Case Report

Syndrome of inappropriate anti-diuretic hormone secretion (SIADH) and posterior cerebral artery ischaemic event: two uncommon complications following posterior fossa decompression

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Abstract

Neurosurgical procedures in cases of Type 1 Arnold Chiari Malformation (ACM) may result in a wide spectrum of complications.[1] We report a case of a sixty-four year lady who underwent an elective posterior fossa decompression for Type 1 ACM. The procedure was complicated by syndrome of inappropriate anti-diuretic hormone secretion (SIADH) and an ischaemic cerebrovascular event affecting the posterior cerebral artery. The association of these complications with the procedure is rarely described in the literature. In spite of the poor prognosis associated with such complications, the patient made a relatively quick and uneventful recovery.

Keywords

Neurosurgery, Arnold Chiari Malformation (ACM), Hyponatraemia, Syndrome of inappropriate anti-diuretic hormone secretion (SIADH), Cerebrovascular accident

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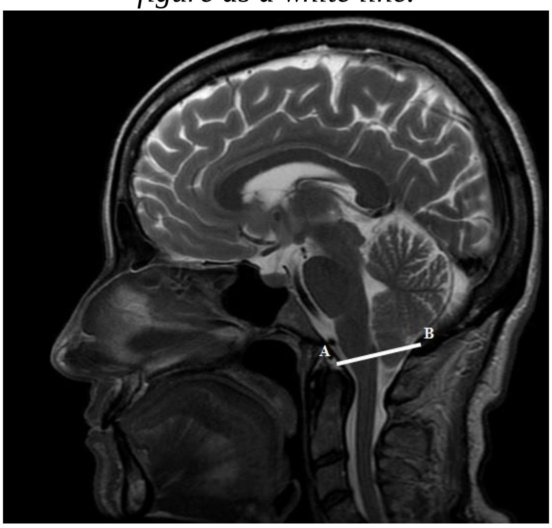
Case Presentation

A sixty-four year old lady was admitted for an elective posterior fossa decompression for Type 1 Arnold Chiari Malformation (ACM). The patient had presented with a longstanding history of headaches and lower limb weakness and numbness. The only positive finding on neurological examination was clonus. The diagnosis of Type 1 ACM was confirmed on magnetic resonance imaging (MRI) of the brain which revealed low lying cerebellar tonsils associated with cervico-medullary kinking (Figure 1). The patient had a past medical history of hypothyroidism and hypertension which were well controlled on medications.

Figure 1: *MR Head: Sagittal T2 view showing the cerebellar tonsils lying 7mm below McRae's line consistent with Type 1 Arnold Chiari Malformation. Mc Rae's line is a radiographic line drawn on a mid-sagittal section of an MRI joining the basion (A) and opisthion (B) which is depicted in this figure as a white line.*



Posterior fossa decompression was carried out uneventfully. Following the procedure, the patient was well and did not have apnoeic episodes throughout the night. On the first post-operative day, she was tolerating oral liquids and solids and her speech was normal. All of the former were indicative of intact brainstem function. However, by the second day post-operatively she started to complain of persistent headaches, nausea and fatigue.

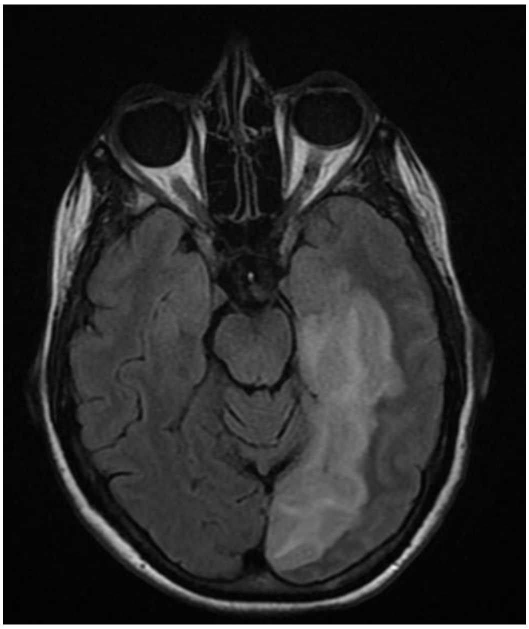
Physical examination revealed a drowsy patient who was afebrile, normotensive and was not tachycardic. She was not cooperative for a full neurological examination; however there was no pronator drift and no apparent focal neurological deficit. The rest of the examination was unremarkable.

Peripheral blood investigations revealed a serum sodium level of 120mmol/L (normal values: 135-145mmol/L). This had dropped from 140mmol/L overnight. Serum osmolality was 253mOsm/kg (normal values: 275-299mOsm/kg), urine osmolality was 728mOsm/kg (normal values: 50-1200mOsm/kg) and urine sodium was 291mmol/L (normal values: 54-190mmol/L). Serum cortisol level was elevated at 1804nmol/L (119-618nmol/L). Complete blood count, thyroid function tests, lipid profile, and total protein and albumin levels were normal. The aforementioned blood tests in addition with the patient's normal blood pressure satisfied the Bartter-Schwartz Diagnostic Criteria for the syndrome of inappropriate anti-diuretic hormone secretion (SIADH).[2] An urgent computed tomography (CT) scan of the brain revealed hypo-density in the left occipito-temporal region but no haemorrhage.

