Case Report

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TANAFFOS

A Patient with Moderate Post-Operative Hypertension Presenting with Posterior Reversible Encephalopathy Syndrome: A Case Report

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Posterior reversible encephalopathy Syndrome (PRES) is a rare syndrome that is reversible in most cases but can rarely lead to irreversible brain damage and death. Most cases occur after rapid rise in blood pressure.

We report a 56 year-old Caucasian male with metastatic adenocarcinoma of the lung who presented with a pleural effusion for which thoracocentesis and thoracotomy were performed. He developed PRES on the third post-operative day following relatively moderate increase in blood pressure. It was diagnosed with diffuse weighted imaging (DWI) MRI. He eventually fully recovered from the event. PRES is a reversible syndrome, in most cases, that can be diagnosed with appropriate imaging studies such as MRI with DWI. This case report is of clinical importance to surgeons as well as neurologists. We speculate that post surgical patients are more prone to developing PRES at lower blood pressures than blood pressures required in healthy individuals to develop PRES. Patients' post surgical blood pressure should be monitored closely and maintained at lower levels to prevent PRES. It is essential to control and diagnose PRES at an early stage since it can be easily prevented and some cases proceed to irreversible damage. It should also be differentiated from an acute cerebrovascular event since its treatment and prognosis are markedly different from PRES.

Key words: Posterior reversible encephalopathy syndrome

INTRODUCTION

Posterior reversible encephalopathy syndrome (PRES), also known as reversible posterior leukoencephalopathy syndrome (RPLS), consists of edema without infarction in the cortical and subcortical regions of the brain involving the white matter which is distributed by the posterior circulation. Diagnosis of PRES can be made using non-invasive techniques such as magnetic resonance imaging (MRI) or computerized tomography (CT) scan. These cerebral lesions are reversible in most cases especially if treatment is initiated promptly. PRES can clinically present with signs and symptoms that include headache, seizures, blindness, and confusion; it rarely results in permanent neurological deficits (1, 2). The symptoms can present acutely or over a span of few days. Most of the patients' symptoms resolve completely after resolution of PRES. It can lead to permanent clinical changes in some cases as well as death in some others.

Its most common etiologies consist of hypertension with a rapid rise in blood pressure (>180/110 mmHg), pre-eclampsia/eclampsia, immunosuppressive treatment, autoimmune diseases and uremia (1, 3). The treatment of PRES is mainly supportive: treating the underlying cause

of PRES to avoid permanent damage to the brain. Controlling the blood pressure is of utmost importance in controlling the progression of PRES.

CASE SUMMARIES

A 56 year-old Caucasian male with a history of recurrent left pleural effusion, a left lung mass and pleural mass was admitted to our hospital. His symptoms included chest pain necessitating use of analgesics, persistent cough, and a documented weight loss of seven kilograms within two months prior to admission. There were no other co-morbidities. He had a 40 pack/year history of smoking and had quit one year prior to presentation.

On examination, the patient had left-sided chest wall tenderness and occasional wheezing. The rest of his exam was unremarkable. A chest x-ray showed a large left-sided pleural effusion. He was taken to the operating room where thoracocentesis, thoracotomy, and lysis of adhesions were performed. A mass was noted and biopsied. The biopsy report yielded diagnosis of moderately differentiated adenocarcinoma of the lung. A bronchopleural fistula was noted and repaired. The hospital medications included albuterol, ipratropium, simethicone, metoclopramide, omeprazole, levofloxacin and epidurally administered naloxone.

On the third post-operative day, the patient developed altered mental status along with agitation. He became stuporous, mute, and demonstrated inability to follow commands. The pupils were dilated and non reactive to light. He also presented with right sided weakness, and status epilepticus. He was started on diltiazem which controlled his blood pressure and levetiracetam which controlled his seizures.

The patient's systolic and diastolic blood pressures before surgery were in the range of 80 to 100 mmHg and 40 to 70 mmHg, respectively. On the day of this event his blood pressure varied from 120 to 160 mmHg systolic and 70 to 90 mmHg diastolic. His heart rate, breathing rate and temperature were within normal limits.

Serum electrolytes, arterial blood gas, blood count, and coagulation profile were within the normal range. Echocardiography and carotid Doppler done after the event demonstrated no significant abnormalities. CT scan of the brain done on the day of the event showed no intracranial pathology. An MRI on the second day after the event showed edema and abnormal signal bilaterally in the occipital lobes, thalami, and inferior medial temporal lobes on T1 weighted images (Figure 1). No evidence of tumor metastases to the brain was seen. On the 3rd day after the event (6th postoperative day), the patient recovered consciousness.

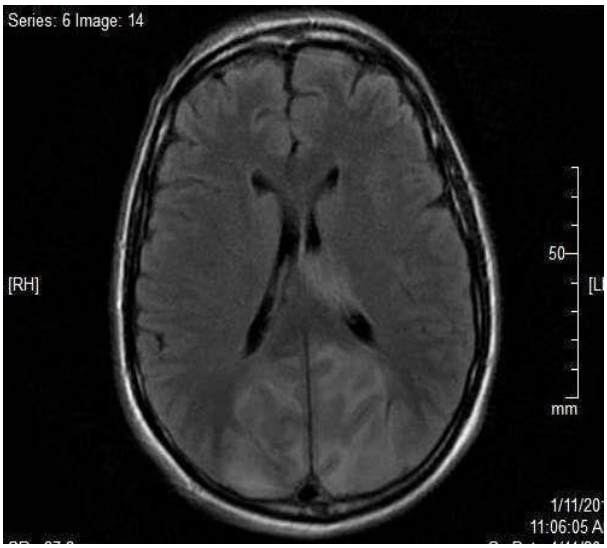

Figure 1. MRI with T1-weighted images showing edema and abnormal signal bilaterally in the occipital lobes, thalami, and inferior medial temporal lobes.

