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PRACTICAL PEARL

Lysergic Acid Amide-Induced Posterior Reversible Encephalopathy Syndrome with Status Epilepticus

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Abstract
Introduction Posterior reversible encephalopathy syndrome (PRES) is known to occur in association with several substances. However, lysergic acid amide (LSA) is not among the previously reported causes of PRES.
Methods We report on a patient with PRES presenting as convulsive status epilepticus associated with hypertensive encephalopathy after LSA ingestion. Magnetic resonance imaging was performed and catecholamine metabolites assayed.
Results The patient achieved a full recovery after aggressive antihypertensive therapy and intravenous anticonvulsivant therapy. The clinical history, blood and urinary catecholamine levels, and response to treatment strongly suggest that PRES was induced by LSA.
Conclusion LSA, a hallucinogenic agent chiefly used for recreational purposes, should be added to the list of causes of PRES.

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Keywords Status epilepticus · Posterior reversible encephalopathy syndrome · Lysergic acid amide · MRI · Intoxication · Hypertensive encephalopathy

Introduction

Posterior reversible encephalopathy syndrome (PRES), first described by Hinchey et al. in 1996 [1] has been reported in association with several substances [2]. We report on a patient who presented with convulsive status epilepticus (SE) due to PRES complicating hypertensive encephalopathy. Ingestion of lysergic acid amide (LSA) was determined to have caused the hypertensive episode. To our knowledge, this case is the first report of PRES associated with LSA.

Case Report

A 39-year-old man, with a history of chronic alcohol abuse, smoking, depression treated with clomipramine 75 mg/day for the last 6 months, and seizure 3 years earlier experienced a witnessed, generalized, tonic-clonic seizure at home. A second seizure occurred during transportation to the hospital by a mobile emergency medical unit. Upon arrival at the emergency room, he was in a state of altered consciousness with a Glasgow Coma Scale of 11, mental confusion, hyperreflexia, mydriasis, and diaphoresis. Seizure activity spontaneously stopped. His body temperature was 38.4°C, blood pressure 185/130 mm Hg, and heart rate 120 beats/min. He was admitted to the intensive care unit after a third seizure occurrence and receiving an intravenous clonazepam bolus that failed to stop the tonic-clonic movements, which lasted more than 10 min. He was

promptly intubated and mechanically ventilated. The seizures stopped after another intravenous clonazepam bolus combined with a 20-min phenobarbital infusion. Laboratory tests showed no metabolic disturbances. Standard toxicology screening tests on blood and urine were negative, except for clomipramine; they included tests for ethanol, cocaine, opiates, and benzodiazepines. Clomipramine was not assayed quantitatively. Cerebrospinal fluid analysis and cerebral computed tomography were normal. No cause to the status epilepticus or hypertension was identified. Blood pressure remained greater than 185/120 mm Hg despite intravenous nicardipine up to 6 mg/h and oral acebutolol 400 mg/day. Magnetic resonance imaging (MRI) 24 h after the end of the seizure showed extensive bilateral high signal predominating in the parietooccipital and posterior-fossa white matter. The signal abnormality was best seen on the fluid-attenuated inversion recovery (FLAIR) sequences (Fig. 1). High signal from the right thalamus was noted on FLAIR sequences (Fig. 1a). Three days after admission, the patient was successfully extubated, and the physical examination showed mental confusion, headache, and visual hallucinations. Blood pressure values greater than 180/120 mm Hg prompted decreases in intravenous nicardipine (up to 8 mg/h) and oral acetabutolol (800 mg/day) dosages; finally, blood pressure stabilized at about 120/80 mm Hg. He then regained full consciousness without psychiatric disorders or further seizures. Seven days later, a repeat MRI was normal (Fig. 2). We gave a diagnosis of PRES with status epilepticus caused by hypertensive encephalopathy.

