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Delayed Onset Anton-Babinski Syndrome following Post-Hypoxic Cortical Injury: A Case Report

Post-Hipoksik Kortikal Hasar Sonrası Gelişen Gecikmiş Anton-Babinski Sendromu: Olgu Sunumu

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ABSTRACT

Delayed post-hypoxic cortical blindness is a rare but clinically significant condition that can manifest days or weeks following a hypoxic event, such as cardiac arrest. During these events, the brain is deprived of oxygen and glucose, leading to selective vulnerability in metabolically active regions, particularly the occipital lobes. Although initial resuscitation may restore spontaneous circulation, secondary injury mechanisms can contribute to delayed and progressive neurological damage. Here, a case of Anton-Babinski syndrome manifesting as sudden cortical blindness and profound anosognosia following successful cardiopulmonary resuscitation was reported. Despite undeniable visual loss confirmed by bilateral occipital ischemia on neuroimaging, the patient insisted she could see and provided confabulatory descriptions, highlighting a rare and intriguing complication of post-hypoxic injury. This case provides a unique perspective to the limited literature on Anton-Babinski syndrome, highlighting the critical importance of early recognition and comprehensive evaluation in uncovering rare and elusive post-hypoxic complications.
Keywords: Cardiac arrest; hypoxia; cortical blindness; cerebral ischemia; hypoxic-ischemic encephalopathy; ischemia-reperfusion injury; Anton-Babinski syndrome.

ÖZ

Gecikmiş post-hipoksik kortikal körlük, kardiyak arrest gibi hipoksik olayları takiben günler veya haftalar sonra ortaya çıkabilen nadir ancak klinik olarak önemli bir durumdur. Bu tür olaylar sırasında beyin, oksijen ve glukozdan yoksun kalır ve özellikle metabolik olarak aktif bölgelerden olan oksipital loblar seçici şekilde zarar görür. İlk resüsitasyon işlemi spontan dolaşımı geri kazandırsa da, ikincil hasar mekanizmaları gecikmiş ve ilerleyici nörolojik hasara katkıda bulunabilir. Bu yazıda, başarılı kardiyopulmoner resüsitasyon sonrası ani kortikal körlük ve belirgin anozognozi ile ortaya çıkan çarpıcı bir Anton-Babinski sendromu vakası sunulmuştur. Nörogörüntülemesinde bilateral oksipital iskemi ile doğrulanan belirgin görme kaybına rağmen hasta görebildiğini iddia etmiş ve çevresini hayali şekilde tanımlayarak konfabülasyon yapmıştır. Bu vaka, Anton-Babinski sendromu üzerine sınırlı veri içeren literatüre özgün bir katkı sağlamakta ve nadir görülen post-hipoksik komplikasyonların ortaya çıkarılmasında erken tanı ve kapsamlı değerlendirmenin kritik önemini vurgulamaktadır.
Anahtar kelimeler: Kardiyak arrest; hipoksi; kortikal körlük; beyin iskemisi; hipoksik-iskemik ensefalopati; iskemi-reperfüzyon hasarı; Anton-Babinski sendromu.

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INTRODUCTION

Hypoxic brain injury is a significant cause of morbidity and mortality, particularly in the context of cardiac arrest, where global cerebral ischemia can lead to extensive neurological damage. The occipital cortex is particularly vulnerable to hypoxic injury

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due to its high metabolic demand, and even brief periods of oxygen deprivation can result in significant and sometimes irreversible neurological damage (1). Although prompt resuscitation following hypoxic events like cardiac arrest can restore circulation, it also introduces secondary injury mechanisms, including oxidative stress, inflammation, and excitotoxicity, which contribute to delayed neuronal damage and functional deficits (2).

Delayed post-hypoxic cortical blindness is a rare but serious neurological complication that may manifest hours to days after the initial hypoxic insult. Despite severe visual impairment, patients often retain pupillary light reflexes due to the preservation of anterior visual pathways. However, in cases where only partial damage to the occipital cortex occurs, patients may experience visual field deficits rather than complete blindness (3).

Anton-Babinski syndrome, characterized by cortical blindness, anosognosia (denial of blindness), and confabulation, is an uncommon condition in clinical practice and even rarer as a complication following cardiopulmonary resuscitation (CPR). The limited literature on post-CPR Anton-Babinski syndrome underscores a significant gap in understanding its pathophysiology, clinical course, and management (4). Early recognition of this condition is crucial, as with other delayed neurological deficits, where postponed diagnosis can significantly impede timely intervention and optimal rehabilitation outcomes (5).

