Posterior reversible encephalopathy syndrome and spinal epidural haematoma in a hypertensive patient

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EDITOR:

A 49-yr-old female suddenly experienced intense lower neck pain irradiating to her right arm and trunk. Within a few minutes, she developed right-sided weakness in her arm and leg. She had recently been diagnosed with hypertension but was on no treatment. On admission to the emergency hospital, she was afebrile with high systolic (190–210 mmHg) and diastolic (130–150 mmHg) blood pressure (BP). A myocardial infarction or a ruptured aortic aneurysm was initially suspected; however, laboratory investigations including normal troponin and creatine kinase levels as well as electrocardiography, echocardiography and thorax computed tomography scan excluded these conditions. A few hours later, the patient began complaining of intense headache and blindness and within a few minutes a prolonged tonic-clonic seizure occurred. On admission to our hospital, she was confused and somnolent. BP was 185/130 mmHg. Neurological examination showed severe right-sided paresis and sensory deficit sparing the face, enhanced tendon reflexes in both upper and lower limbs (right > left), and bilateral upgoing plantars. Her strength was 2/5 in the right upper extremity, 0/5 in the right lower extremity and 4/5 in the left upper and lower extremities.

Brain magnetic resonance (MR) scan showed multiple focal, mainly posterior, white and grey matter hyperintensities on fluid-attenuated inversion recovery (FLAIR)-sequences and diffusion-weighted images (DWI), suggesting vasogenic oedema (Fig. 1a, b). Posterior reversible encephalopathy syndrome (PRES) was diagnosed and intravenous valproate and clonidine were administered. The patient's neurological function slowly deteriorated with increasing weakness of extremities. We performed an MR scan of the spine. It showed a large extraspinal mass lesion compressing the dorsolateral surface of the medulla at the levels C8–T2 (Fig. 1c–e). The lesion was slightly hyperintense on T_1-weighted images and hyperintense on T_2-weighted images, strongly suggestive of spinal epidural hematoma. No abnormally enlarged vessels were observed on T_2-weighted or post-contrast T_1-weighted images, thus excluding the presence of an underlying arteriovenous malformation.

The patient was transferred to the neurosurgery department where she underwent a C4–C7 laminectomy followed by evacuation of a thick blood clot. Over the following days, her neurological condition rapidly improved and brain and spine MR scans repeated 2 weeks later showed almost complete remission. She was discharged to the ward with no further complications.

PRES is a clinico-radiological syndrome associated with several conditions including hypertension, pre-eclampsia/eclampsia, treatment with immunosuppressive drugs, haemolytic uraemic syndrome, acute glomerulonephritis, blood transfusion, i.v. globulin or erythropoietin administration, acute intermittent porphyria and severe hypercalcaemia [1,2]. The favoured pathogenetic theory suggests autoregulatory disturbance with hyperperfusion resulting in blood–brain barrier breakdown with reversible oedema without infarction [1,2]. Accordingly, MR study typically reveals hyperintensity on both echoes of a dual-echo T2-weighted sequence and either iso- or hypointensity on T1-weighted image brain abnormalities, including both grey and white matter. Parietal–occipital lobes are mainly involved although other cerebral structures are also frequently involved [1–3]. DWI sequences detect white matter oedema and also reliably differentiates between vasogenic and cytotoxic oedema. Moreover, high DWI signal abnormalities and apparent diffusion coefficient pseudonormalization often correlate with patient outcome and may represent the earliest sign of nonreversibility as severe vasogenic oedema progresses to cytotoxic oedema, which carries a worse prognosis [2,3].

One of the distinctive characteristics of PRES is the reversibility of radiological abnormalities once that treatment is instituted. Lowering BP, removal or significant reduction of the causative medication as well as the treatment of seizures are mandatory [1,2].

Spinal epidural haematomaseh accounts for 0.3–0.9% of the epidural space-occupying lesions, more often occurring in the cervicothoracic and thoracolumbar regions. It has been associated with anticoagulant therapy, vascular malformation, venous epidural plexus defects, inherited or acquired bleeding disorders and hypertension [4,5]. It typically begins with a sudden, localized, intense pain in the

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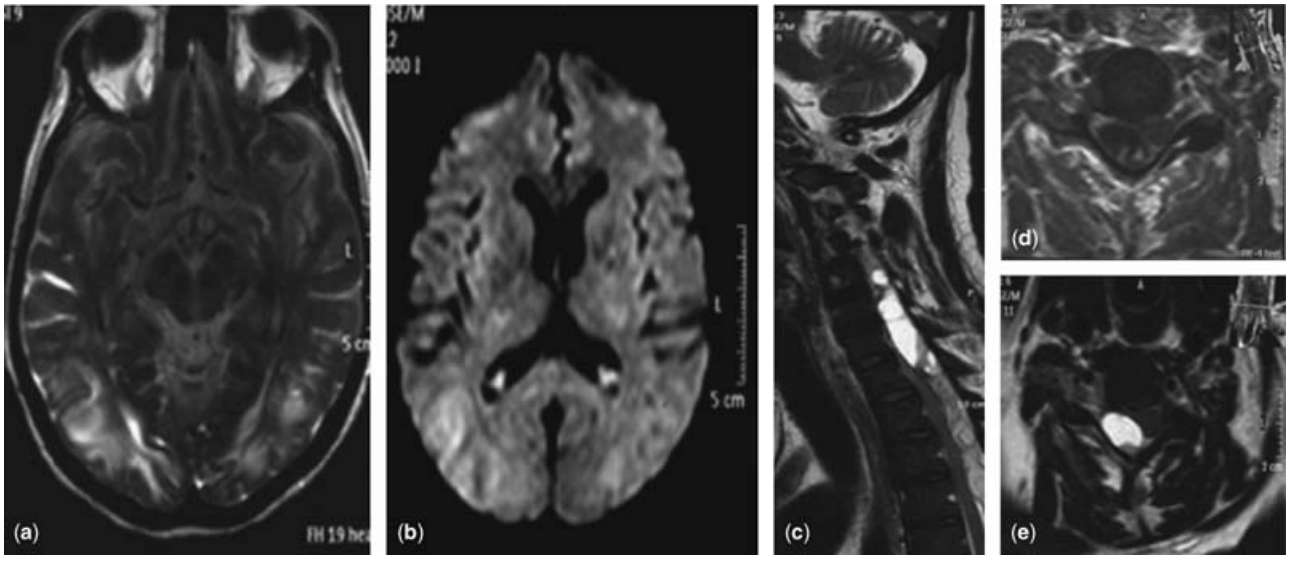