Tests to identify the cause of the malignant hypertension showed a massive sympathetic storm with marked and sustained increases in two specific metabolites of catechol-O-methyltransferase (COMT), normetanephrine, and 3-methoxytyramine (3-MT), measured in urine (Tables 1 and 2). These results indicated strong and simultaneous stimulation of the norepinephrine, dopamine, and serotonin systems associated with extremely high COMT activity, most notably in the norepinephrine (normetanephrine) and dopamine (3-MT) pathways. Blockade of monoamine oxidase (MAO)-A and B activities led to high serotonin (5 hydroxy-tryptamine or 5-HT) levels contrasting with normal urinary 5-hydroxy indoleacetic acid (5-HIAA) levels, high normetanephrine levels with low urinary vanilmandelic acid (VMA) levels, and high 3-MT levels with normal urinary homovanillic acid (HVA) levels. The profile of catecholamine, MAO-metabolite, and COMT-metabolite levels was not consistent with pheochromocytoma [3], for which imaging studies were negative. Plasma levels of renin, aldosterone, and cortisol were within the normal

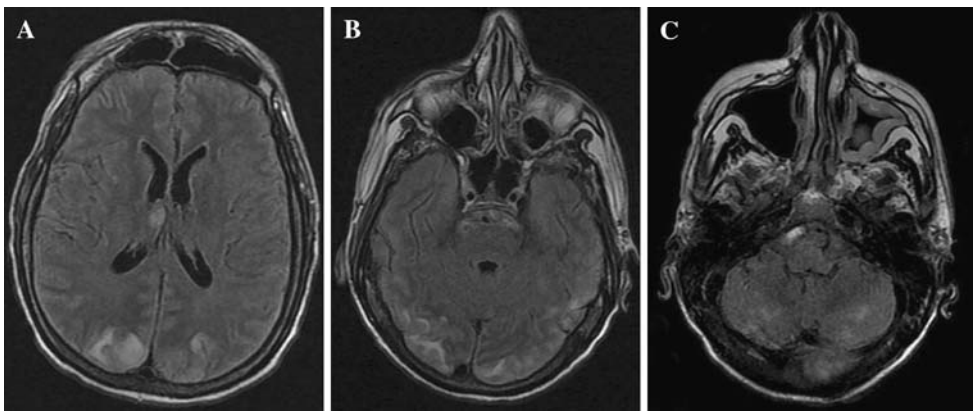

Fig. 1 Magnetic resonance imaging 1 day after status epilepticus: extensive bilateral foci of high signal in the white matter, predominating in the parietooccipital lobes (**a** and **b**) and posterior fossa (**c**) and best demonstrated on fluid-attenuated inversion recovery sequences

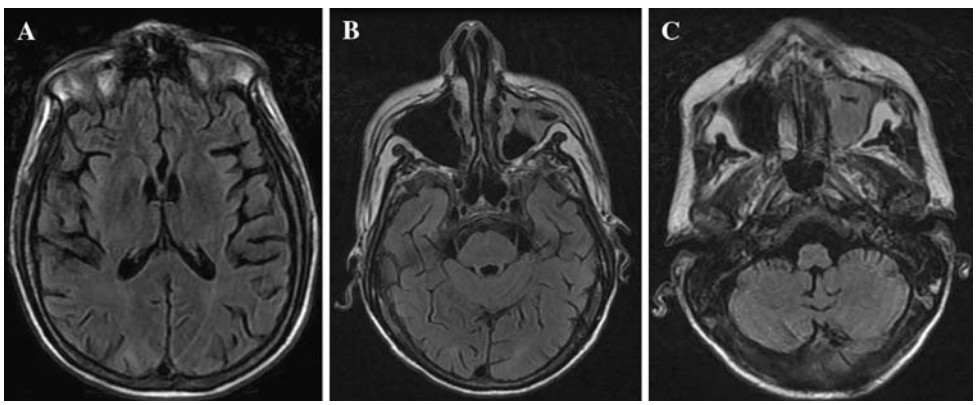

Fig. 2 Magnetic resonance imaging 7 days after status epilepticus: normal signal from the parietooccipital lobes (**a** and **b**) and posterior fossa (**c**)

Table 1 Urinary catecholamine metabolite concentrations during the ICU stay

	Admission	Day 6	Day 11	Day 12	Day 14	Normal value
Normetanephrine[a]	1048	1249	1091	1024	966	40–275
Metanephrine[a]	246	109	79	71	78	15–120
3-Methoxytyramine (3-MT) or 3-O methyl dopamine (3-OMD)[a]	895	575	450	471	428	20–190
Vanilmandelic acid (VMA)[b]	0.36	–	–	–	–	2–3
Homovanillic acid (HVA)[b]	3.1	–	–	–	–	2–4

[a] Concentration in nmol/mmol creatinine

[b] Concentration in μmol/mmol creatinine

Table 2 Platelet and urinary concentrations of serotonin and its metabolite 5-hydroxy indoleacetic acid at ICU admission

