Patients with Chiari I malformation can present with a variety of symptoms, although headaches are a common presentation. Cardiovascular compromise, especially in the young patient, is seemingly rare in this form of hindbrain herniation. Hypertension has previously been treated by microvascular decompression of the brainstem, but results have varied.\(^3\)\(^-\)\(^{10}\)\(^,\)\(^{15}\) We report on a child with preoperative chronic idiopathic hypertension that resolved after decompressive surgery for Chiari I malformation.

**Case Report**

**History and Presentation.** This 16-year-old boy presented with occipital cervical headache, clumsiness, and dizziness. Magnetic resonance imaging revealed a significant (24-mm) Chiari I malformation (Fig. 1) with no syringomyelia. Hydrocephalus and other craniocervical diseases were absent. Since the age of 10 years, this patient had also suffered chronic idiopathic hypertension (mean 142/90 mm Hg, range [systolic] 132–145 mm Hg, and range [diastolic] 81–95 mm Hg). Hypertension had been noted on numerous clinic visits to the pediatrician. There was no significant reduction in blood pressures measured in the upper compared with lower extremities. Diet modification and exercise failed to resolve his hypertension. Pediatric cardiology evaluation included the following tests and parameters: urinalysis, electrolytes, blood urea nitrogen, creatinine, glucose, calcium, phosphorus, uric acid, lipid panel, complete blood count with differential, platelet count, 24-hour urine, urine/serum catecholamines, hormone level (thyroid and adrenal), 24-hour urine sodium excretion and blood pressure monitoring, plasma renin, renal artery Doppler ultrasonography, chest radiography, liver panels, and electrocardiography. All of the above tests were found to be within normal limits. Further evaluation included a workup for the presence of a neoplasm and physical examination, both of which yielded unremarkable results. There were no heart murmurs or other anomalies noted on chest auscultation. No systemic findings of chronic hypertension were demonstrated. Medications included compliant use of labetalol (200 mg) and antacids. With medication, this patient exhibited a mean blood pressure of 129/86 mm Hg. There was no family history of heart disease or hypertension.

In our clinic, physical examination demonstrated hyperreflexia of the left leg compared with the right. Additional neurological parameters were within normal limits. There was good cervical range of motion.

**Operation.** After opening the dura mater over the posterior fossa, the descending cerebellar tonsils were noted to be extremely compressed and frankly herniated through the dural opening. The cervicomedullary junction was caudally displaced as evident by a C-1 obex.\(^1^4\) No anomalously located arteries were observed. Pericardium-augmented duraplasty was performed. Intraoperative blood pressure ranged from 132/91 to 124/88 mm Hg.

**Postoperative Course.** Postoperatively and while receiving antihypertension medication, the mean blood pressure...
During the patient’s 3-day hospitalization was 124/83 mm Hg. After surgery, the child fared well; at 2-month follow-up examination, all symptoms had resolved, including hypertension. The patient no longer receives antihypertension medications, and throughout the follow-up period the mean blood pressure has been maintained at 123/78 mm Hg.

**Discussion**

Neurovascular compression of the brainstem as a causative factor in some patients with arterial hypertension has been discussed. Both experimental and clinical evidence support the notion of neighboring vessel–induced compression of the rostral anterolateral medulla and nerve root entry zones of the ninth and 10th cranial nerves, particularly on the left side.\(^3,8,9,14\)

Microvascular decompressive procedures have been performed for the treatment of essential hypertension, and success has varied.\(^3,8,10,15\) Frank, et al.,\(^7\) reported long-term follow-up results in eight patients who underwent microvascular decompression for essential hypertension; three patients remained normotensive while receiving decreased amounts of antihypertension medications, two patients required gradual increases in antihypertension medications after the 1st year (with arterial blood pressures 10–15% lower than baseline), and three patients suffered serious cardiovascular and renal complications. Jamnetta, et al.,\(^8\) have performed microvascular decompression to treat the left rostral anteromedial medulla in 42 patients and reported normalization of blood pressure in 32 and improvement in four. Patel\(^11\) has recently mapped the retroolivary sulcus in humans and found that stimulation here produces hypotension consistently followed by a reflexive hypertensive response.

Chiari I malformations have not historically been thought of as a cause of idiopathic hypertension. Makhmudov, et al.,\(^10\) have reported on an adult woman with Chiari I malformation in whom preoperative blood pressure was 190/100 mm Hg. After microvascular decompression to treat the left posterior inferior cerebellar artery, this patient’s blood pressure stabilized to 120/80 mm Hg.

Garland, et al.,\(^4\) recently theorized that some cases of orthostatic intolerance are related to hindbrain compression. They concluded, however, that cerebellar tonsillar herniation is not a common cause of orthostatic intolerance. Ireland, et al.,\(^6\) evaluated patients with Chiari I and II malformations, in all of whom there was preoperative abnormal control of heart rate in response to postural change; postoperatively, all patients were clinically asymptomatic. Arcaya, et al.,\(^1\) have reported on an adult patient with Chiari I malformation in whom sleep apnea and disturbances in the central regulation of arterial blood pressure were major components of the symptomatology. After posterior fossa decompression, both symptoms were markedly improved. Dobkin\(^2\) reported on a case of Chiari I malformation and exertional syncope in a patient in whom electroencephalographic slowing was observed without change in pulse rate or blood pressure. Graff-Radford and Godersky\(^5\) have reported on 30 adult patients in whom shunts were placed for symptomatic hydrocephalus. Chiari I malformation was present in one patient and systemic hypertension in all patients. No comment was made with regard to change in blood pressure following shunt insertion in the one patient with Chiari I malformation. Curiously, there has been one report of an adult patient with Chiari I malformation and pregnancy-induced hypertension.\(^12\) Could this case merely represent an exaggerated tonsillar herniation during pregnancy due to increased central venous pressures and induced hypertension? Tanaka, et al.,\(^13\) have reported on an infant with Chiari malformation in whom acute cardiovascular collapse developed (hypotension and bigeminy) during posterior fossa decompression.\(^7\) This compromise was resistant to pharmacological resuscitation and resolved after the decompressive procedure.

**Conclusions**

Although Chiari I malformation is a seemingly rare cause of hypertension, the clinician may wish to consider hindbrain herniation as a potential cause of idiopathic high blood pressure. In addition, future studies aimed at the identification of possible aberrantly located medullary arteries or osseous compression of the anterolateral medulla on the rim of the foramen magnum in patients with Chiari I malformation may shed light on this pathophysiology.

**References**

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