Case Report

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Posterior Reversible Encephalopathy Syndrome (PRES): Deceptive Facade than Meets the Eye

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A B S T R A C T

Introduction: Posterior Reversible Encephalopathy Syndrome (PRES) is a reversible neurological disorder caused by endothelial dysfunction caused by hypertension and risk factors, due to which vasogenic edema crystalizes in the posterior cerebral hemispheres. This syndrome presented varied clinical symptoms including headache, vision changes and seizures. Prompt imaging and treatment is crucial in preventing end-organ complications and mitigating worse prognosis.

Clinical Case: A 67-old-female presented to ED with tonic-clonic seizure. She was given IV midazolam and IV naloxone by EMS while en route to ED. Her admission BP is 187/111 mmHg. Repeat readings showed persistently elevated systolic pressures (160/105 mmHg, later 180/86 mmHg). An MRI brain, there were scattered foci of hyperintense T2/FLAIR signal in the high frontal, parietal and occipital lobes, consistent with posterior reversible encephalopathy syndrome (PRES).

Treatment: She was then transferred to the floor for management of blood pressure, electrolytes replacement, opioid dose reduction and close telemetry monitoring.

Conclusion: PRES is a reversible neurological disorder caused risk factors like hypertension, drugs and autoimmune disorders. Prompt recognition and treatment of risk factors will result in complete regression of pathological associated with PRES. Prognosis of PRES is favorable as long as it is promptly diagnosed and treated.

Keywords: Neurological disorder, Seizures, Hemispheres, Brain, Encephalopathy, Hypertension, Stroke, Reversible White matter lesions, Hyperintensities

Abbreviations: PRES: Posterior Reversible Encephalopathy Syndrome; CT: Computed Tomography; COPD: Chronic Obstructive Pulmonary Disease; IV: Intravenous; MRI: Magnetic Resonance Imaging; FLAIR: Fluid-Attenuated Recovery; DWI: Diffuse Weighted Imaging; ABG: Arterial Blood Gases; EMS: Emergency Medical Services; ED: Emergency Department.

1. Introduction

PPosterior reversible encephalopathy syndrome is a reversible neurological disorder that mainly affects the posterior circulation of the brain1. Vascular dysfunction emanating from lessened ability to acclimatize to the changes in the systemic arterial pressure will evoke heightened vascular exudation into the interstitial space. These microvascular changes form the deep-

rooted basis for unfolding of vasogenic edema in PRES[1]. Prompt management by correcting the underlying cause and managing associated complications completely alters the course of disease by completely reverting the vasogenic edema reminiscent of PRES[1]. It is important to remember that it is one of those clinical syndromes that can have a very good prognosis given that it is diagnosed and treated at the earliest, thus warranting high clinical suspicion. We report the case of an elderly woman who presented to our facility with seizures, altered mental status, and hypertensive emergency. She was initially suspected of opioid overdose, but was subsequently diagnosed with PACE based on characteristic MRI findings. We would like to emphasize the importance of this completely rescindable neurological disorder, thus appraising its clinical significance in daily clinical.

2. Case Presentation

A 67-year-old female with a past medical history of hypertension hyperlipidemia, COPD, bipolar disorder, breast cancer (s/p B/L mastectomy), abdominal aortic aneurysm (s/p endovascularn repair) and chronic opioid use (Morphine 30mg, Oxycodone) for pain management was brought to the emergency department due to altered mental status. Family members found her AMS and called the ambulance suspecting she had mistakenly overdosed on morphine from her pill organizer.'

2.1. Prehospital Course

At home, EMS found her unresponsive with shallow respirations and pinpoint pupils. She was given 2 mg IV (Intravenous) naloxone en route with partial improvement. She experienced a generalized tonic–clonic seizure approximately 5 minutes prior to arrival, for which 2 mg IV midazolam was administered. She was then transported to the ED.

2.2. ED Evaluation & Management

On arrival, she was somnolent and not oriented. Her vitals were significant for hypertension of 187/111 mmHg, tachycardia (131 bpm), tachypnea (22/min) and SpO2 was 96% on room air and Temp 98.2 °F. Repeat readings showed persistently elevated systolic pressures (160/105 mmHg, later 180/86 mmHg). On neurological examination she was somnolent, disorientated, transient rightward tongue deviation, horizontal nystagmus and had spontaneous movement of extremities. Lab findings were significant for raised leukocytes of 12.9 × 103/μL, low hemoglobin of 9.1 g/dL, hypokalemia of 2.7 mmol/L, hypomagnesemia of 1.5 mg/dL, high anion gap of 18 mmol/L and elevated troponin of 584 ng/L, with repeat 3237 ng/L. ABG showed alkalemia of pH 7.49 with mild hypoxemia (PaO2 67 mmHg). Other blood work including blood glucose was normal. Urine toxicology was positive for benzodiazepines and cannabinoids but was negative for opioids. CT head showed no acute hemorrhage or infarct and CTA head & neck was negative for any large vessel occlusion. Chest X-ray at admission showed mild left-sided atelectasis. She received IV fluids, electrolytes replacement for potassium and magnesium, aspirin, IV labetalol & hydralazine. Her home morphine dose was reduced from 30 mg to 15 mg BID. She was then transferred to the floor for further management.