However, he had periods of drowsiness and was disoriented to time and place but oriented to person. He had complete loss of his right and partial loss of his left visual fields. His vision and memory gradually improved over the ensuing week. He could walk 300 feet slowly with minimum assistance and was consistently improving. An MRI done on the 7th day after the event (10th post-operative day) showed improvement in the edema involving the thalami, occipital region and temporal lobes bilaterally (Fig. 2). He was transferred to inpatient rehabilitation unit on the 7th day after the event.

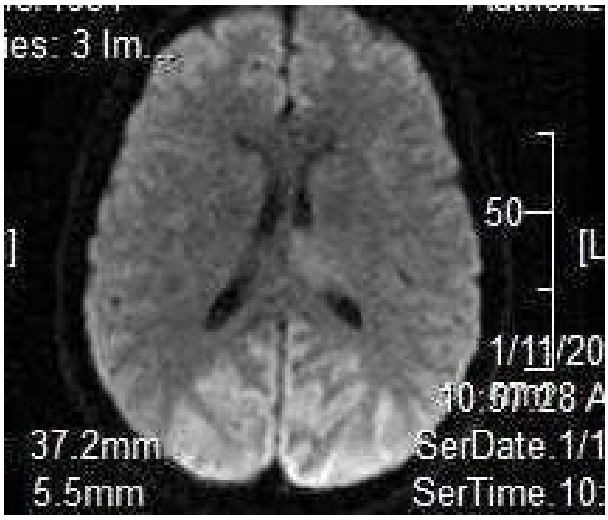

Figure 2. MRI with diffusion weighted imaging (DWI) showing improvement in the edema involving the thalami, occipital region and temporal lobes bilaterally.

DISCUSSION

The sequence of neurological events followed by a near-total recovery is compatible with PRES. Hinchey et al. described PRES as a well-defined syndrome in 1996 (4). PRES is generally a reversible syndrome if the cause is treated expeditiously. It can occur under various conditions such as hypertension, preeclampsia, eclampsia, and post- chemotherapy organ transplantation (5). The manifestations of this syndrome include transient blindness, confusion, seizure, headache, paresis and focal neurological deficits. Generalized seizures leading to coma can also develop. Seventy to eighty percent of patients have moderate to severe hypertension preceding PRES (6, 7). However, twenty to thirty percent of patients have blood pressures within a normal range. At times, the symptoms can occur within days to weeks of the precipitating event (1,6,7).

Two hypotheses have been proposed for the pathophysiology of PRES. One states that cerebral vasospasm is the cause, resulting in edema of the involved regions of the brain. The second states that there is a breakdown in cerebrovascular auto-regulation leading to cerebral edema (8). The latter is the more favored hypothesis in which acute hypertension can overcome the auto-regulation of blood pressure control. This in turn causes vasodilation, disruption of the blood-brain barrier, and transudation of fluid, resulting in cerebral edema. PRES can be diagnosed by using T2-weighted and T1 weighted MRI images or by a CT scan. In T1 weighted images most PRES lesions do not enhance. Contrast enhancement in T2- weighted images may yield more information related to the duration of lesion and severity of symptoms (1). Imaging modalities such as diffusion weighted imaging (DWI) with apparent diffusion coefficient (ADC) maps can prove the etiology of edema in PRES as either vasogenic or cytotoxic (1). DWI utilizes the measurement of Brownian motion principle of molecules. This increases its sensitivity in detection of early changes following a cerebrovascular event and also PRES compared to T1 and T2-weighted images. It also helps in elucidating the etiology of the event.

The syndrome mostly involves the posterior cerebral circulation as the name implies. The posterior circulation has poor sympathetic innervations as compared to the anterior circulation, predisposing the posterior territory to a less effective auto-regulation in case of an acute hypertensive event (9). Clinical and radiological changes are usually reversible but ischemic damage can occur if PRES is not identified in time and adequate therapy is not initiated (10, 11).

Regardless of the etiology of PRES, early detection and control of the cause is essential along with supportive therapy in the resolution of this syndrome. In the case of our patient we speculate that the transient and moderate post- operative rise in his blood pressure might have resulted in PRES. His post-surgical status may have resulted in PRES even without very high blood pressure.

A few cases of this syndrome, excluding pediatric cases and the ones with brain mass involvement, have been reported in the immediate post-operative period. One case reported the syndrome in a non-hypertensive patient with elevated post- operative blood pressure of 160 mmHg systolic and 90 mmHg diastolic, with no other identifiable etiology. The other four cases reported blood pressures up

to a maximum of 170 mmHg (systolic) and 110 mmHg (diastolic) (12). The post operative patients might be more prone to developing PRES. The blood pressures that are generally considered to be within the normal range post surgically may in fact lead to PRES. Patients who develop clinical manifestations such as visual loss and seizure, with focal neurological deficits in the post operative period should undergo an MRI or CT scan of the brain immediately. If PRES is suspected, supportive therapy is usually recommended with lowering of the blood pressure and close monitoring to reverse the process. Without blood pressure control the continued edema can lead to permanent brain damage and death.

CONCLUSION

In conclusion, we speculate that post surgical patients are more prone to PRES at a relatively lower blood pressure. This case report emphasizes the importance of tight control of blood pressure in post-surgical patients. The prevention of this syndrome is important since some cases of PRES can lead to irreversible brain damage and death. Prevention can also avert unnecessary and potentially complicating treatments for other clinical entities such as cerebrovascular accident. This case report is of importance to health care professionals providing care to post-operative patients.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Competing Interests

The authors declare that they have no competing interests.

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