In view of the hyponatraemia, the patient was kept nil by mouth and started on 0.9% saline infusion which was restricted to 1.5 litres daily. Despite this management, the patient's sodium level was on the decline. After four hours, the sodium level decreased further to 115mmol/L and clinical symptoms worsened. In view of the risks of seizing, the patient was transferred to the intensive therapy unit for administration of intra-venous 1.8% hypertonic saline.

Twelve hours after the administration of hypertonic saline, the patient's clinical condition improved and her serum sodium level increased to 120mmol/L. Neurological examination was repeated since the patient was now more co-operative. The only positive finding was a right sided homonymous hemi-anopia. She underwent an urgent MRI brain which revealed an acute ischaemic stroke in the left posterior cerebral artery territory with a small focus in the medial aspect of the right cerebellar hemisphere (Figure 2). A magnetic resonance angiogram (MRA) revealed that a thrombus had occluded the left posterior cerebral artery. She was therefore started on aspirin and dipyridamole.

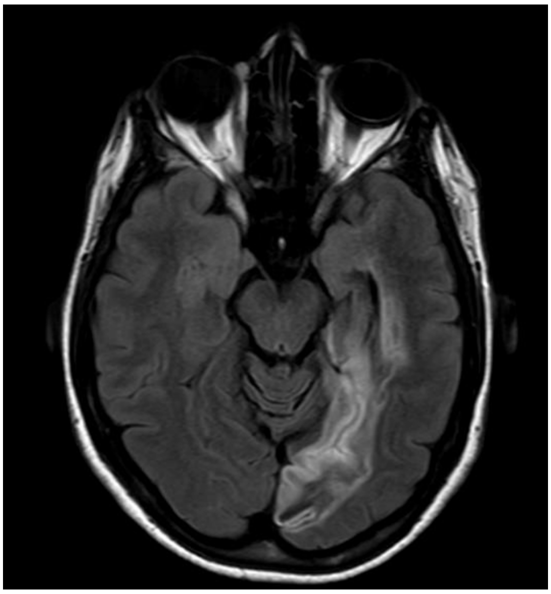
Figure 2: *MR Head: Axial FLAIR view showing hyper-intensity in the left posterior cerebral artery territory indicative of an acute ischaemic stroke.*



After four days of hypertonic saline administration, the serum sodium level gradually increased to 136mmol/L. At this point, the patient was transferred to the neurosurgical ward. She made a steady recovery with the help of the multi-disciplinary team. The patient was discharged fifteen days post-operatively with a serum sodium level of 137mmol/L and a visual field assessment which revealed right sided superior quadrant-anopia. By the time of discharge she was completely independent.

The patient was reviewed one month later at an outpatient appointment. She remained well. Her serum sodium level was 144mmol/L. A repeat MRI brain revealed post-infarct macrocystic encephalomalacia in the left posterior cerebral artery territory (Figure 3).

Figure 3: *MR Head: Axial FLAIR view showing post-infarct macrocystic encephalomalacia in the left posterior cerebral artery territory.*



Discussion

Arnold Chiari Malformation (ACM) is a group of congenital hindbrain and spinal cord abnormalities, characterized by herniation of the posterior fossa contents into the spinal canal through the foramen magnum. Type 1 ACM is characterized by the caudal descent of the cerebellar tonsils through the foramen magnum by at least 3-5mm. It may be associated with an elongated fourth ventricle, syringomyelia and medullary kinking.[1]

Type 1 ACM classically presents in adult life with symptoms of headaches and neck pain which are made worse with coughing and the Valsalva manoeuvre.[3] Other symptoms may include weakness, numbness and unsteadiness.[4] Presenting signs consist of a foramen magnum compression syndrome, a central cord syndrome or a cerebellar syndrome.[4] Diagnosis is confirmed on MRI as this reveals essential details on the anatomy of the cranio-cervical junction and any associated syringomyelia.[1, 5] In symptomatic patients, treatment involves posterior fossa decompression. Complications following such a procedure may include: respiratory depression, cerebrospinal fluid (CSF) leak, aseptic meningitis, wound infection, failure of procedure and pseudo-meningocoele formation.[4, 6]

In our case, posterior fossa decompression resulted in two complications, these being hyponatraemia secondary to SIADH and an ischaemic cerebrovascular event outside the brainstem. These are both uncommon complications of the procedure. Hyponatraemia is particularly common in neurosurgical patients. Its incidence is generally reported following subarachnoid haemorrhage, traumatic brain injury, intracranial tumours and hypophysectomy; however it is rarely seen in patients undergoing spinal procedures such as posterior fossa decompression.[7] In addition, ischaemic events following posterior fossa decompression usually involve the brainstem following injury to the vertebral arteries or posterior inferior cerebellar arteries (PICA). The vertebral artery is at increased risk of injury during dissection of the posterior arch of cervical vertebra 1. The PICA can be damaged during extra-dural exposure or during intra-dural dissection.[8] In this case, the ischaemic event involved a thrombus occluding the posterior cerebral artery.

