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BILATERAL THALAMIC INFARCTS LEADING TO THERMOREGULATORY DYSFUNCTION

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Abstract

Bilateral thalamic infarctions are rare cerebrovascular events that disrupt critical neural networks involved in sensory integration, motor regulation, and autonomic stability. Among these, thermoregulatory dysfunction is a complex and often under-recognized complication due to the involvement of thalamic pathways that relay information to the hypothalamus, the body's primary thermoregulatory center. This case highlights a 55-year-old male presenting with neurological deficits and persistent fever unresponsive to conventional antipyretics or antibiotics. Neuroimaging revealed bilateral infarctions in the occipital lobes, cerebellum, thalamus, and right hemipons. The absence of systemic infection and the clinical response to bromocriptine, a dopamine agonist, confirmed the diagnosis of hyperthermia secondary to thalamic infarction. Central hyperthermia following thalamic infarction poses diagnostic and therapeutic challenges due to its nonspecific presentation and resistance to routine fever management. In this case, bromocriptine effectively reduced fever spikes by modulating hypothalamic dopamine receptors, illustrating its therapeutic potential in similar cases. This report highlights the importance of advanced imaging modalities for accurate localization of lesions and a multidisciplinary approach for effective management. Further research into targeted therapies for thalamic infarction-induced dysautonomia is crucial. Timely recognition and intervention are essential to prevent complications and improve patient outcomes in these rare but impactful cases.

INTRODUCTION

The thalamus, a crucial relay center within the central nervous system, plays an essential role in integrating sensory and motor signals. It also modulates autonomic and regulatory functions, including thermoregulation, through its connections with the hypothalamus and other brain regions (1). Bilateral thalamic infarcts are rare cerebrovascular events that disrupt these vital networks, leading to profound neurological and autonomic dysfunctions. Among these, thermoregulatory dysfunction represents a particularly complex manifestation due to the interplay of neural circuits governing body temperature homeostasis (2). Thermoregulatory dysfunction arising from bilateral thalamic damage highlights the significance of thalamic contributions to autonomic stability. The intricate neural pathways disrupted in such infarcts often result in a constellation of symptoms, including hyperthermia, hypothermia, or fluctuating temperature regulation (3). These conditions present significant diagnostic and therapeutic challenges, underscoring the need for a deeper understanding of these infarcts' pathophysiology and clinical implications (4).

Bilateral thalamic infarctions typically result from occlusions of the paramedian thalamic arteries, often involving the artery of Percheron—a rare anatomical variant supplying both thalami. These infarcts are frequently associated with deficits in consciousness, memory, and sensory processing, reflecting the thalamus's integrative functions (5). The addition of autonomic disturbances, particularly thermoregulatory dysfunction, further complicates the clinical picture, suggesting broader involvement of thalamic and hypothalamic connections (6). Research has demonstrated the pivotal role of the thalamus and hypothalamus in thermoregulation. The hypothalamus acts as a primary thermoregulatory center, receiving and processing input from peripheral and central thermoreceptors through thalamic relays (7,8). Damage to these connections can lead to failure in maintaining thermal homeostasis, manifesting as either hyperthermia or hypothermia, depending on the lesion's extent and location (3,8).

Reports of thermoregulatory dysfunction in bilateral thalamic infarcts often emphasize the involvement of posterior hypothalamic regions, which are critical for heat production and conservation (1). Disruption of these pathways has been implicated in several neurological conditions, including stroke and traumatic brain injury (4,6). Moreover, neuroimaging studies have shed light on the specific structural and functional alterations associated with such infarcts, offering insights into their pathophysiology (2).

Understanding the neural substrates of thermoregulation and their disruption in bilateral thalamic infarcts is essential for developing targeted diagnostic and therapeutic approaches. Such cases provide a window into the thalamus's multifaceted role and highlight the broader implications of autonomic dysfunction in cerebrovascular diseases (7).

