ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
National Library of Medicine - ID: 101731606

International Journal of Medical Science and Innovative Research (IJMSIR)
IJMSIR : A Medical Publication Hub
Available Online at: www.ijmsir.com
Volume – 9, Issue – 1, February – 2024 , Page No. : 67 – 69

Posterior Reversible Encephalopathy Syndrome Following Cardiac Arrest: A Case Report

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Citation this Article: Dr Manjot Kaur, Dr Gurpreet Singh Battu, Dr Harinder Pal Singh, Dr Kolarikal Reva George, "Posterior Reversible Encephalopathy Syndrome Following Cardiac Arrest: A Case Report", IJMSIR - February - 2024, Vol – 9, Issue - 1, P. No. 67 – 69.
Type of Publication: Case Report
Conflicts of Interest: Nil

Abstract

Hincheyetal.in1996 coined the term posterior reversible encephalopathy syndrome (PRES). Lethargy, nausea, seizures and visual impairment are the common clinical presentations of PRES.[1]

Many clinical conditions have been found to be associated with PRES, of which most common are hypertension, sepsis, immunosuppressant's, preeclampsia, eclampsia, shock, infections, cancer chemotherapy, bone marrow and stem cell transplant.[2-5]

Nowadays, PRES is more easily and rapidly recognised due to advancements in brain imaging technology.

Rapid diagnosis and management is crucial to prevent secondary complications that manifest as intracranial bleeding, status epileptic us and cerebral infarction which increase the risk of morbidity and mortality.[6]

Keywords: PRES, Rapid Diagnosis, Cancer.

Case Report

We report a case of 76 year old male, who presented to the emergency department of our hospital in an unconscious condition, following a history of fall at home. He was a known case of hypertension. All the monitors were attached and IV line was secured.

On examination carotid pulse was not palpable and BP was unrecordable, PEA was noted on the monitor. CPR was started immediately according to AHA -ACLS protocol. After about 20 minutes of CPR, return of spontaneous circulation was achieved. The patient was then intubated and shifted to intensive care unit for further management. I on tropic support and mechanical ventilation was started.

12-lead ECG showed anterior wall MI and TROP-T obtained was positive. Bedside ECHO revealed RWMA-hypo kinetic mid basal posterior wall, mid basal lateral wall, severe left ventricular dysfunction with EF:30-35%,mildMR,traceTR, grade 2 diastolic dysfunction. Patient was started on cardiac drugs. After 24 hours of ICU care, no improvement in GCS was noted, CT head was planned to rule out CNS involvement, which revealed no obvious haemorrhagic or focal lesions. Simultaneously, HRCT chest was done in view of

reduced air entry on auscultation, which was suggestive of changes of interstitial lung disease.

On day 2, the patient had generalized tonic-clonic seizures, which got controlled with anti epileptics. On day 3, for further investigation for the etiology of generalized tonic-clonic seizures and low GCS, 3-D MRI of brain was planned, which revealed nearly symmetrical areas of altered signal intensity involving bilateral temporooccipital, parietal lobes and bilateral basal ganglia and thalami suggestive of PRES. The patient's neurological condition remained largely unchanged for 5 days. From day 6, there was gradual improvement in the patient's GCS and eventually full recovery. He was extubated on day 10.

Fifteen days later he was discharged from the ICU in normal neurological condition, but with CAD on cardiac medications.

Discussion

Clinically diagnosing PRES is a challenge for the intense visit due to its non-consistent and brief presentation. The etiologies of PRES have been studied to be hyper tension, nitroglycerine, renal failure with uremic encephalopathy, pre-eclampsia, eclampsia, severe infection, use of immunosuppressant's, blood transfusion, bone marrow and stem cell transplant, chemotherapeutic and novel targeted techniques.[2-5]

PRES is a clinico-radiological entity which is seen as headache, vomiting, altered mental status, seizures, encephalopathy, motor signs and visual disturbances. Imaging may show holohemispheric watershed superior frontal sulcus, dominant parietal/occipital, partial and/or asymmetric PRES.[6]

Few pathophysiological mechanisms have been proposed till date, of which most common appears to berevers able vasospasm and hyper pre fusion resulting from dysfunction of blood brain barrier.

An increase in blood pressure above the cerebral auto regulatory limit causes extravasations of proteins and fluid sin to interstitial space resulting in vasogenic oedema and disrupted microcirculation.[1,8,9] Further, rise in haematocrit results in excessive release of prostaglandins, calcium ions, serotonin, endothelin, nitric oxide causing endothelial dysfunction.[10]

The posterior region of brain is considered more suspectable to impaired auto regulation due to relatively less sympathetic innervations, manifesting as over perfusion and oedema.[11]

Clinical and radiological recovery of neurological symptoms occur within few weeks in many patients without epilepsy.

However, in some cases permanent tissue damage is also reported owing to sepsis, chemotherapy, hypoxemic brain damage or an underlying co-morbidity. Aggressive symptomatic care in ICU is also requires in severe cases. The longer the time taken to control the triggering factors the poorer is the prognosis.

Some patients may show discrepancy between clinical and radiological recovery where resolution of MRI lesions is delayed. [12,13,14]

Conclusion

To conclude, early recognisation and rapid treatment of the causative factor is pivotal to attain reversibility and good prognosis. However, literature also reports permanent neurological damage and mortsality in 15% of patients with PRES.

More research, better knowledge, a high index of suspension and prompt management can decrease morbidity and mortality prompting early recovery.

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