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Case Report

Posterior Reversible Encephalopathy Syndrome: An Unusual Complication of Benzodiazepine Poisoning: A Case Report

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ABSTRACT

Posterior Reversible Encephalopathy Syndrome (PRES), also known as Reversible Posterior Leukoencephalopathy Syndrome, presents with rapid onset symptoms, including headache, seizures, altered consciousness, and visual disturbance. It is seen most frequently in settings of acute hypertension and is usually related to eclampsia. Only a few cases in the literature described PRES syndrome following benzodiazepines. We present a young male with benzodiazepine poisoning brought to the hospital in a deep coma, hypoxia, acidosis, and shock. Diagnosis of PRES was made on history, clinical examination, and radiologic findings of symmetric bilateral hyperintensities on T2 weighted Magnetic Resonance Imaging (MRIs) representing vasogenic edema.

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1. Case presentation

A 21-year-old male presented to the emergency department with a history of intake of syrup containing Dextromethorphan 200 ml and an unknown quantity of Benzodiazepine tablets. The patient was unconscious when brought to the emergency department with nonrecordable blood pressure and constricted pupils. The patient had a pulse rate of 120 per minute, respiratory rate of 30 per minute and body temperature was 98.70 F at presentation. The patient had SpO_2 level of 70% and blood sugar level of 238 mg/dl. The patient had no significant past medical history, there was no history of hypertension, seizure disorder , connective tissue disorder, or exposure to any drugs , patient was not on any immunosuppressive drugs. On examination, respiratory system bilateral air entry was present, and bilateral crept heard all over the lung fields. Central nervous system bilateral pupils non-reactive to light and bilateral plantar mute, with a motor loss with power in all limbs, was zero, GCS score of 3/10. Cardiovascular and per abdominal examinations were unremarkable. Basic laboratory studies, including EKG, were normal (Table 1) other than ABG and Urine screen for drugs and toxins mentioned in Tables 2 and 3.

MRI imaging Figure 1 showed T2 flair/hyperintensity in the bilateral thalami, which were reduced in size. Cerebral hemispheres showed mild diffuse edema with mild flattening of the sulci and gyri. The Cerebral hemispheres also showed new changes in bilateral focal large focal areas of edema in the occipital lobe (more changes on the left side), parietal lobe (more changes on the right side), and frontal lobe. MRI Angiography with TOF showed the spasm of the left posterior cerebral artery beyond the 1st segment and right posterior cerebral artery beyond the 2nd 3rd segments. MRI findings showed mucosal thickening of the bilateral maxillary sinus (left more than right). Right-sided deviation of the nasal septum Mild hypertrophy of the bilateral nasal turbinated was also found (left more than right).

2. Treatment

The patient was given treatment with Fumezenil 0.2 mg intravenously stat dose, Methylprednisolone 40 mg intravenously every 12 hours. The patient was given ventilatory and ionotropic support with Adrenaline at 8 ml/hour, Noradrenaline at 10 ml/hour, and Dopamine at 5 ml/hour. Antibiotics Meropenem 1gm IV every 8 hours and Clindamycin 600 mg IV every 8 hours were given for sepsis cover. Neurotrophic agent ceravate (cerebroprotien hydrolysate) 60 mg IV every 12 hours given intravenously. Prophylactically antiepileptic Levetiracetam 500 mg IV every 8 hours. Supportive treatment with paracetamol, nebulization, pruning, multivitamins, and physiotherapy. Tracheostomy was done because of prolonged ventilatory support. GCS improved from 3 to 10, and power in the upper and lower limbs improved. The patient was discharged after one month with a tracheostomy tube, followed up after a week, followed by 15 days, and slowly weaned the tube with vocal physiotherapy. The patient improved in a span of two months.