Hyponatraemia is an important electrolyte disorder in neurosurgical patients. Signs and symptoms of hyponatraemia may be more pronounced in such patients due to the presence of co-existent factors that may cause cerebral irritation. In this case, breathing assessment and close monitoring of the patient’s oxygenation were of paramount importance in view of the close relationship of the procedure with the brainstem. Hyponatraemic seizures may occur at higher than usual plasma sodium concentrations in the presence of cerebral irritation from hypercapnia, hypoxia and/or cerebral oedema.[7] The two most common causes of hyponatraemia following neurosurgical procedures are SIADH and cerebral salt wasting (CSW).[9] Differentiating the two conditions is essential, as their treatment is different.[10]

In SIADH, there’s excessive unbalanced free water retention secondary to inappropriate anti-diuretic hormone (ADH) secretion. In CSW, the exact mechanism is still not completely understood, however natriuretic peptides play an important role. SIADH and CSW share common features i.e.: high urine osmolality, low plasma osmolality, low serum sodium level and high urine sodium level. The main distinguishing feature is the extracellular fluid volume state of the patient.[10] In SIADH, there’s a volume expanded state resulting in a euvolaemic or hypervolaemic patient. In fact, a euvolaemic status is one of the Bartter-Schwartz Diagnostic Criteria for SIADH (summarized in table 1). On the other hand, in CSW, there's renal salt wasting resulting in a contracted extracellular fluid volume, hence a hypovolaemic patient. Table 2 summarizes some of the differences between SIADH and CSW.

Table 1: *Bartter-Schwartz Diagnostic Criteria for SIADH*

Bartter-Schwartz Diagnostic Criteria for SIADH[2]	Patient's Case
Hypo-osmolality (Plasma osmolality <280mOsm/kg)	253mOsm/kg
Inappropriate urine concentration (Urine osmolality >100mOsm/kg)	728mOsm/kg
Elevated urinary sodium (>40mmol/L) despite normal water and salt intake	291mmol/L
Patient is clinically euvolaemic	Normotensive with good urinary output
No diuretic use	None used
Exclude hypothyroidism and glucocorticoid deficiency	None present

Table 2: *Biochemical and clinical features of SIADH and CSW[11]*

Characteristic	SIADH	CSW
Extracellular Fluid Volume	Normal, Increased	Decreased
Urine Osmolality	High	High
Plasma Osmolality	Low	Low
Serum Sodium	Low	Low
Urine Sodium	High	Very high
Urine Output	Normal or Low	High
Treatment	Fluid Restriction	Fluids &/or mineralocorticoids

Hyponatraemia is a serious co-morbidity in neurosurgical patients as untreated this may lead to seizures, apnoea, coma and death. Hence identifying and treating the cause is essential. SIADH is managed according to the severity of the symptoms. Initially in mild to moderate severity, the patient should be managed with fluid restriction, however if symptoms worsen, hypertonic saline should be administered.[11] Fluid restriction should not be used in CSW as these patients are hypovolaemic and their blood pressure can drop further if they are deprived of intra-venous fluids. Instead they require 0.9% or hypertonic saline to maintain circulation.[12] In both situations, hyponatraemia should be corrected slowly at a rate of < 8mmol/L in 24 hours so as to avoid the risk of central pontine myelinolysis.[13]

This case also highlights the importance of having a low threshold to perform a CT brain scan in hyponatraemic patients after neurosurgical procedures. This is useful so as to assess the level of cerebral oedema and exclude any haemorrhagic or ischaemic insults. In this patient, the CT brain revealed hypodensity in the left occipito-temporal region indicating that an ischaemic stroke had occurred. This was valuable since the patient's confused state secondary to hyponatraemia, made visual field and neurological assessment challenging. Following the confirmation of an acute ischaemic event due to thrombus formation in the left posterior cerebral artery, the patient was started on anti-platelet agents to prevent further neurological sequalae.

Most posterior cerebral artery ischaemic events are caused by emboli from cardiac or proximal vertebral-basilar arteries. Local artherothombotic stenosis or occlusions of the posterior cerebral artery, as in this case, are less common causes of infarction.[14 - 15]

In conclusion, both hyponatraemia and ischaemic strokes outside the brainstem are uncommon complications following posterior fossa decompression. Studies have also shown that the development of hyponatraemia is a negative prognostic marker in patients with ischaemic stroke resulting in a longer hospital stay and an increased mortality rate.[16] Despite this, our patient was discharged fifteen days post-operatively with the only clinical deficit being superior quadrant-anopia, making this case noteworthy.

Acknowledgements

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