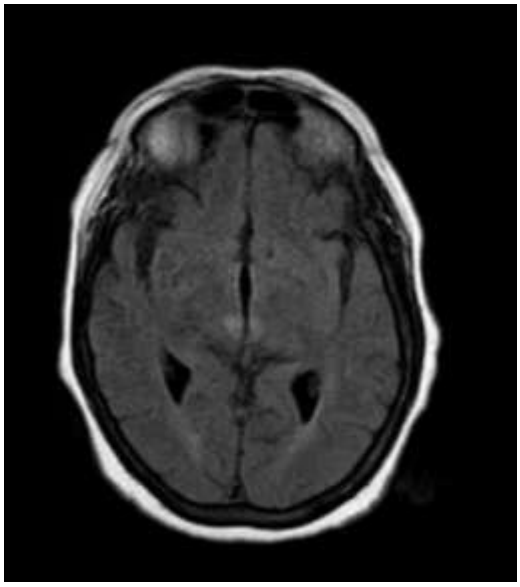
CASE DETAILS

A 55-year-old male with a known history of systemic hypertension (diagnosed four years ago and on irregular medication) and chronic alcohol use (last binge one month prior) presented with complaints of weakness in the right upper and lower limbs, slurred speech, and deviation of the mouth to the left, all of which began two days before admission. He also reported a two-day history of giddiness, headache, blurred vision, and diplopia. There was no history of loss of consciousness, neck stiffness, fever, chest pain, or palpitations.

On examination, the patient was conscious, oriented, and afebrile. His vital signs were stable with a blood pressure of 140/80 mmHg, pulse rate of 100/min, respiratory rate of 18/min, and oxygen saturation of 98% on room air. Capillary blood glucose was 110 mg/dL. Systemic examination revealed no cardiovascular, respiratory, or abdominal abnormalities. Neurological evaluation showed right-sided hypertonia, significantly reduced power (2/5) in all four limbs, and exaggerated reflexes in the biceps, triceps, knee, and ankle bilaterally. Plantar reflexes were extensor bilaterally, and pupils were sluggishly reactive to light. The Glasgow Coma Scale (GCS) score was 14/15 (E4V5M5). An brain MRI demonstrated bilateral infarcts in the occipital lobes, cerebellum, thalamus, and right hemipons.

On the fourth day of admission, the patient developed high-grade fever (103°F) without associated chills or rigors. He denied symptoms of burning micturition, cough, abdominal pain, vomiting, or loose stools. The fever persisted Despite initiating intravenous antibiotics, antipyretics, and cold saline infusions. Investigations revealed a total leukocyte count of 13,000/mm^3 with neutrophilia (70%), lymphocytes (30%), CRP of 12 mg/L, ESR of 35 mm/hr, and a procalcitonin level of 0.25 ng/mL. Urine analysis and cultures from blood and urine showed no growth. Imaging, including a chest X-ray and ultrasound of the abdomen, was unremarkable.

A repeat MRI of the brain confirmed the presence of bilateral infarcts in the occipital lobes, cerebellum, thalamus, and right hemipons. Based on these findings and the lack of infection-related etiology, the patient was diagnosed with hyperthermia secondary to thalamic infarction. Treatment with bromocriptine was initiated, leading to a reduction in fever spikes.



DISCUSSION

Thalamic infarctions, particularly bilateral ones, are rare and often involve the paramedian thalamic arteries supplied by the artery of Percheron. The thalamus, an integral diencephalon component, is a relay station for sensory, motor, and autonomic signals. Its role extends to regulating homeostasis, including thermoregulation, through connections with the hypothalamus and brainstem. Damage to the thalamus disrupts these connections, which can result in dysautonomia, including hyperthermia.

The hypothalamus is recognized as the primary thermoregulatory center, with the anterior hypothalamus promoting heat dissipation and the posterior hypothalamus facilitating heat conservation and production. The thalamus acts as a relay, transmitting sensory input regarding body temperature from the periphery to the hypothalamus. Infarction in the thalamic region can impair this feedback loop, leading to unregulated heat production or retention, manifesting as hyperthermia. Such disruptions are associated with inflammatory responses and oxidative stress, further exacerbating neuronal injury (1,3).