3. Discussion

Posterior Reversible Encephalopathy Syndrome (PRES) was first described by Hinchey et al. in 1996

Table 1. Complete Blood Count (CBC), Liver Function Test (LFT), Renal Function Test (RFT)

CBC	Hb	TLC	Platelet Count	DLC	PCV
LFT	12.70	18,000	1.40Lac	N93,L4,B0,E0,M3	40
	T.BIL	DIRECT BIL	INDIRECT BIL	SGOT/SGPT	ALP
RFT	1.25	0.35	0.9	48/37	32
	Urea	S.Creat	Uric acid	Na+	K+
	51.30	1	3.2	145	3.5

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Table 2. Arterial blood gas analysis

pH	HCO3	PCO2	O_2	Lactate
7.1	13	78	60	4.4

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Table 3. Toxicological analysis

Urine toxicological analysis for drugs and toxins	Positive for Benzodiazepine, Negative for Cocaine, Phencyclidine, Opiates, Amphetamines, and Barbiturates

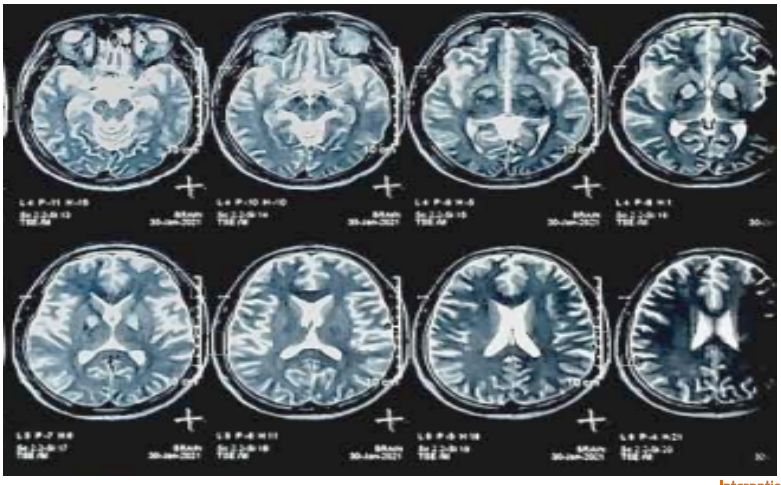

Figure 1. T2 flair/hyperintensity in the bilateral thalami

[1]. It is characterized by a milieu of seizure activity, impaired consciousness, headaches, visual symptoms, nausea/vomiting, and focal neurological signs [2]. PRES is often related to various conditions, all of which end in cerebral vasogenic edema, which seems to be the crucial pathogenic mechanism [3, 4]. As the name suggests, it's typically reversible once the underlying cause is removed. The exact incidence of PRES is unknown. Patients undergoing calcineurin inhibitor therapy with renal transplantation develop PRES syndrome in about 4%-8% of cases [5, 6].

The term PRES has supported the similarity in an appearance on imaging, the specific location of the parietaloccipital lobe, or the "posterior" location of the lesions. The exact pathophysiological mechanism of PRES remains unclear. Three hypotheses have been proposed till now, which include 1) cerebral vasoconstriction causing subsequent infarcts within the brain, 2) failure of cerebral autoregulation with vasogenic edema, and 3) endothelial damage with barrier disruption further resulting in fluid and protein transudation within the brain. The distinct imaging patterns in PRES are Holohemispheric watershed, Superior frontal sulcus, Dominant parietal/occipital, and Partial and/or asymmetric PRES. The reversible nature of PRES has been challenged recently, supported by new reports of permanent neurological impairment and mortality reaching 15% [7].

Among the reported cases, common ones include hypertensive emergency, renal disease, preeclampsia/eclampsia, and immunosuppressive agents [8]. Other reported causes include sepsis, autoimmune diseases such as systemic lupus erythematosus, systemic sclerosis, tumor lysis syndrome, GuillainBarres syndrome, AIDS, thrombotic thrombocytopenic purpura, and acute intermittent porphyria [9, 10]. PRES associated with late postpartum eclampsia has been reported before [9, 11, 14].

4. Conclusion

As indicated by its name, appropriate treatment is predicted to ensure a full recovery. However, permanent complications and fatalities are reported. Recurrence of symptoms has been observed in very few cases. Management of such patients depends mainly on the underlying pathology; early diagnosis and treatment of the underlying condition have better outcomes.

Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this research.

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Authors' contributions

All authors equally contributed to preparing this article.

Conflict of interest

The authors declared no conflict of interest.

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