2.3. Inpatient Course

By the next morning, she was alert but oriented to person only. She had complete amnesia of the events leading to her hospitalization (she had no clue how she got there). She was surprised to learn about the past day's events. Blood pressure was improved to 162/91 mmHg, managed with oral tamsulosin. Labs showed partial correction of electrolytes (K 3.0, Mg 2.1) and declining troponin (2465 ng/L) and she received IV magnesium sulphate 2gm and oral potassium chloride 120 mEq total in her hospital course. Cardiology evaluated and recommended no intervention as her troponins were elevated due to demand ischemia rather than infarction. US 2D transthoracic echocardiogram was also negative for any wall motion abnormality. An MRI brain was performed to rule out possible stroke. It showed slightly increased DWI signal in the posterior cerebral hemispheres. On T2 and FLAIR, there were scattered foci of hyperintense T2/FLAIR signal in the high frontal, parietal and occipital lobes. The findings were consistent with Posterior Reversible Encephalopathy Syndrome (PRES) (Figure 1). She was given supportive treatment with blood pressure control, electrolytes replacement, opioid dose reduction and close telemetry monitoring. Her neurological status improved steadily, returned to baseline and no further seizure activities were observed during her hospital course.

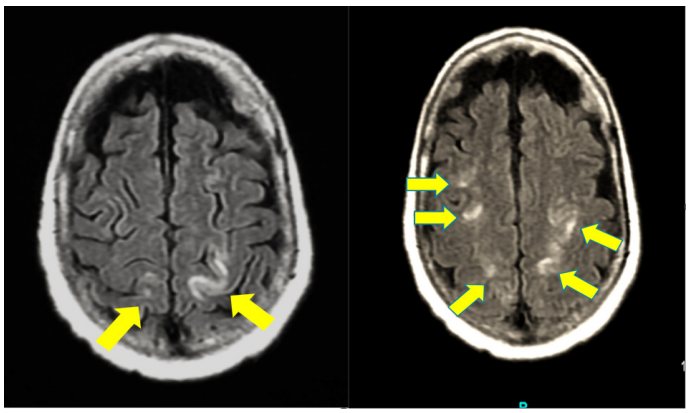

Figure 1: MRI Brain WO: MRI T2-weighted images demonstrate hyperintensities involving the temoporal, parietal and occipital lobes. These hyperintensities suggest fluid accumulation and edema between cortical and subcortical layers of in the cerebral cortex of aforementioned lobes. This transpires secondary to loss of autoregulation of cerebral micro-vasculature, thus eliciting percolation of fluid from the capillaries into the interstitial space. These turns of events will enkindle and crystalize the evolution of cerebral edema in the posterior territory of the brain.

2.4. Outcome and Discharge

By the discharge day, she was hemodynamically stable, her blood pressure was 134/72 mmHg and heart rate was 116 bpm. Labs showed a normal Potassium level of 5.1 mmol/L, Magnesium at 1.7 mg/dL, mg/dL. She was fully alert, her mental status returned to her baseline, there were no focal deficits, she was ambulating independently and tolerating oral intake. She was discharged home with her morphine dose reduced to 15 mg BID from 30 mg BID, she was given outpatient magnesium supplementation (magnesium oxide 400 mg daily), with her chronic home medications continued (statin, aspirin and psychiatric medications). She was counselled on safe medication practices. She was scheduled for neurology follow-up within 2 weeks, primary care follow-up in 1 week and a repeat brain MRI in 3 months to confirm resolution of PRES (Table 1).

Table 1: Vital signs during hospitalization.

Time Point	Blood Pressure (mmHg)	Heart Rate (bpm)	Resp Rate (/min	Temp (°F)	SpO, (%)
On arrival in ER	187/111	131	22	98.2	96 (RA)
In ER (repeat)	SBP: 160-180 DBP: 86-104	132	36	98	97
Floor admission	162/91	~100	20	98.6	96
Hospital course	SBP: 140-160 DBP: 70-90	95-110	18-22	97.8	95-97
Discharge	134/72	116	17	98.5	93 (RA)

