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**Case Report**

# 'Crescendo transient ischemic attack'—an uncommon presentation of a very common disease: a case report on capsular warning syndrome

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**Abstract**

Capsular warning syndrome is a rare presentation of transient ischaemic attack, which described as recurrent episodes of motor and/or sensory deficits which typically sparring the cortical function. It has a significant risk to progress into a massive stroke with permanent disability, thus important to be recognise early. Here, we report a middle-age gentleman with no known medical illness presented with eight episodes of transient ischaemic attack within the span of 24 h. He was treated with double anti-platelet for 21 days and was not subjected to thrombolysis at time of presentation because it was outside the window period of 4.5 h, and has fully recovered after each episode. The purpose of this case report is to share the uncommon clinical presentation of transient ischaemic attack, which is still not fully understood and warrant more studies especially on the treatment that can affect the progression of the disease.

## INTRODUCTION

Capsular warning syndrome (CWS) was first described in 1993 by Donnan *et al.* [1] as recurrent episodes of transient ischaemic attacks (TIA) which predominantly involve the internal capsule, presenting with motor and/or sensory deficits and sparing the cortical function.

Although this syndrome is rare and only reported in <5% of all cases of TIA, but it has a high risk of developing imminent stroke [2]. The pathophysiological mechanism of symptoms fluctuation in this syndrome is still not yet fully understood. As this syndrome is rare, there is still no consensus on the treatment that will modify the disease progression.

## CASE REPORT

A 62-year-old gentleman with no known medical illness presented to the emergency department with recurrent episode of left sided body weakness for the past 12 h. He described that each episode lasted for 15–30 min before recovery and was associated with difficulty to stand and walk during the weakness. He suffered about six episodes prior to admission and another two episodes while he was in the emergency department. He claims that each episode will follow a full recovery. He has no preceding head injury, no fever, no loss of consciousness, no visual disturbance and no slurring of speech. On further history, he is not taking any regular prescribed or over the counter medications, and never take any traditional or anti-aggregation medication. There was no similar condition occur in the family. He is a chronic cigarette smoker who smoke one pack year cigarette for the past 40 years, and do not drink alcohol. He works as a constructional contractor in his own company. Upon arrival to the emergency department, he was alert, conscious and fully orientated to time, place and person. His blood pressure was 180/100 mm Hg, pulse rate was 100 bpm, and it was regular rhythm with normal volume, respiratory rate was 20 bpm and he was afebrile. On physical examination, the muscle power for both upper and lower limb was full, there was no sensory abnormality, no ataxia and his cranial nerve was intact. Examination of the cardiorespiratory system reveal no abnormality, and his abdomen was soft, not tender and there were no palpable organomegaly nor mass noted. Initial blood investigations were taken and shows normal cell counts, renal and liver function, lipid profile and fasting blood glucose. There was no electrolyte abnormality noted (as shown in Table 1). Plain computed tomography (CT) scan of the brain shows no intracranial bleeding nor any infarction. While waiting for admission in the emergency department, he developed another two episodes of transient left sided hemiparesis which lasted for 15 min. Urgent CT angiography of the brain was done and shows small

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**Table 1.** Initial blood investigation upon admission shows normal blood cells count, renal profile and liver function, no electrolyte abnormality, normal lipid profile and fasting blood glucose

| Blood parameters | Result | Normal range |
|---|---|---|
| Haemoglobin | 14.5 g/dl | 12–18 g/dl |
| Platelet | 282 × $10^9$/L | 150–400 × $10^9$/L |
| White blood cell | 7.5 × $10^9$/L | 4.0–11.0 × $10^9$/L |
| Creatinine | 86.4 $\mu$mol/L | 60–120 $\mu$mol/L |
| Sodium | 140 mmol/L | 135–150 mmol/L |
| Potassium | 3.9 mmol/L | 3.5–5.0 mmol/L |
| Urea | 4.7 mmol/L | 1.7–8.0 mmol/L |
| Magnesium | 0.86 mmol/L | 0.66–1.07 mmol/L |
| Corrected calcium | 2.28 mmol/L | 2.14–2.58 mmol/L |
| Albumin | 40 g/L | 35–50 g/L |
| Alkaline phosphatase | 56 U/L | 50–150 U/L |
| Alanine transaminase | 30 U/L | 5–35 U/L |
| Total bilirubin | 9.2 $\mu$mol/L | 0–13 $\mu$mol/L |
| Total cholesterol | 5.08 mmol/L | <5.2 mmol/L |
| HDL | 0.95 mmol/L | 1.20–3 mmol/L |
| LDL | 3.65 mmol/L | <3.80 mmol/L |
| Triglycerides | 1.05 mmol/L | <1.70 mmol/L |

