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Drawing the Curtains: A case of Posterior Reversible Encephalopathy Syndrome (PRES) Post Upper GI Endoscopy

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ABSTRACT

Background

Posterior reversible encephalopathy syndrome (PRES) is a devastating condition with distinct clinical and radiological manifestations. Clinical features vary from acute illness with symptoms of transient headache, changes of mental status, seizures, and visual loss. Radiological features associated with PRES include extensive white matter changes mainly in posterior and parietal lobes consistent with brain oedema 1,2.

Clinical Case

A57-year-old man with oesophageal stricture presented to Endoscopy Unit planned for Upper GI Endoscopy and oesophageal dilatation. He has background history of benign stricture, GORD, and CLL. After the procedure, patient had low blood pressure associated with headache followed by sudden loss of vision and seizure. Urgent referral to Anaesthesia and on call medical team were requested. Initial CT Angiogram showed no intra or extracranial atheromatous disease and patent vertebra-basilar system. This was followed with MRI scan which showed high signal on diffusion with cerebellar and occipital hemispheres bilaterally with results favouring vasogenic PRES syndrome. Due to patient condition, he was admitted to ICU and treated medically with amlodipine and levetiracetam. Patient's symptoms gradually resolved with medical treatment and was discharged home after a week.

Patient was then seen in outpatient clinic and was doing well. No further symptoms. As patient requiring repeated Endoscopy and dilatations, further procedures were conductedunder GA. No intra or post procedural complications were observed.

Conclusion

Patients who develop PRES could have catastrophic neurological complications. Hence, high level of suspicion for PRES must be considered in patient who develops altered mental status, seizures and acute visions with hypertension post endoscopic procedure or administration of medications. The gold standard diagnostic imaging is MRI. Blood pressure control and discontinuation of any causative agents is the treatment.

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KEYWORDS: Upper GI Endoscopy, Blindness, PRES

BACKGROUND

POSTERIOR reversible encephalopathy syndrome (PRES), is a constellation of symptoms with clinical-radiological entity. It is also known as posterior leukoencephalopathy.

Posterior reversible encephalopathy syndrome was first described in 1996 by Hinchey et al and then proposed by Casey et al in 2000 as acute illness with symptoms of transient headache, changes of mental status, seizures, and visual loss in combination with radiological finding of extensive white matter changes consistent with brain oedema mainly affecting posterior and parietal brain lobes.[1-3]

Many factors act as trigger for this syndrome, including hypertension, pre-eclampsia, abnormal renal function and the use of immune suppressions. Other described causes include autoimmune disease, post organ transplant, systemic infection, and allogenic bone marrow transplantation. In the literature, majority of published articles are of case reports or case series. Of these reported cases of PRES are: post endoscopic Resection of pituitary adenoma13, in patient with SLE[11], in pre-eclampsia [9], post bevacizumab treatment [7], post Immunosuppression in patient with IBD[12] and post Transfusion with cerebral vasoconstriction[14].

CLINICAL CASE

This is a case of 57-year-old male scheduled for Upper GI Endoscopy and oesophageal dilatation with background history of benign oesophageal stricture. Other medical problems include; CLL, Hypertension and heartburns. He had 2 previous successful upper GI endoscopy and dilatation with no post procedure problems.

On Arriving to Endoscopy Unit, patient did not report any issues and his BP was 151/104 mmHg. Procedure was performed as planned with IV sedation, 5mg midazolam, 100mcg fentanyl. Finding: Oesophageal stricture which was dilated to 19mm with no problem during procedure.

In recovery, patient complained of severe headache. His BP post procedure was 174/96 mmHg and patient dose of amlodipine was given. Patient continue to complain of headache followed by generalised seizure and bilateral loss of vision. Patient was reviewed by stroke team and anaesthetist in recovery and decision for intubation was made. Urgent CT Head and Angiogram were performed to rule out CVA. This showed no intra or extracranial atheromatous disease and patent vertebra-basilar system.

MRI was then performed and showed high signal on diffusion with cerebellar and occipital hemispheres bilaterally involving the cortical surface favouring the diagnosis of PRES. Appearance in keeping with vasogenic rather than cytotoxic process. Patient was transferred to ICU and levetiracetem 750mg twice a day was started. Patient symptoms have improved and discharge to the ward on day four.