Figure 1.
*(**a**–**e**). Fluid-attenuated inversion recovery (**a**) brain MR image showing increased white/grey matter predominantly affecting the parieto-occipital areas and (**b**) diffusion-weighted image showing elevated intensity signal in a pattern typical for posterior reversible encephalopathy syndrome. (**c**) Sagittal T2-weighted, (**d**) axial T1-weighted and (**e**) T2-weighted spine MR images showing the spindle-shaped epidural hematoma on the cervicothoracic spine with characteristic signal intensity.*

neck or spine that progresses rapidly and often shows a radicular radiation. Neurological signs may appear asymmetrically and develop within minutes or even hours, corresponding to the level of the spinal cord compression. Clinical diagnosis is often difficult and it may be easily misdiagnosed as the differential diagnosis includes several chest and abdominal diseases [4,5]. Furthermore, progressing myelitis and polyradiculitis, myelocompressive diseases, including tumours and disc herniations should also be considered. MR scan is the diagnostic tool of choice as it allows the correct position and the extent of the haematoma, cord compression and to determine the presence of spinal cord oedema. Spinal epidural haematom may result in permanent neurological deficit or death if not properly treated. The standard management is emergency evacuation of the haematoma and spinal cord decompression. The more rapid is the surgical decompression, the more favourable is the outcome [4–6].

After the exclusion of coagulopathies, vascular malformations and other sources of haemorrhage, we suggest that the hypertension was the cause of both PRES and SEH in this patient. Moreover, persisting high BP values were documented at admission. The high BP and the increased systemic pressure exceeding the autoregulatory mechanisms of the cerebral vasculature might have been sufficient to overcome the blood–brain barrier and allow extravasation of fluid into the brain as well as of blood in the epidural space. Thus, not only the brain but also the spinal cord should be considered as a possible, albeit less common, site of hypertension-induced nervous tissue damage.

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Complete atrioventricular block following etomidate

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EDITOR:

Etomidate is an induction agent known for its cardiac stability; however, it has been reported to cause cardiac arrhythmias. A case of complete atrioventricular block caused by etomidate, a side-effect not previously reported in human subjects, is reported.

A 71-yr-old female was admitted to the psychiatric unit for a course of electro-convulsive therapy (ECT). She had well-controlled hypertension and had undergone an uneventful course of ECT the previous year. Her medications included nifedipine, doxasosin, dipyridamole, atorvastatin, indapamide, olanzapine, venlafaxine and lithium carbonate. She was not allergic to any medications. Examination of her cardiovascular and respiratory systems was unremarkable and assessment of her airway did not suggest any possible problems. Her blood biochemistry was within normal limits.

During her previous course of ECT, etomidate and suxamethonium had been used for induction of anaesthesia on each occasion. Her first treatment in this course of ECT had proceeded uneventfully when 18 mg etomidate and 50 mg suxamethonium were used. Induction was the same for the next two treatments; however, on both occasions after the suxamethonium was administered she developed what was reported to be a marked sinus bradycardia of 20 beats min^{-1} responding to intravenous (i.v.) atropine 600 μg within about 30 s. The treatments were otherwise uneventful and she made a full recovery each time.

At the next treatment, after the institution of monitoring and siting of an i.v. cannula, a pre-emptive dose of 600 mcg of i.v. atropine was given. Her heart rate (HR) increased from 100 to 110 beats min^{-1}. She was preoxygenated and induction of anaesthesia commenced with etomidate. After the administration of 17 mg of etomidate she developed complete heart block with the atrial rate remaining at about 100 per minute and intrinsic ventricular rate of 10 per minute with a good volume palpable carotid pulsation. The duration of this event was approximately 30 s when the rhythm reverted to normal sinus rhythm before any further treatment could be considered. The suxamethonium was not given and the treatment was abandoned. The patient made a full and uneventful recovery.

Subsequent echocardiogram and 24-h electrocardiography monitoring revealed no abnormalities and the cardiology team felt no further cardiac investigations were required. The decision was made that continuation of the course of ECT was warranted due to her ongoing psychiatric illness. She therefore continued her course of treatments with anaesthesia being administered by a consultant anaesthetist in the theatre recovery room, rather than the more isolated psychiatric unit, with external pacing pads applied as a precaution, prior to induction of anaesthesia. For the remainder of the course, anaesthesia was induced with propofol 1% with no problems.

Initially, it was thought that this patient's bradycardia was brought about as a side-effect of suxamethonium. However, it became clear that etomidate was the likely causative agent. Etomidate is presented as a colourless solution in an aqueous vehicle of water and 35% propylene glycol [1]. It is noted for its lack of cardiovascular side-effects, but rare known side-effects are transient bradycardia and cardiovascular instability. It is possibly not etomidate itself that causes the bradycardia, but its carrier propylene glycol [2]. It has, however, been shown to cause atrioventricular dissociation in isolated guinea pig heart studies [3].

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