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Frequent use of imaging modalities makes diagnosis of PRES Syndrome easier

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Abstract

Presentation

81 years old female presented via ambulance in ED in University Hospital Limerick with complaints of dizziness, vomiting for 4 days. During her stay in hospital, she developed diarrhoea with slight tenderness in in epigastric region. Her blood pressure medications were withheld due to fear of dehydration. which resulted in high blood pressure. Patient developed sudden onset confusion, irritability and speech problems.

Diagnosis

CT Brain and CT Angiogram showed no intracranial haemorrhage, acute territorial infarction or extra-axial collection, no space-occupying lesion, mass effect or midline shift. She had an MRI Brain which showed Diffuse T2/FLAIR hyperintensity seen in a subcortical distribution predominantly within the posterior occipital and parietal regions and to a lesser extent periventricular distribution. In the context of a hypertensive episode, Posterior Reversible Encephalopathy Syndrome (PRES) was clinically and radiographically the appropriate diagnosis.

Treatment

Patient was admitted to HDU and remained under constant monitoring. She was given anti hypertensives to bring down the blood pressure. Her symptoms resolved completed after 3 days of inpatient management of her blood pressure and was discharged home on antihypertensives with instructions to follow up in 4 weeks.

Discussion

With the improvement in imaging modalities such as MRI Brain, PRES being a rare disorder now is being reported increasingly. The diagnosis of these reversible cases is important, because usually they do not require any further investigations and symptomatic treatment is needed.

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Introduction

Posterior Reversible encephalopathy syndrome (PRES) is a neurological disorder in which patient presents clinically with acutely altered mentation, seizures, or headache with radiographic findings as white matter oedema usually found in the posterior cerebrum in a bilateral symmetric distribution and the symptoms are usually reversible.[1] These symptoms may take hours to days to develop and often with a background of uncontrolled hypertension.[2] Typical findings in MRI are hyperintense T2 signals or hypointense T1 signals in posterior cerebrum. Moreover, of this syndrome is not managed promptly, it can lead to irreversible brain damage such as cerebellar herniation and persistent focal neurological deficits.[3,4] Risk factors include hypertension, hypovolemia, cytotoxic medications or immunosuppressants leading to secondary hypertension, renal failure, sepsis and autoimmune conditions.[5] Among them, hypertension remains the most significant risk factor leading to complication such as PRES.[6] In the past owing to limited awareness, PRES has been underdiagnosed but now with improved imaging such as MRI, PRES is being studied more efficiently.

Case Report

An 81 years old female presented via ambulance in ED in University Hospital Limerick with complaints of dizziness, vomiting for 4 days. She reported that she felt dizzy, stumbled on her way to washroom, had a fall and then vomited. She did not lose consciousness, had recollection of the event and denied any chest pain or weakness of any side of body. She also mentioned that she had eaten preserved and frozen food that day, but did not have any diarrhoea, neither abdominal pain on presentation.

She had past history of non-insulin dependent diabetes mellitus, hypertension and alcohol excess. Her regular medications comprised of Aspirin 75mg once a day (OD), Rosuvastatin 40mg OD and Doxazacin 4mg OD. Her vitals on admission were blood pressure 121/66mmHg, pulse 76/min, temp 36.4 C, respiratory rate 18/min. She was noted to have irregularly irregular heart rate, ECG and Holter confirmed atrial fibrillation and considering her CHADS-VASc score of 5, she was started on anticoagulation Rivaroxaban 20mg Once a day and Rosuvastatin 40mg. Otherwise, she was neurologically intact along with cardiorespiratory and abdominal examination was normal. During her stay in hospital, she developed diarrhoea with slight tenderness in in epigastric region, her doxazocin was withheld. She had subsequent investigations for diarrhoea including CT Abdomen and Pelvis which showed diffuse mural thickening and mucosal hyper enhancement throughout the pylorus, duodenum and jejunum. There were small volume of non-enhancing low attenuating free fluid surrounding the abnormal duodenum and jejunum and regional mesenteric prominent nodes. Findings were suggestive of enteritis specifically involving the duodenum and jejunum. Her stool culture including viral panel, celiac serology, stool difficile was negative. She was managed accordingly and her diarrhoea settled with fluids while electrolytes were monitored.

During her admission, she was then noted to have high blood pressure ranging from 190//90 mmHg to 204/81 mmHg, for which regular antihypertensive medications were restarted along with Lercanidipine 10mg PO OD, Bisoprolol 1.25mg Once a day PO, Ramipril 5mg Once a day PO and a stat dose labetalol 200mg while monitoring her heart rate and she initially responded. Her 24-hour ABPM showed 178/84mmHg. Her blood glucose monitoring remained stable. However, she suddenly started appearing confused and developed expressive dysphasia. Immediately stroke team assessed her and she got CT Brain and CT Angiogram which showed no intracranial haemorrhage, acute territorial infarction or extra-axial collection, no space-occupying lesion, mass effect or midline shift. Prominence of the ventricles and CSF spaces was seen in keeping with age-related cerebral volume loss. Bilateral periventricular white matter hypo attenuation was seen in keeping with mild background small vessel white matter disease. Within an hour, her speech became appropriate and gradually in few hours she became oriented. For further investigation, MRI Brain was planned. Within 24 hours, she appeared confused again and showed fluctuating response on commands, occasionally speech was comprehensible but at times unable to follow commands. However, on neurological examination, she remained conscious, but not oriented in time, person or place. She was slow in following commands with poor memory recall. In regards to her speech, she had difficulty in finding words but was coherent. No dysarthria noted. Her cranial nerves examination was intact. Her motor examination was grossly intact with intact power and reflexes with downward plantar response. There was no nuchal rigidity. Considering hypertensive encephalopathy as a possibility, she was monitored in High Dependency Unit (HDU) with Labetalol infusion with a target blood pressure of 130-150 mmhg systolic blood pressure. She was also given GTN Patch and dose of Lercanidipine was increased from 10mg to 20mg and Bisoprolol dose increased to 2.5mg from 1.25mg OD and started Indapamide 1.5mg OD.

