the vertebral artery (VA) to compress the left rostral ventrolateral medulla oblongata ([RVLM]; Fig. 1). Any change in the position of the head did not influence the patient’s blood pressure.

As she was being placed in halo traction, her blood pressure suddenly dropped to 90/60 mm Hg; antihypertension medication has not been needed since then. After an uncomplicated transoral resection of the anterior ring of C-1 and the odontoid process of C-2, the patient was placed prone and an occiput–C3 posterior fusion was performed using occiput rods (Cervifix system; Synthes, Oberhof, Switzerland) and autologous bone grafting. The patient was discharged 1 week later. Four weeks after surgery she returned to her studies at the university. She has been followed up for 18 months and has remained normotensive (110–130/70–80 mm Hg; Fig. 2).

This case conforms to the concept that there is a causal relationship between vascular compression of the left RVLM and essential hypertension.

References


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