	Admission	Normal value
Platelet serotonin (5 hydroxy-tryptamine; 5-HT)[a]	0.03	0.25–1.4
Urinary serotonin (5 hydroxy-tryptamine; 5-HT)[b]	85.6	3–55
Urinary 5-hydroxy indoleacetic acid (5-HIAA)[a]	2.1	0.7–3.6

[a] Concentration in μmol/mmol creatinine

[b] Concentration in nmol/mmol creatinine

ranges. Urinary porphyrins and their precursors were normal. We found none of the disorders known to cause hypertensive encephalopathy (e.g., renal parenchymal or vascular disease, autonomic hyper-reactivity, or idiopathic hypertension) [4]. When the patient was sufficiently recovered for an in-depth interview, he reported taking LSA immediately before the beginning of the seizure. LSA is an analogue of lysergic acid diethylamide (LSD) derived from plants. The known ability of LSA to cause hypertension [5], chronological link between LSA use and seizure onset, and negative findings from extensive investigations for other causes of hypertension strongly support a causal link between LSA and hypertension in our patient.

The patient was discharged after 9 days in the ICU. His blood pressure was normal with oral acebutolol 400 mg/day. The neurological evaluation was normal. The patient later stopped his acebutolol treatment when his blood pressure was found normal during follow-up visits.

Discussion

PRES is a clinical and neuroimaging entity characterized by acute neurologic disorders (including altered mental status, seizure or status epilepticus [6], headache, visual loss, nausea, and vomiting), associated with characteristic brain imaging abnormalities that are best identified by MRI [1]. The typical MRI pattern consists of bilateral symmetric signal abnormalities from the subcortical white matter of the parietooccipital lobes [7]. Involvement of the overlying cortex is seen occasionally. Less often, the frontal lobes, temporal lobes, or posterior fossa are affected. T2-weighted and FLAIR sequences show high signal, whereas diffusion-weighted imaging may be normal. The apparent diffusion coefficient (ADC) is increased when the lesions are reversible but decreased when irreversible infarction occurs [7]. Reversible postictal edema, the main differential MRI diagnosis, typically manifests as high signal in the involved cortex on diffusion-weighted images, whereas T2 images may be normal; furthermore, the ADC is consistently decreased in the hippocampal formation and/or pulvinar nucleus of the thalamus and/or involved cortex [8]. However, postictal edema may explain the unilateral high-signal focus in the right thalamus shown in our patient by FLAIR sequences (Fig. 1a).

The main causes of PRES are hypertensive encephalopathy, eclampsia, thrombotic microangiopathies, immunosuppressants, and several other drugs (Table 3) [1, 2, 6, 9–54]. The pathophysiology of PRES is incompletely understood. Impaired cerebral autoregulation responsible for increased blood flow is one of the two main hypotheses, the other being endothelial dysfunction, most notably in cases related to cytotoxic therapy. Both mechanisms result in blood-brain barrier dysfunction with vasogenic edema [1]. Another hypothesis involves vasospasm leading to cytotoxicity, which in turn may cause extracellular edema [55].

The immediate treatment of PRES includes aggressive management of the hypertensive encephalopathy and administration of anticonvulsant drugs adapted to the severity of the seizure. In the absence of prompt treatment followed by a rapid response, irreversible brain damage with functional impairment or death may occur [2].

LSA, also known as ergine, is an alkaloid of the ergoline family. Structurally, LSA closely resembles the well-known synthetic hallucinogen lysergic acid diethylamide (LSD), which is a chemical derivative of lysergic acid found in the rye fungus ergot and having an indole structure similar to that of serotonin. LSA is found in varying concentrations in the seeds of several plants such as *Argyreia nervosa* (Hawaiian baby woodrose), *Ipomoea*