She had an MRI Brain which showed Diffuse T2/FLAIR hyperintensity seen in a subcortical distribution predominantly within the posterior occipital and parietal regions and to a lesser extent periventricular distribution. There was no associated restricted diffusion. No space-occupying lesion was noted. Involutional atrophy with associated dilation of ventricles and CSF spaces. No abnormal signal from the bilateral temporal/hippocampus lobes. There was no extra-axial collection. There was no mass effect or midline shift. The paranasal sinuses were clear. The globes and orbits were unremarkable. Hence impression was diffuse bilateral predominantly posterior occipital and parietal subcortical deep white matter changes suggestive of vasogenic oedema without associated restricted diffusion. In the context of a hypertensive episode Posterior Reversible Encephalopathy Syndrome (PRES) was clinically and radiographically the appropriate diagnosis. Her symptoms resolved completed after 3 days of inpatient management of her blood pressure and was discharged home on antihypertensives with instructions to follow up in 4 weeks.

Discussion

With the improvement in imaging modality such as MRI Brain, PRES being a rare disorder now is being reported increasingly. Although it is associated with cytotoxic therapies, dehydration and pre-eclampsia, but uncontrolled hypertension is the most common risk factor.[6] Our patient initially presented with dizziness and vomiting and then she developed diarrhoea which led her to have dehydration. Although she was managed for these symptoms but during the course she developed uncontrolled hypertension which played the main contribution towards developing her neurological symptoms. In her MRI, she was diagnosed with PRES. As she was managed for her uncontrolled hypertension her symptoms resolved completely and was discharged with the proper instructions. The learning point is this case report is to consider PRES as a differential in the workup whenever hypertension is associated with acute neurological symptoms. Prognosis is good considering the reversible nature of its symptoms.[7] The mainstay of treatment is largely symptomatic with strict control of hypertension and prevention of any seizures.[8,9]Therefore, increased awareness should be given to healthcare staff to avoid any permanent damage which could have been prevented during the course of the management of the patients.

Declarations of Conflicts of Interest:
None declared.

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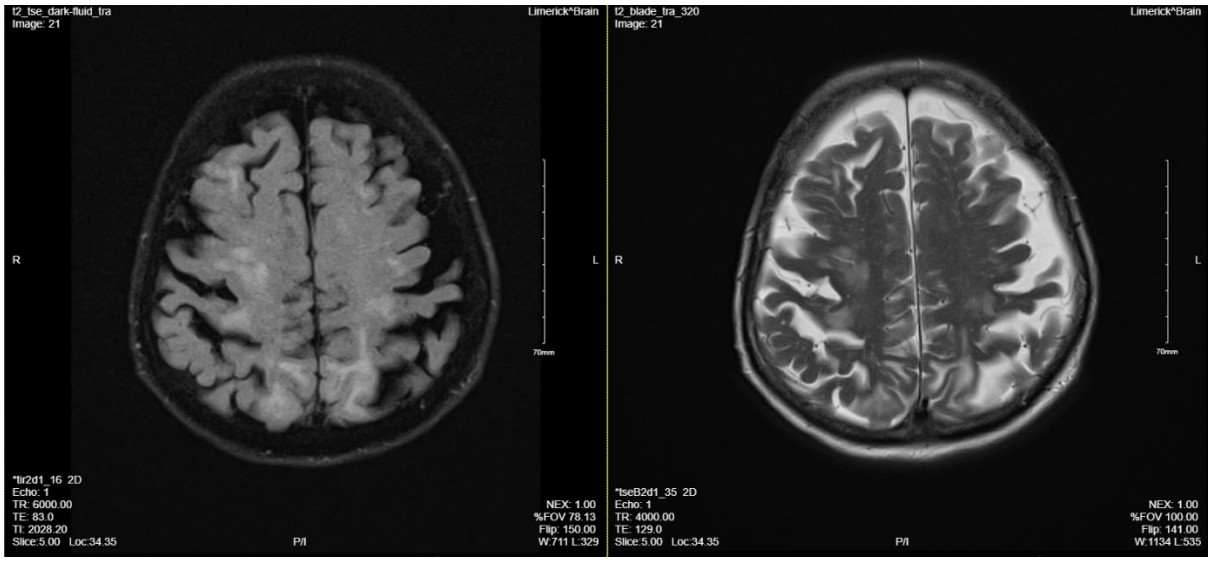
Images:



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