This report aimed to present a case of an 80-year-old female who developed delayed-onset Anton-Babinski syndrome following successful CPR. By detailing this case, we aimed to contribute to the limited literature and emphasize the importance of early recognition, comprehensive evaluation, and multidisciplinary management of this rare but critical complication in post-hypoxic patients.

CASE REPORT

An 80-year-old female with a history of controlled hypertension and atrial fibrillation, but no prior neurological deficits, was brought to the emergency department following a witnessed cardiac arrest. Immediate CPR was initiated by bystanders, and emergency services continued advanced cardiac life support. Return of spontaneous circulation was achieved after approximately 10 minutes of resuscitation. Upon arrival at the hospital, the patient underwent coronary angiography due to electrocardiographic evidence of an acute myocardial infarction. Angiography revealed critical stenosis of the left anterior descending artery, necessitating immediate percutaneous coronary intervention with stent placement. Neurologically, the patient showed no deficits in the immediate postoperative period, exhibiting full motor and sensory function and following commands. On the second postoperative day, the patient reported diminished vision, describing her surroundings as "dim" and "blurred." However, family members observed that she was unable to see, frequently colliding with objects and showing no visual response to stimuli. Despite this, the patient insisted she could see and provided inaccurate descriptions of her environment. This discrepancy, combined with confabulation, led to the suspicion of cortical blindness. Neurological examination revealed no visual response to light, and there were no signs of motor or sensory deficits. Despite her blindness, the patient's pupillary reflexes were intact. During the initial evaluation, laboratory analysis revealed elevated serum lactate levels of 3.8 mmol/L (reference, 0.5-2.2 mmol/L) and a mild metabolic acidosis with a pH of 7.32 and bicarbonate level of 18 mmol/L, indicating significant tissue hypoxia and supporting the diagnosis of acute hypoxic injury. Brain magnetic resonance imaging (MRI) demonstrated bilateral ischemic lesions localized to the occipital cortices, providing definitive radiological confirmation of cortical blindness (Figure 1). Although visual evoked potentials (VEP) and electroencephalography (EEG) were initially planned to assess the functional integrity of the visual pathways and cortical activity, these procedures could not be completed due to the patient's cognitive confusion and restlessness. To objectively support the preliminary diagnosis, an ophthalmologic evaluation was conducted, which confirmed the cortical nature of the visual loss by demonstrating intact anterior visual pathways and ruling out ocular or optic nerve pathologies as potential causes of the blindness. A comprehensive supportive management approach, including structured neurological rehabilitation and close clinical monitoring, was initiated to optimize neurological recovery and functional outcomes. The patient experienced significant visual recovery one month later, with visual field deficits nearly resolved.

DISCUSSION

Anton-Babinski syndrome is characterized by cortical blindness accompanied by anosognosia and confabulation (4). The denial of visual loss, a hallmark of the syndrome, is believed to result from damage to neural pathways responsible for visual awareness, while primary visual input pathways remain unaffected. This explains why patients, despite profound blindness, confidently assert they can see and provide vivid but incorrect descriptions of their environment (6). In our case, the patient persistently claimed to see her surroundings, offering inaccurate visual details, despite clear evidence of blindness and frequent collisions with objects. Notably, pupillary reflexes remained intact, as the anterior visual pathways, including the optic nerves and brainstem structures, were preserved. This dissociation between visual perception and pupillary response is a critical diagnostic clue for cortical blindness (7). The patient's confabulation and denial of blindness further confirmed the diagnosis of Anton-Babinski syndrome, emphasizing the importance of thorough neurological assessment in post-hypoxic patients presenting with visual disturbances.