Table 3 Agents associated with posterior reversible encephalopathy syndrome

Chemotherapeutic agents (in combination) [14, 25, 44, 46]
Cytotoxic agents
Alkylating agents
Cisplatin [20, 48]
Oxaliplatin [15, 35]
Carboplatin [37]
Anti-metabolites
Gemcitabine [30, 34]
Cytarabin [39]
Mitotic inhibitors
Vincristine [51]
Irinotecan hydrochloride [38]
Others
L-asparaginase [43]
Anti angiogenic therapy
Bevacizumab [19, 38, 40]
Sunitinib [36]
RAF kinase inhibitor BAY 43-9006 [16]
Immunomodulatory cytokine
Interferon-alfa [1, 52]
Interleukin-2 [53]
Anti CD 20 monoclonal antibodies
Rituximab [23]
Intravenous immunoglobulin [10, 22, 31, 41, 64]
Anti-lymphocyte globulin [17]
Immunosuppressive agents
Cyclosporine A [1, 6, 9, 11, 21, 27, 28, 47]
Tacrolimus (FK 506) [1, 9, 26, 42]
Sirolomus [65]
High dose steroid therapy (eg. dexamethasone,methylprednisolone) [12, 24, 32]
Blood transfusion [49]
Others agents
Granulocyte stimulating factor [10, 54]
Antiretroviral therapy [29, 33]
Linezolid [13]
Erythropoietin therapy [50, 52]
Cocaine abuse [45]
Ephedra plant *Ephedra sinica* (traditional Chinese medicine) [18]

violacea (morning glory), *Rivea corymbosa* (ololiuhqui), and *Stipa robusta* (sleepygrass). The seeds can be chewed then swallowed, crushed and soaked for a few hours in cold purified water or alcohol then filtered and drunk, or left in water for a few days then eaten. LSA can also be extracted from the seeds as a paste after treatment with various agents such as ether and oil. Ingestion of only 2–5 mg of LSA produces hallucinations that last 4–8 h. LSA is less powerful than LSD, which is hallucinogenic in a dose of about 200 μg [5].

The mechanisms of action of LSA seem related to agonist, partial agonist, and antagonist effects on serotonin, dopaminergic, and adrenergic receptors. The hallucinogenic properties of LSA, as well as of LSD, were recently ascribed to agonist effects on serotoninergic 5-HT2 A [56, 57] and dopaminergic DA2 receptors [58]. In addition, the hallucinogenic effect may be related to overproduction of O-methylated catecholamine catabolites, most notably 3-MT, which is the major dopamine metabolite produced by the enzyme COMT [59]. A genetically determined COMT variant characterized by increased enzyme activity was recently found to be associated with schizophrenia [60]. O-methylated catabolites may also contribute to block MAO enzymes. Interestingly, our patient exhibited marked increases in urinary 3-MT and normetanephrine, possibly caused by COMT overactivity in response to massive catecholamine overproduction induced by LSA and exacerbated by MAO A and B blockade.

In our patient, PRES was probably the result of marked LSA-induced stimulation of noradrenaline, 5-HT, and dopamine production with severe vasospasm responsible for hypertension. Noradrenaline and 5-HT exert direct vasoconstricting effects; with 5-HT, these effects vary across vascular territories and vessel diameters. 5-HT induces contraction of vascular smooth muscle cells, as well as increased capillary permeability, via effects mediated by 5-HT2A receptors [57]. In addition, high levels of 5-HT exert proinflammatory effects [61]. High levels of dopamine (reflected in our patient by the high 3-MT) cause vasoconstriction via α-adrenergic stimulation [58]. All these effects may have contributed to induce blood-brain barrier dysfunction responsible for the vasogenic and extracellular edema that characterize the hypertensive encephalopathy seen in PRES. The exact contribution of each neurotransmitter remains unclear.

Importantly, the cerebral and peripheral vascular abnormalities in our patient may have been exacerbated by blockade of two major pathways for inactivation of 5HT and norepinephrine. Clomipramine causes about 80% to 90% inhibition of 5-HT transporter-mediated serotonin reuptake by neurons and blood platelets [62], and its main metabolite desmethylclomipramine inhibits norepinephrine reuptake by neurons [63]. Furthermore, the MAO-A and B blockade in our patient was too severe to be ascribable to smoking alone, suggesting MAO-inhibiting effects of LSA [58]. These two mechanisms of monoamine inactivation blockade may have considerably magnified the 5-HT- and norepinephrine-mediated cardiovascular and neuropsychiatric effects of LSA. Furthermore, our patient experienced protracted hallucinations (3 days) and hypertension (1 month), concomitantly with sustained increases in urinary 3-MT and normetanephrine levels.

Conclusion

The data from our patient strongly suggest a diagnosis of PRES induced by LSA. This agent, which is mainly used as a recreational hallucinogenic drug, deserves to be added to the list of causes of PRES.

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