ER: Emergency Room; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure

3. Discussion

Posterior Reversible Encephalopathy Syndrome (PRES) is clinical syndrome illustrated by edema of the posterior part of the cerebral hemispheres[1]. Hypertension is the most common risk factor for instigating PRES syndrome[5]. In our patient, hypertension and hyperlipidemia are only risk factors present. Hypertension (>160 mmhg) along with atherosclerosis can trigger the loss of autoregulation of cerebral vasculature, that is the ability to constrict and dilate for regulation of cerebral blood flow to the brain tissues[2,3]. Due to this, a chain of events starting with cerebral vessel desecration, blood brain barrier mutilation, increased vascular permeability, exuding of proteins & fluid into interstitial space materializes[2,4]. As a consequence, cerebral edema and petechial hemorrhages are the final repercussions[2,3]. Other less common causes that can be responsible for PRES can range from kidney diseases, preeclampsia, liver diseases, chemotherapy, immunotherapeutic agents to autoimmune diseases[3,4]. Preeclampsia, immunosuppressive drugs, sepsis, shock, infections, transplantation and autoimmune disease are more inclined to provoke endothelial dysfunction & vascular instability, thence triggering hypoxia and vasogenic edema[2,4]. All the aforementioned etiological factors are more destined to precipitate vasoconstriction, hypoperfusion and ischemia. A hybrid blend of these factors is known to be pivotal in instigating the commencement of PRES syndrome in the posterior zones of cerebral hemispheres. While the exact reason for this predilection to the posterior territory of brain is obfuscated at present, some clinical researchers speculated some theories behind this eventuation. This revolves around the premise that posterior circulation has less ability to acclimatize to the alterations in the blood pressure, thence in their capability to restrict blood brain barrier crumbling, fluid exudation and vasogenic edema inception as compared to anterior circulation[1,6]. On the grounds of this, occipital lobe and parietal lobe are involved in most cases. Notwithstanding temporal lobe, frontal lobe, thalamus and pons is involved in 91%, 82%, 68% and 50% respectively[6]. In our patient, there is predominant involvement of occipital, parietal and parietal lobes. This pathological fluid accumulation in the posterior brain tissues will crop up with clinical manifestations such as headache, encephalopathy, visual disturbances, seizures, hypertension, vomiting, confusion, lethargy, somnolence, restlessness, agitation, stupor, memory disturbances, altered speech and focal neurological deficits[1,3,4]. Specifically, spectrum of visual disturbances identified in this disorder include blurred vision, binocular diplopia, hemianopia, quadrantanopia, hemianopia, visual neglect, frank cortical blindness and anton syndrome[1,3,4,7]. With such a varied presentation, clinical judgement is inclined to be blurred due its impersonating neurological disorders including basilar artery thrombosis, venous sinus thrombosis, herpes simplex encephalitis, intracerebral hemorrhage, meningitis, vasculitis, hypoxic ischemic encephalitis, stroke and Transient Ischemic Attack[2]. It is not uncommon to encounter such a clinical scenario in the hospital setting, instantly when due diligence, high degree of clinical suspicion and swift workup will be highly valued in delineating the correct pathology towards PRES. CT scan brain might be helpful in some cases, but MRI brain should visualize specific changes including edema of the white matter in the vascular watershed areas of the parietal and occipital lobes of both cerebral hemispheres[5,2,3,8]. It is not unusual to find out cortical involvement, asymmetric lesions and hemorrhages[5,2,8]. It is always a challenge to fathom out these from changes from bilateral infarction of the posterior cerebral hemispheres. Within this context, sparing of the calcarine and para-median fissures of occipital lobe provides necessary omen for this delineation[3]. Moving forward, PRES patients who are subjected to periodic follow up MRI scans demonstrate dwindling and vanishing white matter lesions in contrast to stable or enlarging lesions in stroke[9]. Sometimes, magnetic resonance angiography or venography might be useful in differentiating CNS vasculitis or sagittal sinus thrombosis[1,2]. Reports suggest that PRES can occasionally involve cerebellum, brain stem, basal ganglia and frontal lobes[10-12]. Rarely, involvement of the grey matter and disruption of blood brain barrier was reported in few studies[10,13-15]. As PRES is identified, managing and careful monitoring of hypertension along with discontinuation of causative drugs provides remission of clinical symptoms. Towards this end decreasing blood pressure to 130-150/80-100 mm hg, keeping in mind that goal of blood pressure control should be no more than 25% during first 6h[1]. If necessary, admission to the intensive care unit and appropriate control of blood pressure might be necessary[16]. Treatment of seizures with antiepileptic drugs might be necessary in addition to control of blood pressure[17,18]. Although corticosteroids are known to helpful in relieving the vasogenic edema related to PRES, none of the clinical reports published so far afforded any tangible therapeutic benefit in their application and thus they are discouraged in PRES. The prognosis of PRES is pretty encouraging given that it is recognized early and appropriate interventions are given in a timely manner. Nevertheless, unrecognized PRES can be fatal as complications such as cerebellar herniation, focal neurological deficits, status epilepticus and intracerebral hemorrhage[3,19,20]. Finally, counselling for aggressive management of blood pressure along with lifestyle modifications for attenuating atherosclerotic related changes in the cerebral microvasculature is the secret recipe for prevention of this vascular neurological disorder.

4. Conclusion

PRES develops due to excess fluid extravasation from the cerebral microvasculature, thus spawning in vasogenic edema in the cerebral hemispheres. Hypertension and drugs are the most likely culprits. It has most proclivity to develop in the posterior aspect of the cerebral hemispheres, particularly in the occipital and parietal lobes. MRI can be instrumental in bringing to light hyperintensities in the watershed areas of posterior cerebral hemispheres. Management of hypertension, seizures and discontinuation of drugs involved might afford prompt symptomatic relief and also provides required impetus for backpedaling of white matter lesions seen in PRES.

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