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Capsular warning syndrome: Nighttime blood pressure drops, clinical awareness, and therapeutic approach

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Abstract:

Capsular warning syndrome (CWS) is a rare condition marked by recurrent, stereotypical transient ischemic attacks (TIAs) affecting the face, arm, and leg, without cortical involvement. It is associated with a high risk of a full-blown stroke within 7 days. The exact pathophysiological mechanism and optimal management strategies remain debated. It is crucial to distinguish CWS from crescendo TIAs and consider reperfusion therapy if new episodes occur within the therapeutic window for systemic reperfusion, in order to prevent a disabling stroke. We present the case of a 53-year-old male who arrived at the emergency department (ED) with right hemiparesis and facial weakness lasting for 1 h. He had experienced four recurrent, stereotypical episodes over the past 7 h and was diagnosed with a TIA, despite being within the therapeutic window for thrombolysis. He was started on dual antiplatelet therapy, high-dose statins, and management of other vascular risk factors. However, within 24 h, his condition progressed to a complete stroke with severe hemiparesis and facial weakness. Magnetic resonance imaging confirmed infarction in the left hemisphere, while a computed tomography angiogram was normal. This case underscores the importance of prompt recognition of CWS in the ED, enabling activation of stroke services and the consideration of reperfusion therapy when appropriate, to minimize the risk of a disabling stroke.

Keywords:

Blood pressure fluctuations, capsular warning syndrome, emergency department, small vessel disease, stroke, transient ischemic attack

Introduction

Capsular warning syndrome (CWS) is a rare condition, accounting for 1.5%–4.5% of transient ischemic attacks (TIAs), but it carries a high risk of progressing to a full stroke, with rates as high as 60% within the 1st week, most commonly within the first 48 h.[1-3] Atherosclerotic disease, artery-to-artery embolization, and hemodynamic instability due to blood pressure (BP) fluctuations are the potential underlying mechanisms.[3,4] This case illustrates hemodynamic instability as a cause of CWS and highlights the challenges of managing acute neurological deficits in the emergency department (ED). It emphasizes the importance of involving acute stroke services early to assess the potential for thrombolysis before the progression to a completed stroke.

Case Report

A 56-year-old male with diabetes mellitus (DM) was brought to the ED after experiencing multiple episodes of right-sided weakness over the past 10 h. He described each episode as sudden

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weakness affecting his face, right arm, and leg, with complete recovery within 15 min. The first two episodes occurred during the night while he was asleep, and he woke up to find himself experiencing the weakness. The patient had no history of smoking or alcohol use and worked as a driver. On physical examination, his BP was 170/110 mm Hg, heart rate was 96 beats per minute and regular, respiratory rate was 15 breaths per minute, and he was afebrile. Neurological examination was unremarkable. Initial computed tomography of the head revealed old left lentiform lacunar hypodensities, while computed tomographic angiography showed patent major intracranial arteries [Figure 1]. He was admitted as a case of TIA and managed with aggressive intravenous hydration, dual antiplatelet therapy (DAPT), high-dose statins, and subcutaneous insulin. Permissive hypertension was allowed for the first 72 h of his admission. Approximately 10 h later, during the night, he experienced another episode of weakness, with right leg strength at 2/5, right arm strength at 3/5, and increased facial weakness. His National Institutes of Health Stroke Scale score was 5, and there was no subsequent recovery. During this period, his BP dropped to 132/70 mm Hg, as shown in the BP chart [Figure 2]. Magnetic resonance imaging of the brain revealed well-defined hypodensities at the dorsal aspect of the left putamen and the tail of the caudate nucleus, extending into the left corona radiata, consistent with acute infarction [Figure 3]. A diagnosis of CWS was established. Written informed consent was obtained from the patient for participation in this study.