well defined hypodensities at the body of right caudate nucleus. The major intracranial arteries have normal calibre and there is no filling defect to suggest thrombosis over the vertebrobasilar arteries (as shown in Fig. 1). He was started on dual oral anti-platelet (aspirin 300 mg loading dose then 150 mg daily, and clopidogrel 300 mg loading dose then 75 mg daily thereafter). The clopidogrel was plan for 21 days only, and then he will continue with single anti-platelet thereafter for life. He was also started on oral atorvastatin 40 mg once every night, and oral perindopril 4 mg daily. He was discharge after no further episode of transient hemiparesis for 48 h observation in the ward. He was schedule for magnetic resonance imaging of the brain later to assess the brainstem. Upon follow-up via teleconsultation after 3 months, the patient is well with no residual weakness. He continues to take his oral aspirin 150 mg daily, oral atorvastatin 40 mg once every night and oral perindopril 4 mg daily.

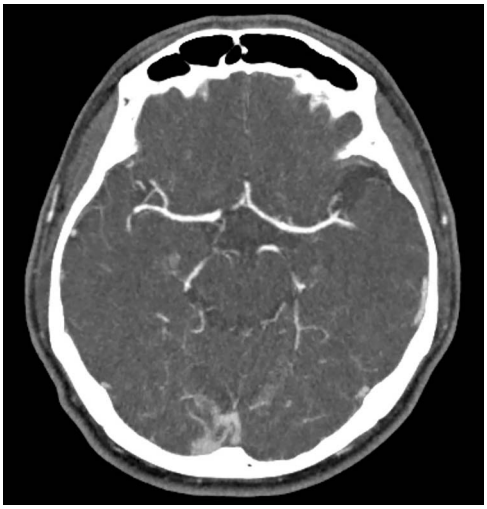

**Figure 1.** Computed tomographic angiography of the brain shows small well defined hypodensities at the body of right caudate nucleus. The major intracranial arteries have normal calibre and there is no filling defect to suggest thrombosis over the vertebrobasilar arteries.

## DISCUSSION

CWS is a term to coined multiple episodes of stereotyped TIA which usually occur in the proximity of each other in time.

It was first described by Donnan *et al.* in 1993 [1] as a recurrent episode of motor or sensory, or both sensorimotor deficits in the absence of cortical symptoms. CWS is rare, where the incidence was only 1.5%–4.5% of TIA. However, it is particularly important because it has a high risk of developing massive ischaemic stroke with a permanent neurological deficit. According to a report from a population study, the 7-day stroke risk following a CWS is as high as 60% [2]. The classical clinical presentations of CWS are unilateral pure motor or sensory or sensorimotor deficits that involve at least two of the three (face, arm or leg). CWS characteristically do not have any cortical signs [1]. This is because CWS commonly affect the internal capsule area [3], or in some cases, the pons, midbrain and thalamus [3, 4].

The exact pathophysiological mechanism of CWS is still remain unknown. There were studies that relate comorbidities with hypertension, diabetes, dyslipidaemia, cigarette smoking and other stroke risk factors to developing CWS, which suggest that atherosclerosis of the small-penetrating arteries may be involved in the pathogenesis [4, 5]. There was also some speculation that CWS was associated with small-penetrating arterial disease where intermittent hemodynamic changes due to structural arterial changes or hypertension leading to ischaemia [4]. However, the reasons behind fluctuations of symptoms are still yet to be discover.

Up to date, there is still no consensus on the definite treatment for CWS. Intravenous (IV) thrombolysis,

oral anti-platelet therapy whether double or single, oral anticoagulants and some less common like vasopressors have been used to treat CWS patient. It has been an ongoing debate whether CWS should undergo thrombolysis or not. Some studies shown thrombolysis was effective in making the symptoms disappear [6, 7], whereas other studies showed there was no significant different in functional outcome between CWS patient that undergone thrombolysis and those who did not [4]. However, IV thrombolysis has been safe for CWS patients, and there was no bleeding complication have been reported. Therefore, the author believes IV thrombolysis is an alternative treatment for CWS patient that has a prolonged clinical event and present within the therapeutic window of 4.5 h after the onset of symptoms.

Our patient presented with a CWS symptoms and was treated with double anti-platelet. He did not fulfil the criteria for thrombolysis because he presented outside the window period of 4.5 h, and his symptoms was mild and short lived before complete recovery. Immediate plain CT scan of the brain was normal, but CT angiography of the brain later confirm that there is an acute infarction over the internal capsule that correlate with the clinical presentation.

In conclusion, capsular warning syndrome is a rare spectrum of transient ischaemic attack which has high tendency to develop a full-blown stroke. There is still no general consensus on the treatment options of the condition. Therefore, further research that focused on the treatment that will alter the disease progression should be made.

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## CONFLICT OF INTEREST STATEMENT

None declared.

## CONSENT

Consent from the patient was taken for the writing and publication of this case report.

## ETHICAL APPROVAL

No ethical approval is required for case report in our centre.

## GUARANTOR

Alvin Oliver Payus.

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