Anton-Babinski syndrome can arise from various etiologies, including bilateral occipital lobe infarction, traumatic brain injury, hypoxic-ischemic events, and, less commonly, neoplastic or inflammatory processes affecting the visual cortex (4). In evaluating the etiology of cortical blindness in this case, several differential diagnoses were considered, including hypoxic injury, cardiac embolism, posterior circulation stroke, vasculitis, and metabolic disturbances. However, the bilateral and symmetrical nature of the occipital ischemic lesions, along with the absence of focal neurological deficits typically associated with embolic events, favored hypoxic injury as the primary

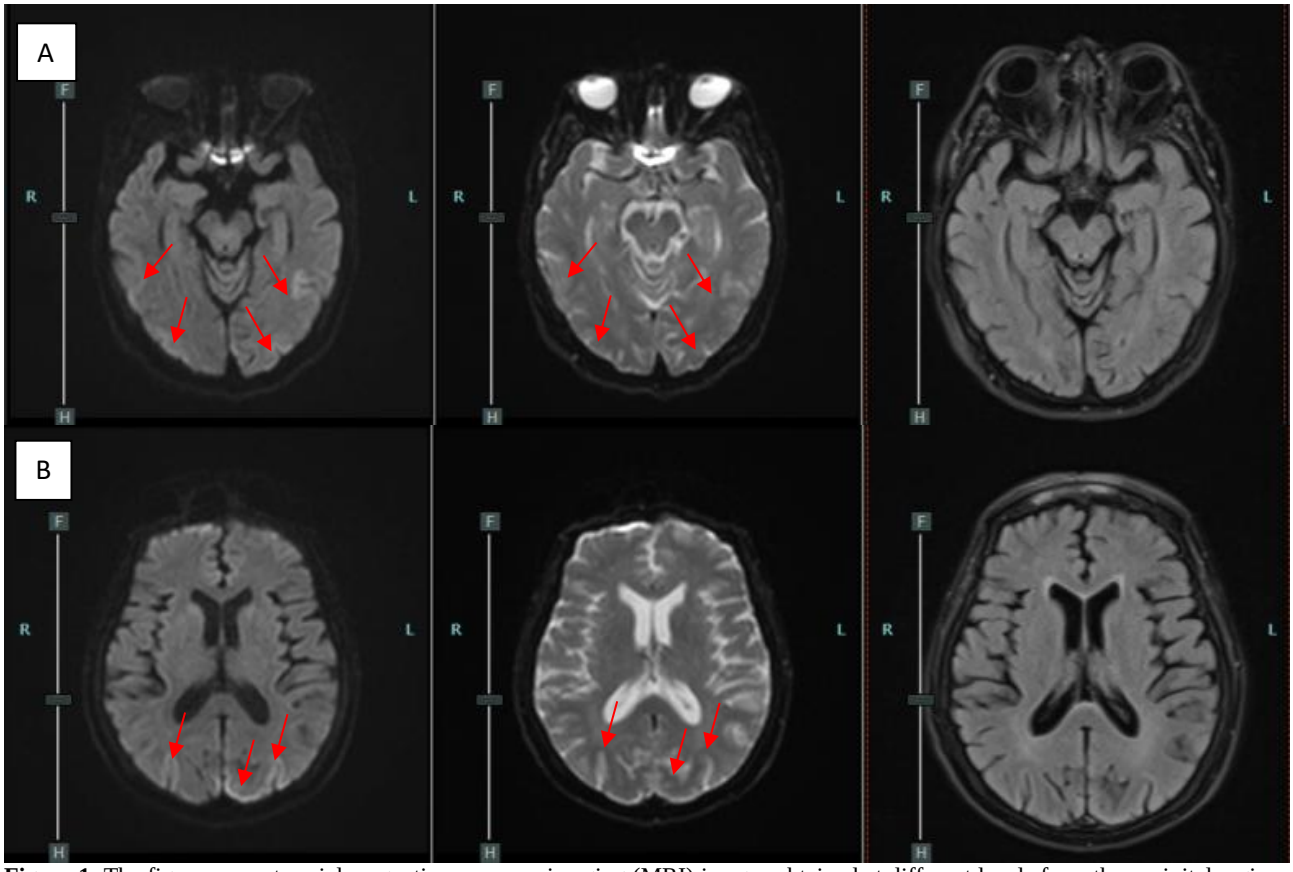

Figure 1. The figure presents axial magnetic resonance imaging (MRI) images obtained at different levels from the occipital regions using various sequences. Diffusion-weighted imaging (DWI) reveals restricted diffusion in the affected areas bilaterally. Corresponding apparent diffusion coefficient (ADC) maps show these regions as hypointense to isointense. T2-weighted fluid-attenuated inversion recovery (FLAIR) sequences demonstrate predominantly isointense lesions with limited focal areas of hyperintensity

cause. Elevated lactate levels and metabolic acidosis observed during the initial evaluation further supported the presence of significant global hypoxia. Moreover, imaging studies revealed no evidence of vascular occlusion, malignancy, or inflammatory processes that would indicate an embolic, neoplastic, or vasculitic origin. Taken together, these clinical and radiological findings reinforced the conclusion that hypoxia, rather than embolic phenomena, was the predominant factor contributing to the development of cortical blindness in this patient.