Discussion

CWS was first described by Donnan *et al*. in 1993, who identified the syndrome as occurring after at least three stereotyped episodes of sensory and/or motor symptoms involving the face, arm, and leg, without cortical involvement, within a 24-h period.[5] Other timeframes used to define the syndrome include 48 h or even up to 7 days.[1,6] The absence of cortical signs in CWS is due to its localized involvement of the internal capsule, and occasionally the midbrain, thalamus, pons, or striatum.[5,7] Various treatment modalities, including BP control, antiplatelet or anticoagulant therapy, and thrombolysis, have been recommended; however, the optimal clinical management remains a topic of debate.[3] Literature suggests that prior TIAs are generally associated with a favorable outcome in nonlacunar ischemic strokes; however, this ischemic tolerance is not observed in lacunar strokes associated with small vessel disease.[8] Our case illustrates nocturnal hemodynamic instability as a potential etiology of CWS and fits the profile of cases that exhibit partial recovery, with DM and hypertension identified as key risk factors, particularly in male patients.[5] Lipohyalinosis and endothelial dysfunction, resulting from chronic arterial hypertension in diabetic patients, are among the most common risk factors and may contribute to vasculopathy in the lenticulostriate arteries, potentially leading to CWS. Additionally, some reports suggest that fluctuations in BP may play a role in causing neurological deficits.[9] Lalive *et al*. reported several cases where fluctuations in BP coincided with neurological deterioration, leading to complete strokes. In contrast, one patient who had their BP maintained with vasopressors made a full recovery.[9] They concluded that BP fluctuations could contribute to the occurrence of lacunar infarctions. Additionally, another study highlighted BP augmentation as an effective strategy to mitigate the stuttering course of lacunar infarctions.[10] A study by Watanabe *et al*., involving elderly women, found that either an inappropriately low nocturnal BP or an excessive nocturnal BP drop was linked to ischemic silent cerebrovascular lesions.[11] Several studies have indicated that cerebral blood flow (CBF) in humans remains unaffected by changes in mean arterial pressure (MAP) within a range of approximately 60–150 mm Hg, a phenomenon known as static cerebral autoregulation (CA). The optimal BP for an individual depends significantly on underlying pathophysiology and other personal factors. In patients with chronic

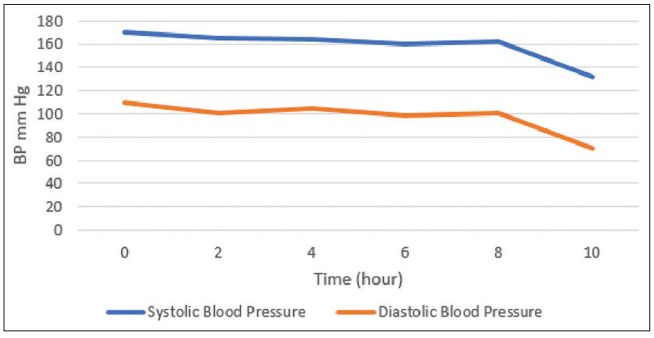

Figure 1: Blood pressure chart during admission. BP: Blood pressure

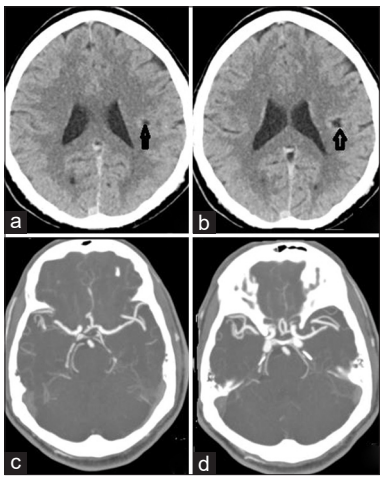

Figure 2: Computed tomography head showed old left lentiform hypodensities (a and b), and computed tomographic angiography showed patent all major intracranial arteries with no flow limiting stenosis (c and d)