Central hyperthermia, resulting from thalamic or hypothalamic damage, is a rare but serious complication of cerebrovascular accidents. Patients often present with unrelenting fever that is unresponsive to conventional antipyretics or antibiotics. In this case, the patient demonstrated persistent fever spikes despite antibiotic therapy, with no evidence of systemic infection, supporting the diagnosis of hyperthermia secondary to thalamic infarction. Neuroimaging plays a critical role in diagnosing thalamic infarcts and associated complications. Magnetic resonance imaging (MRI) can reveal ischemic changes in the thalamus, cerebellum, or other involved regions, as observed in this case. Additionally, advanced imaging techniques, such as diffusion tensor imaging, may provide insights into the disrupted neural pathways associated with thermoregulatory dysfunction (9,10). Management of central hyperthermia focuses on addressing the underlying cause, such as thalamic infarction, while mitigating the hyperthermic state. Bromocriptine, a dopamine agonist, has effectively reduced fever by modulating hypothalamic dopamine receptors. This patient's clinical improvement following bromocriptine therapy highlights its role in managing central hyperthermia. Supportive measures, including cooling techniques and fluid resuscitation, are essential in preventing complications such as rhabdomyolysis, renal failure, and further neuronal damage (11,3).

Bilateral thalamic infarctions carry a poor prognosis due to their association with significant neurological and autonomic dysfunction. Long-term sequelae can include persistent dysautonomia, cognitive deficits, and motor impairments. Hyperthermia, if not managed promptly, can exacerbate neuronal injury and lead to poor outcomes. Multidisciplinary care involving neurologists, intensivists, and rehabilitation specialists is crucial for optimizing recovery (12,13).

Alemdar (2012) described a bilateral mesencephalothalamic infarction where hyperthermia was a prominent symptom. The patient had persistent fever unresponsive to conventional treatment, attributed to damage in

thermoregulatory pathways involving the thalamus and hypothalamus. This report emphasized the importance of recognizing central hyperthermia in cases of thalamic infarction for timely diagnosis and management. (1)

Sung et al. (2009) detailed cases where acute thalamic infarction resulted in central hyperthermia due to disrupted connections between the thalamus and hypothalamus. They noted that the fever resolved only with targeted pharmacological therapy, including dopamine agonists. (9) Bazille et al. (2005) highlighted cases of heatstroke-induced brain injury where the thalamic nuclei were particularly vulnerable. Although the mechanism involved hyperthermia from external factors, it drew parallels to central causes due to the thalamus's sensitivity to temperature dysregulation. (3)

Kiyatkin (2019) reviewed cases demonstrating brain hyperthermia due to ischemic lesions, including thalamic and hypothalamic involvement. The report suggested that even minor infarcts in these areas could disrupt neural thermoregulation pathways, leading to persistent hyperthermia. (13) Walter and Carraretto (2016) presented cases of persistent fever after stroke linked to thalamic damage. The study demonstrated how pharmacologic interventions like bromocriptine could mitigate fever spikes, reinforcing the role of thalamic pathways in temperature control. (14)

A greater understanding of the neural mechanisms underlying central hyperthermia remains needed. Future research should focus on developing targeted therapies that address the disrupted pathways in thalamic infarctions. The role of neuroprotective agents and advanced imaging techniques in predicting and improving outcomes in such cases warrants further exploration (14,15).

CONCLUSION

Bilateral thalamic infarctions, though rare, can result in significant complications, including central hyperthermia, due to the disruption of critical neural pathways involved in thermoregulation. This case highlights the importance of recognizing hyperthermia as a non-infectious consequence of thalamic damage, which is often misdiagnosed, leading to delays in appropriate treatment. The use of neuroimaging, such as MRI, is indispensable for identifying the underlying lesion and guiding management strategies. In this case, the successful response to bromocriptine shows the role of dopamine modulation in managing central hyperthermia and emphasizes the need for tailored therapeutic approaches. Further research into the neural mechanisms of thermoregulation and the effects of ischemic lesions on autonomic function is crucial for developing targeted treatments. Ultimately, a multidisciplinary approach involving early diagnosis, supportive care, and pharmacological intervention is essential for improving outcomes in patients with thalamic infarctions and associated autonomic dysfunctions. This case reinforces the necessity of heightened clinical vigilance in identifying and addressing rare but serious complications of cerebrovascular events.

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