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Acute Ischemic Leukoencephalopathy Secondary to Circulatory Shock and Metabolic Acidosis

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INTRODUCTION

- Acute leukoencephalopathy remains a challenging diagnosis due to a myriad of etiological factors and varying clinical presentations.
- Acute ischemic leukoencephalopathy secondary to circulatory shock and metabolic acidosis is rarely reported, with sparse data on the cognitive and functional outcomes.
- Aggressive treatment should be pursued, despite persistent neuroimaging findings of acute ischemic leukoencephalopathy, in view of possible complete clinical resolution.

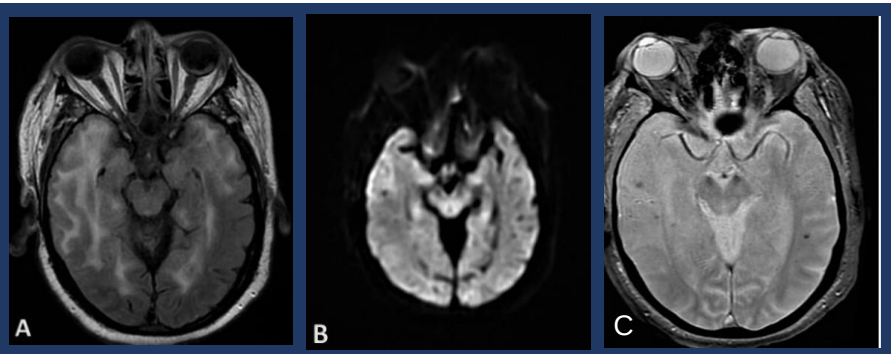
CASE DISCUSSION

A 74-year-old Hispanic male with a past medical history of hypertension, lumbar spondylosis, and bipolar disorder type 1, was brought to the emergency room by his wife after an intentional overdose of prescribed medications, including 50 tablets of combination amlodipine-benazepril, 30 tablets of carvedilol, and 15-20 tablets of ibuprofen.

Vital signs revealed a rapid decline in blood pressure from 115/56 to 54/25 mmHg, with a mean arterial pressure 35 mmHg, heart rate 67 beats/min, respiratory rate 15 breaths/min, and temperature 97.7 F. Pinpoint pupils with sluggish response and positive doll's eye reflex were found on routine eye examination. The Glasgow coma scale deteriorated from 14 to 7, requiring emergent orotracheal intubation for airway protection. The patient was started on fluid resuscitation with normal saline (3 L), vasopressor infusions including dopamine infusion (20 mcg/kg/min), neosynephrine (20 mcg/min), norepinephrine (30 mcg/min), and intermittent epinephrine (300 mcg/hr).

The patient was admitted to the intensive care unit for close neuro-hemodynamic monitoring. An electrocardiogram revealed bradycardia, sinus rhythm, and left ventricular hypertrophy. Complete metabolic panel revealed blood urea nitrogen 30 mg/dL, creatinine 2.03 mg/dL, lactate 3.14 mmol/L. Arterial blood gas showed pH 7.05, pCO_2 67 mmHg, and bicarbonate 18 mEq/mL. Brain magnetic resonance imaging (MRI) fluid-attenuated inversion recovery (FLAIR) sequences revealed T2-weighted white matter hyperintensities in the bilateral temporal lobes (right > left). Electroencephalogram revealed poorly-organised theta activity with intermixed triphasic waves. Transthoracic echocardiogram showed a preserved ejection fraction of 55-59%.

With gradual weaning of sedation and hemodynamic support, his cognitive function improved, resulting in extubation on day six. His cranial nerve and sensorimotor examinations did not reveal any deficits. Brain MRI on day nineteen continued to show changes consistent with acute ischemic leukoencephalopathy. Repeat neuroimaging with MRI was advised in four weeks, however, the patient was lost to follow up.



(A) *Brain MRI FLAIR sequences revealed prominent white matter hyperintensities in the temporal lobe, right greater than the left. These findings suggest right greater than left temporal leukoencephalopathy with scattered foci of hemosiderin.* ***(B)*** *Brain MRI diffusion-weighted imaging (DWI) sequencing showed no evidence of restricted diffusion.* ***(C)*** *Gradient recalled echo (GRE) sequence revealed multiple small foci of hemosiderin in the bilateral temporal lobes.*

DISCUSSION

- Acute ischemic leukoencephalopathy could specifically be related to oligodendroglial and axonal injury, occurring independently of soma ischemic injury [1]. This is likely compounded by the widely dispersed arterioles in the white matter [1].
- Another hypothesised etiology is the susceptibility of GABAergic neurons to anoxic insult [1,2]. In the white matter, GABAergic neurons form connections between the neuron and the neuroglial-2 precursor derived oligodendroglia, providing neuroprotection from acute anoxia [2].
- Severe acidemia, as seen in our patient, did result in cardiovascular collapse secondary to decreased peripheral vascular resistance, decreased cardiac output, and increased resistance to catecholamines [3]. Metabolic acidosis also increases oxygen binding to hemoglobin, thus, in our patient, could have led to an intensified ischemic insult [3].
- Hypercapnia, in the presence of hypoxemia, has been hypothesized to exacerbate weakening of the blood brain barrier [4].
- In our case, we propose that the ischemic hypoperfusion, along with a compromised blood-brain barrier, resulted in white matter edema, that presented as acute ischemic leukoencephalopathy [1,5].

CONCLUSION

Acute ischemic leukoencephalopathy, though rare, can occur secondary to hemodynamic instability resulting in circulatory shock, and concurrent metabolic abnormalities. Interestingly, the patient experienced clinical resolution despite persistent white matter lesions on neuroimaging. This case suggests physicians should continue aggressive therapeutic intervention, despite findings of acute ischemic leukoencephalopathy.

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DISCLOSURES

The authors report no disclosures.

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