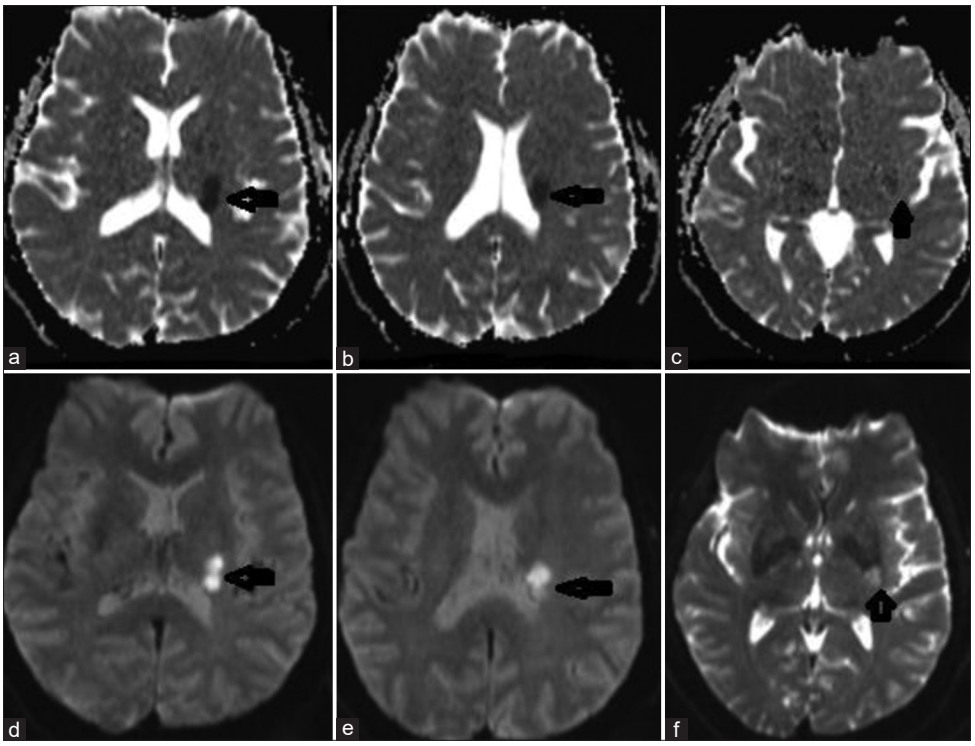

Figure 3: Magnetic Resonance Imaging Head. There is abnormal area of diffusion restriction (a-f) with corresponding high T2/ FLAIR signal involving the dorsal aspect of the left putamen as well as tail of the caudate nucleus extending into the left corona radiata. representing acute infarction

hypertension, the relationship between CBF and MAP shifts toward higher BP levels. This is also observed in individuals with diabetes. In hypertensive patients, CA may be impaired even at "normotensive" MAP levels. In people with poorly controlled chronic hypertension, CBF may passively fluctuate with MAP, and a drop in MAP to lower levels may result in a rapid decline in CBF.[12]

Our patient presented with a high BP of 170/100 mm Hg upon admission to the general ward. However, during the night at 03:40 AM, he experienced another episode, which did not resolve. At that time, his BP dropped to 132/70 mm Hg, as recorded. We speculate that this significant drop in BP may have contributed to the progression to a completed stroke. One study documented that neurological worsening was associated with a drop in systolic BP of 30 mm Hg or more.[7] Various treatment options have been proposed for these patients, including intravenous recombinant tissue plasminogen activator (rtPA), oral anticoagulants, and vasopressors. Despite the availability of several therapeutic options, there is no consensus on the most effective treatment for CWS, and there remains a lack of evidence to support an improvement in the disease course with any of these interventions.[3,5]

In addition to these potential treatment options, elevating BP has been suggested as a strategy to improve distal vessel hypoperfusion. This, in turn, may enhance perfusion to the affected brain region and reduce the risk of adverse outcomes.[3] The duration of TIA episodes has been documented to vary between 6 and 24 min.[1] Our patient experienced multiple episodes lasting 10–15 min, with residual neurological deficits. He was initially loaded with DAPT, but rtPA could not be administered as he was beyond the 4.5-h therapeutic window. There is a need to revise the definition of CWS to include both TIAs and patients with infarctions who experience transient symptoms, as the majority of diagnoses are made based on clinical presentation.[13] A subsequent analysis of the WAKE-UP trial demonstrated promising functional outcomes for lacunar infarct patients who received rtPA, compared to other stroke subtypes, suggesting that rtPA may be an effective treatment option for CWS. However, further studies are needed to confirm this.[14]

To date, available studies have shown no statistically significant differences in therapeutic effects or functional outcomes between CWS patients treated with rtPA or antiplatelet drugs.[2,3] Moreover, there is limited evidence supporting the efficacy of anticoagulant treatment in the acute phase of stroke. However, a few case studies have suggested potential benefits of DAPT over single-antiplatelet use.[15]

Conclusion

This case highlights the importance of augmenting BP and avoiding fluctuations in the context of acute ischemic

stroke and TIA. Another crucial point is the need for awareness of this clinical entity, which should prompt physicians to consider rtPA for long-duration TIAs. Due to the rarity of this syndrome, there is limited data on optimal management; however, DAPT has been used safely in noncardioembolic CWS and appears to be a reasonable option before initiating anticoagulation in cardioembolic strokes. More research and randomized trials are needed to identify the treatment options that can effectively impact disease progression.

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Author contributions statement
ES contributed for conceptualization, collecting patient's data, writing the initial manuscript. RS revised the manuscript.

Conflicts of interest
None Declared.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understand that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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