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Case Report

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Posterior Reversible Encephalopathy in Opiate Toxicity

Marwa Najaf*

Introduction

Posterior Reversible Encephalopathy Syndrome (PRES) was first described by Hinchley et al. [1]. It is a clinico-radiological condition comprising neurological signs and symptoms such as visual disturbance, headaches, seizures, as well as radiological evidence of white matter vasogenic edema affecting the posterior occipital and parietal lobes of the brain [2]. Initially, it was thought that PRES is caused by acute hypertension which led to failure in autoregulation, cerebral hyperperfusion causing vascular leakage and vasogenic oedema [3]. However, as 20-30% of PRES cases present with no hypertension4 additional precipitant have been proposed. We present a case of PRES following general anaesthesia and opiate toxicity.

Case

A 77 year old female presented due to symptoms of belching and severe reflux. Her past medical history includes hypertension. She underwent repair of esophageal hiatus hernia repair using the abdominal approach. During the post-operative period she was given 10 mg intravenous morphine and an epidural was in situ with 2mch/mL fentanyl at 10mL per hour. Post operatively, she became drowsy and her respiratory rate declined to six. She had pin point pupils and was snoring. Naloxone was prescribed with good effect but she remained drowsy with a systolic blood pressure ranging from 89 to 90 throughout the night. She was treated with IV fluids and was started on a naloxone infusion. Her blood pressure responded and she became more alert. The following day (day one post-operatively), she developed complete blindness. On examination, she had complete loss of acuity bilaterally, full range of movements bilaterally and all other cranial nerves remained intact. The tone, power, reflexes and sensation of upper and lower limbs were also normal. An urgent CT scan of her head was arranged which showed ischaemic changes bilaterally in the occipital lobes, in the deep cerebellum and middle cerebellar peduncle with small asymmetrical cortical – subcortical focus of ischaemia in the left posterior frontal region. The image appearances were suggestive of PRES (Figure 1). Her epidural was now switched to plain epidural and she was reviewed by both neurologist and ophthalmologist who both agreed a diagnosis of PRES is most likely. It was decided to not prescribe any further doses of opiates and to monitor her blood pressure tightly to avoid any fluctuations. Three days later she began to regain her vision slowly. Her case was discussed at the neuroradiology multidisciplinary team meeting and a decision was made to perform an MR of the brain including a stroke sequence. MR of the brain showed ischaemic changes affecting predominantly the posterior occipital and parietal regions symmetrically (Figure 2).

Discussion

Although the exact pathophysiology of PRES is not fully understood, three hypothesis have been suggested [1]. Failure of cerebrovascular autoregulatory mechanism [2]. Cerebral ischaemia secondary to cerebral vasoconstriction [3]. Endothelial dysfunction with blood–brain barrier disruption [2].

Affiliation:

Queen Elizabeth Hospital Birmingham, UK

***Corresponding author:**
Marwa Najaf, Queen Elizabeth Hospital Birmingham, UK.

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A continuous cerebral blood flow is normally maintained irrespective of systemic blood pressure fluctuations as cerebrovascular autoregulation takes place in response to carbon dioxide reactivity and pressure amongst other substances [4,5,6]. Acute fluctuations in blood pressure or autonomic activity or arterial hypertension can render this system to fail [7,8]. This is the hypertensive theory often used to explain the pathophysiology of PRES and it proposes that fluctuations in blood pressure can either cause cerebral ischaemia (during periods of hypotension) or cerebral hyperperfusion (during times of hypertension when blood pressure rises above the autoregulatory limit) [2].

This patient was a chronic hypertensive with a baseline systolic at 140-170 mmHg. Prior to surgery she remained at her usual baseline blood pressure. During surgery this was monitored closely and was uneventful. Post-surgery she had opiate toxicity with pin point pupils, reduced GCS and reduced blood pressure. The possibility that this patient developed PRES due to elevated blood pressure prior to surgery is unlikely as chronic hypertensive patients often have a higher autoregulatory limit of blood pressure in comparison to normal individuals (mean arterial pressure >150–160 mm Hg) [3]. As such the magnitude of hypertension in this patient is unlikely to have been the primary precipitant of PRES.

A second hypothesis for the pathophysiology of PRES suggests that vasoconstriction and hypoperfusion causes brain ischaemia which leads to vasogenic oedema [3]. This patient could have developed PRES during the period of hypotension during the opiate toxicity on day zero post op. Her vision in the immediate postoperative period was not monitored and would have been difficult to assess due to reduced GCS. She was started on a naloxone infusion and IV fluids. When blood pressures GCS normalized the visual deficit was picked up.