A repeat MRI was requested 5 days post initial event and showed increased T2 signal within the posterior temporal and occipital white matter and within the right cerebellar hemisphere. These radiological finding are still consistent with diagnosis of PRES. Patient was discharge home day 8 post incident for follow up in outpatient clinic with both surgeons and stroke team.

During his clinic visit, the patient was symptom free and his blood pressure was under control with amlodipine. It was discussed with the patient that a repeat endoscopy and dilatation are still required for his stricture as his symptoms were back. It was explained that further endoscopic procedures will be done in main theatre under GA and he will be staying overnight for observations. Patient was referred for pre-op assessment and scheduled for his repeat endoscopic procedure. The repeat procedure was performed under general anaesthesia with endoscopic dilatation with uneventful intra/ post-operative events. Patient stayed in High Dependency Unit for one night and then discharged home for repeat procedure in 2 months. Two subsequent procedures were performed with uneventful events.

DISCUSSION

The incidence of PRES has not been established and the cause in not yet completely understood. Previously reported cases are shown in the table below. There are 2 proposed pathophysiological theory regarding cerebral autoregulation: cytotoxic theory and vasogenic theory. The cytotoxic theory suggests that the rapid increase in blood pressure causes subsequent cerebral vasoconstriction and oedema formation. This theory was supported by Ito et al and Tajima et al. [4,6]The limitation of this theory is the inability to explain PRES development in patients showing no large vessel occlusion or vasospasm.[5] The vasogenic theory suggests that hypertension results in cerebral autoregulation abnormalities and subsequently cause cerebral vasodilatation and oedema. This is the aetiology in this case.

Table: Previously reported cases

Author	Country	Year	Journal	Reported Cases
Aygun et al.	Turkey	2010	J Turkish-German Gynecol Assoc	pre-eclampsia
Streck et al	Brazil	2011	Rev Bras Reumatol	patient with SLE
Seet and Rabinstein	USA	2011	Q J Med (International Journal of Medicine)	post bevacizumab treatment
Dou et al	Taiwan	2014	Headache The Journal of Head and Face Pain	post Transfusion with cerebral vasoconstriction X many cases
Silvestri	USA	2014	Journal of Clinical Neuroscience	secondary to blood transfusion
Rajan et al.	India	2014	Indian Journal of Anaesthesia	Post C-section under Spinal Anaesthesia
Zhang et al	China	2015	Quantitative Imaging in Medicine and Surgery	Late postpartum eclampsia complicated with posterior reversible encephalopathy syndrome
Bonington et al.	UK	2016	ACG Case Report Journal	post Immunosuppression in patient with IBD
Villelli et al	USA	2017	World Neurosurgery	Post Endoscopic endonasal Pituatary surgery

Although CT scan is likely to be obtained first to exclude a cute haemorrhage, MRI is the imaging modality of choice to diagnose PRES [5]. Characteristic radiological finding is the presence of oedema involving the white matter of posterior portion of both cerebral hemispheres, especially parieto-occipital region. Although PRES normally associated with white matter, involvement of grey matter on MRI was described in up to 94% of cases [3]. PRES is not just limited to parieto-occipital lobes, structures like cerebellum, brain stem, and frontal and temporal lobes can also be involved.

Other structures such as the brain stem, cerebellum, and frontal and temporal lobes may also be involved. Abnormalities of the subcortical white-matter are the rule, but the cortex and the basal ganglia are eventually affected

The currently accepted therapy for PRES regardless of the mechanism of cause is blood pressure control and discontinuation of any medication linked to its cause.

Post treatment, most patients usually have complete resolution of clinical and radiological findings as in this case. However, certain patients will have permanent radiological finding including, infarction (13-25%), post haemorrhage residual, atrophy, and laminar necrosis. The extent of imaging abnormalities has been found by Covarrubias et al to correlates with the severity of patient outcome[2].

CONCLUSION

Although it is rare and poorly understood, PRES must be considered in patient who develops altered mental status, seizures and acute visions with hypertension post endoscopic procedure or administration of medications. Treatment is of blood pressure control and discontinuation of any causative agents.

LIST OF ABBREVIATIONS

BP: Blood Pressure
CLL: Chronic lymphocytic leukemia
CT: Computed Tomography
CVA: Cerebrovascular accident
GA: General Anaesthesia
GI: Gastrointestinal
HDU: High Dependency Unit
ICU: Intensive Care Unite
MRI: magnetic resonance imaging
PRES: Posterior reversible encephalopathy syndrome

Conflicts of interest

Author declares no conflict of interest

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