Anton-Babinski syndrome is an exceptionally rare but clinically significant neurological complication that may develop in elderly individuals who are more susceptible to post-hypoxic cortical injury due to factors such as reduced cerebrovascular reserve, age-related neuronal vulnerability, and comorbid conditions that can exacerbate ischemic damage (6). Although delayed post-hypoxic leukoencephalopathy secondary to white matter injury is infrequent, it has been documented in 2.75% to 10% of patients following carbon monoxide poisoning, emphasizing the potential for delayed neurological sequelae after hypoxic insults (5). Cortical blindness, another rare yet impactful complication, can result from hypoxic-ischemic injury to the occipital lobes. In this context, Anton-Babinski syndrome, defined by the coexistence of cortical blindness and anosognosia, has been specifically associated with bilateral occipital lobe damage, further underscoring the vulnerability of this region to hypoxic injury (8).

In this case, the development of Anton-Babinski syndrome following a hypoxic event underscores the complexity of post-hypoxic neurological outcomes, particularly in elderly patients with vascular comorbidities such as hypertension and atrial fibrillation. Although the initial neurological examination post-resuscitation was unremarkable, the patient later developed delayed-onset cortical blindness accompanied by anosognosia and confabulation, consistent with Anton-Babinski syndrome. The occipital lobes, being metabolically active, are particularly vulnerable to oxygen deprivation. While reperfusion restores oxygen supply, it also introduces reactive oxygen species, leading to oxidative damage, mitochondrial dysfunction, and calcium overload. These processes can initiate apoptosis, contributing to delayed neuronal injury and the subsequent onset of cortical symptoms days to weeks after the initial hypoxic insult (9). The pathophysiology of this case can be attributed to a combination of the initial hypoxic insult and secondary injuries triggered by reperfusion. Elevated lactate levels and metabolic acidosis observed during the initial evaluation indicated significant tissue hypoxia, underscoring the severity of the cerebral injury. The abrupt restoration of blood flow post-resuscitation likely initiated a cascade of secondary damage mechanisms, including oxidative stress, mitochondrial dysfunction, and excitotoxicity, which are known to contribute to delayed neuronal injury, particularly in the metabolically vulnerable occipital cortex (10). These pathophysiological

processes provide a plausible explanation for the delayed onset of cortical blindness, which emerged despite an initially uneventful neurological recovery following resuscitation. The onset of visual symptoms two days after resuscitation aligns with known patterns of delayed post-hypoxic cortical blindness, which can emerge after an initial recovery period (11).

The patient's partial visual recovery following comprehensive supportive care and structured neurological rehabilitation highlights the potential for neuroplasticity and functional improvement, even in elderly individuals with severe cortical injury. This case underscores the importance of early diagnosis and multidisciplinary management in optimizing outcomes for post-hypoxic complications. Consistent with previous reports, Anton-Babinski syndrome emerged as a delayed complication of post-hypoxic cortical injury (6-8). While some cases in the literature describe persistent visual deficits, others, like our patient, demonstrate significant recovery over time.

The observed improvement, characterized by the gradual resolution of visual field deficits over one month, was likely facilitated by early initiation of targeted rehabilitation, including visual scanning exercises, orientation training, and environmental adaptations to enhance visual perception and compensatory strategies. Additionally, psychological support was integral in addressing anosognosia and confabulation, core features of Anton-Babinski syndrome (12).

This case contributes to the limited literature by emphasizing that early and individualized rehabilitation can significantly influence visual outcomes, even after severe cortical injury. It also reinforces the necessity for long-term, multidisciplinary follow-up to monitor neurological progress and adapt rehabilitation strategies accordingly. Ultimately, this case highlights the value of a proactive, patient-centered approach in managing delayed post-hypoxic neurological deficits to optimize functional recovery.

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