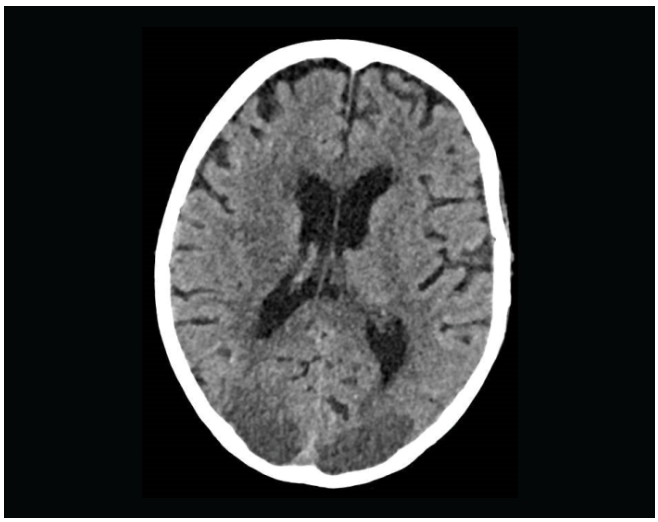

Figure 1: CT Head showing bilateral, symmetrical abnormal cortical and subcortical ischaemic change is seen in both occipital lobes. There is also mild local mass effect with gyral swelling.

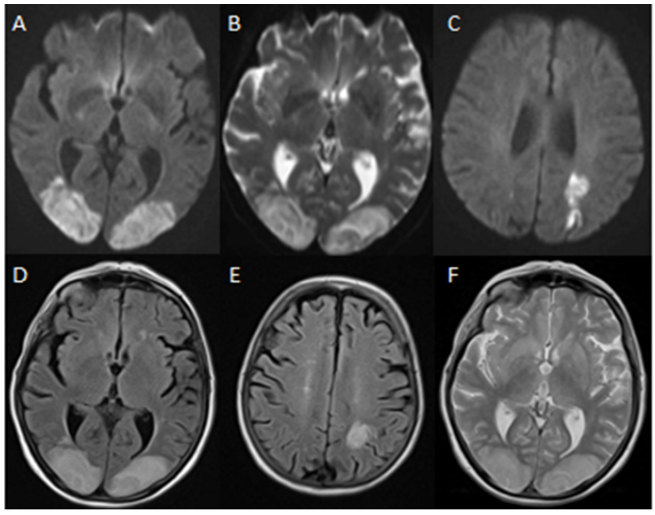

Figure 2: (A-C) Echo planar 2-dimentional diffusion Axial MRI. (D-F) Proton density T2 Axial MRI showing ischaemic changes affecting the posterior occipital and parietal regions symmetrically.

A final hypothesis describes endothelial dysfunction secondary to endogenous or exogenous toxins [3]. Endogenous toxins include those released during (pre)eclampsia or sepsis while exogenous toxins include immunosuppressive agents or chemotherapy. These toxins trigger endothelial cell activation and release of vasoactive agents which in turn increases vascular permeability leading to the oedema characteristic of PRES [9]. In addition, the vascular endothelial cells release vasoconstrictive agents which are thought to cause cerebral vasospasms [10]. In this hypothesis, the causes of PRES reported in literature are numerous. Table 1 highlights the common associations with PRES.1 [11-19]. However, the exact mechanism of why these cause PRES is again not fully understood. Use of opiates, spinal or general anaesthesia is not a recognised association.

Eran and Barak [20] report a case of PRES following general anaesthesia and intrathecal morphine. They describe a female admitted for video-assisted thoracoscopic wedge resection of a right lung lesion. The procedure was uneventful but post anaesthesia however similarly to this case post operatively the patient did not regain full GCS post reversal of anaesthesia [20]. Clinical and radiological findings were consistent with PRES in the absence of any other known risk factors for PRES. Rangi et al. [21] also report a case of post general anaesthesia PRES on day six post op. Although in this case risk factors were observed as the patient was receiving cytotoxic drugs for metastatic gestational trophoblastic disease. Torrillo et al. [22] describes a case of PRES on day eight post-operative abdominal aortic surgery. Hypertension was noted post operatively as well as renal ischaemia [22]. Chiu-Ming and Kwok-Hon [23] also report a case of PRES two days following 12mg 0.5% hyperbaric bupivacaine spinal anaesthesia. The procedure was successful with no complications at first attempt.

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In this case report, the patient had both hypotension, morphine toxicity and epidural fentanyl. Given the number of cases reported of PRES following general and spinal anaesthesia and intrathecal morphine, there should remain a suspicious that these are associated risk factors for PRES.

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Conflict of interest

There are no conflicts of interest to declare in writing this case report.

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