ISSN 2515-8260 Volume 10, Issue 1, 2023

A CASE OF CORTICAL BLINDNESS SECONDARY TO PRES

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ABSTRACT

Blindness brought on by an intact anterior visual pathway but due to bilateral occipital lobe involvement is known as cortical blindness. Compared to partial blindness, it is less frequent. The posterior reversible encephalopathy disease has a well-documented history of reversible cortical visual blindness (PRES). The neurological condition PRES is characterised by reversible subcortical vasogenic brain oedema and sudden onset neurological symptoms. This illness can be reversed with prompt diagnosis and treatment. It frequently occurs in conjunction with disorders like eclampsia, cancer, kidney disease, hypertension, and hypertension. This case involves a male teenager who was infected with COVID and later developed PRES. The presence of Anton's blindness complicated PRES. Utilizing

ISSN 2515-8260 Volume 10, Issue 1, 2023

radiological investigation, an early diagnosis was obtained, and once therapy was started, all symptoms disappeared.

Keywords: Cortical blindness, PRES, Hypertension, COVID

INTRODUCTION

There has been growing evidence pointing to the neurological symptoms of the COVID-19 pandemic since it emerged in China [1]. Headache, vertigo, encephalopathy, psychosis, and olfactory and gustatory problems are examples of some of the neurological symptoms. Other neurological complications include meningitis-encephalitis, Guillain-Barré syndrome, acute transverse myelitis and cerebrovascular accidents (CVA) [1, 2]. Latest theories suggest that direct viral invasion or maladaptive inflammatory responses may be the underlying cause for these symptoms [2].

In this report, we highlight a case of SARS-CoV-2 pneumonia coupled with posterior reversible encephalopathy syndrome (PRES), worsened by Anton's cortical blindness. This is one of the few known examples of persistent cortical blindness as a result of COVID-19 pneumonia, and is also the first incidence of persistent cortical blindness in our tertiary medical centre.

CASE REPORT

A 15-year-old male with no known systemic illness was admitted to the hospital, with complaints of sudden onset, throbbing occipital headache and non-projectile, non-bilious vomiting since three days followed by sudden painless loss of vision in both eyes since one day. Within two hours of admission, patient had two episodes of generalised tonic clonic seizures with post ictal confusion lasting for only a few minutes. Patient gives history of high-grade fever since 15 days, intermittent in nature showing no diurnal variation, which reduced on taking Tab Paracetamol. The last febrile episode was 5 days prior to admission.

On examination, the patient’s blood pressure was 180/110 mm Hg. The patient’s Glasgow Coma Scale Score was 15/15. There was no motor or sensory loss in the patient. Impairment of visual acuity was the most notable clinical finding on examination. The patient was walking into objects in the ward confirming that he had no light perception in both eyes. However, patient vehemently denied his blindness suggesting he had Anton Syndrome. Pupillary reflexes were intact, suggesting an intact Anterior visual pathway with unremarkable fundus examination. All other cranial nerve examination was normal and there were no signs of meningeal irritation.

ISSN 2515-8260 Volume 10, Issue 1, 2023

On the basis of clinical signs and symptoms, many differentials such as subdural, intracerebral or subarachnoid haemorrhage, Venous Sinus Thrombosis, Ischaemic Stroke, Meningitis and Posterior reversible encephalopathy syndrome (PRES) were taken into consideration.

To rule out differentials and to reach a diagnosis, laboratory and radiological investigations were performed.

All laboratory investigations were normal. COVID RTPCR was negative. However, patient was positive for COVID antibodies (6061) with IgM level of 5320.

HRCT thorax showed bilateral peripheral basal consolidations/ Ground Glass Opacities suggestive of recent COVID-19 infection with a CT severity score of 5/25.

The T2/FLAIR hyperintensities and accompanying T1WI hypointensities in the cortical and subcortical white matter of bilateral parietal, occipital and cerebellar hemispheres suggestive of vasogenic edema s/o PRES were seen during MRI Brain (Plain + Contrast) with venography.

In this patient, the initial trigger was hypertension secondary to an infection. Based on the CT findings and antibody testing, and after ruling out all other possible causes of hypertension, the infection was determined to be COVID-19.

A diagnosis of Posterior reversible encephalopathy syndrome (PRES) due to hypertension secondary to COVID-19 infection was made and treatment was started accordingly.

Treatment involved

•Inj. Levetiracetam 1gm IV stat f/b 500mg BD

•Inj. Mannitol 100ml IV TDS (tapered)

•Inj. Labetalol 20mg IV stat

•Tab. Cilnidipine 10mg HS

•Tab Azithromycin 500mg OD for 5 days

After initiating treatment, the patient started showing improvement in signs and symptoms on the second day itself. By day 2 of treatment, patient's vision improved from no perception of light to visual acuity of 6/18 in both eyes and blood pressure reduced to 160/100 mm Hg on supine position. On day 3, patient's vision returned completely and visual acuity was 6/6 in both eye. Blood pressure was 140/90 mm Hg. The patient was under observation for 12 days and on the last day of hospital stay, patient's blood pressure was 130/90 mm Hg. The patient was discharged and was asymptomatic on follow-up visits.

ISSN 2515-8260 Volume 10, Issue 1, 2023

DISCUSSION

It has been nearly 25 years since PRES was originally described (3). Headache, nausea, and vomiting, as well as changed mental status, seizures, coma, fluctuating blood pressure, and visual disruption, are all symptoms of PRES [4]. Cortical blindness is the most prevalent visual impairment, but homonymous hemianopia, visual neglect, and fuzzy vision are also common. Surprisingly, the COVID19 pandemic has seen a significant increase in PRES patients. Clinicians can now diagnose this illness using readily available MRI brain imaging. Despite the fact that the specific pathophysiology of PRES is unknown, various pathogenic processes have been proposed. Direct effect of virus and cytokines on the endothelium causing disruption of the blood-brain barrier is one of the proposed theory. Another theory explains endothelial injury as a result of rapid variations in blood pressure (especially hypertension).

Furthermore, by interacting with ACE 2 receptors on endothelial cells of lung and brain parenchyma and brain microglia (5), COVID-19 modifies the Renin Angiotensin Aldosterone System (RAAS) to favour the classical route, causing vasoconstriction and probable alterations in the blood pressure (6).

This could impact the cerebral vasculature directly or indirectly, resulting in PRES (7).

PRES is a rare condition and early detection of its signs and symptoms can help patients to avoid developing long term neurologic disability. Variability in the clinical course of PRES can be seen from day to day, or even hour to hour.

Although there is no specific treatment for PRES, it is assumed that symptoms can be reversed once the underlying cause is addressed (8). Appropriate management of hypertension and related inflammation is commonly regarded to be critical in the treatment of PRES.

Proficiency of radiologist and ophthalmologist with PRES is essential. For proper management and to spare patients from needless testing, ophthalmologists should conduct a thorough clinical examination to assess all other possible causes of visual abnormalities, and radiologists should be able to recognise the distinctive imaging features of this reversible and treatable pathological process. Last but not least, medical professionals treating these patients need to be informed of this reversible but potentially fatal illness.

ISSN 2515-8260 Volume 10, Issue 1, 2023

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