CASE REPORT

A Case of Posterior Reversible Encephalopathy Syndrome Mimicking Stroke

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Abstract
Posterior reversible encephalopathy syndrome (PRES) is characterized by headache, altered sensorium, visual disturbances, and diagnosed by magnetic resonance imaging (MRI). Here we report a case of cisplatin-induced PRES which was mimicking stroke and diagnosed by serial MRI and recovered completely on treatment, emphasizing that early diagnosis, removal of cause, and treatment can prevent the complication.

Keywords: Cisplatin, Magnetic resonance imaging, Posterior reversible encephalopathy syndrome.

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Introduction
Posterior reversible encephalopathy syndrome (PRES) is a clinicoradiological entity characterized by headache, altered mental status, seizures, and visual disturbances and is associated with characteristic reversible lesions on neuroimaging in a severe arterial hypertension setting.1 This condition is usually seen in patients who had received immunosuppressive therapy, eclampsia, hypertension, and autoimmune diseases.2 Typically, PRES involves parieto-occipital lobes and can also involve other cerebral areas, such as brain stem and cerebellum.3 Early recognition and treatment are important to prevent neurologic sequelae. PRES is reversible once the underlying precipitating cause is treated. Here, we present a case of cisplatin induced PRES mimicking stroke with atypical magnetic resonance imaging (MRI) features.

Case Description
A 46-year-old female patient admitted with complaints of repeated vomiting, aphasia, and right upper limb weakness since 12 hours. Patient was operated case of Ca colon 5 months back. She was on chemotherapy and received eighth cycle of cisplatin 8 days back.

On Examination
Vitals stable. Central nervous system (CNS) examination conscious, following verbal commands, aphasia present, no neck stiffness, pupils – B/L 2 mm reacting to light, moving all 4 limbs, right upper limb grip weak, and plantars-flexor. Rest examination was normal. GCS E3 M6 V2 = 11/15.

On investigation: Complete blood count, liver function test, and kidney function test was normal. Computed tomography (CT) of thr brain was normal. Magnetic resonance imaging brain suggestive of restricted diffusion involving bilateral cerebral white matter, including centrum semiovale, corpus callosum, and posterior limb of internal capsule suggestive of chemotherapy induced neurotoxicity (Fig. 1). MRI venography was normal.

Patient was treated with mannitol, dexamethasone, and levetiracetam. The patient responded to the treatment and aphasia recovered. But on next day evening, the patient again had an episode of aphasia and focal convulsions. The patient was treated with fosphenytoin and continued with antiepileptics, mannitol, and steroids. Patient responded well, aphasia recovered. MRI brain done after 2 days showed complete resolution of white matter abnormalities (Fig. 2). Patient’s right upper limb grip and aphasia improved completely, and no further episode of convulsion was observed.

Fig. 1: Magnetic resonance imaging brain DWI image showing restricted diffusion in cerebral white matter in centrum semiovale, corona radiata, corpus callosum and posterior limb of internal capsule

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Discussion

The mechanism by which immunosuppressive and cytotoxic agents cause PRES is the alteration in blood–brain barrier due to direct impairment of endothelium results in extravasation of fluid leading to vasogenic edema.4–6 Many conditions may resemble PRES including ictal or post-ictal state (with or without status epilepticus), infectious encephalitis, vasculitis, cerebral venous sinus thrombosis, and ischemic stroke.7

Chemotherapy agents that can cause PRES are cisplatin, gemcitabine, oxaliplatin, carboplatin, cytarabine, methotrexate, vincristine, bevacizumab, sunitinib, rituximab, and infliximab.7–10 Condition that can cause PRES eclampsia, preeclampsia, autoimmune diseases, and hypertensive crisis.

MRI brain is the first investigation of choice for the diagnosis of PRES. Nonconvulsive status epilepticus should be ruled out by electroencephalogram.

Treatment includes correction of underlying cause of PRES, control of blood pressure, antiedema measure, and antiepileptic drugs. Patient may require withdrawal of chemotherapy or immunosuppressive drugs, cesarean section in case of eclampsia, and other interventions. Risk of ischemia and bleeding can be reduced by treating the underlying precipitating cause.

Conclusion

Although the clinical presentation of PRES is nonspecific, most patients have variable symptoms. High index of suspicion should be kept for patients of PRES having new onset of seizures, altered sensorium, and new onset of neuro deficit with normal CT brain on presentation and presence of strong predisposing factors such as recent chemotherapy, PIH, and uncontrolled hypertension.

MRI is crucial for diagnosing PRES, monitoring the course, and assessing the treatment effectiveness. Repeated cerebral imaging helps to support the diagnosis. Risk of ischemia, bleeding, and death can be prevented by early diagnosis and treatment of underlying cause of PRES.

Our case is important as patient presented with symptoms mimicking of left-sided stroke with initial CT brain normal on presentation and MRI suggestive of atypical findings. MRI should be the first investigation of choice for diagnosing PRES. Early recognition and resolution of the underlying cause lead to complete recovery.

Consent of Patient

The authors certify that they have done all appropriate patient consent formalities. Patient has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names, and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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