



# Aura attacks from acute convexity subarachnoid haemorrhage not due to cerebral amyloid angiopathy

Cephalalgia

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## Abstract

**Background and purpose:** Convexity subarachnoid haemorrhage (cSAH) has recently been recognised as a cause of recurrent aura-like symptoms, mimicking transient ischaemic attacks (TIAs). Subarachnoid haemorrhage and recurrent aura-like episodes can occur in patients with cerebral amyloid angiopathy (CAA), which has been the presumed cause in the majority of reported cases. However, this syndrome can occur following cSAH secondary to other conditions, and it is important for clinicians to investigate and manage such patients appropriately.

**Method:** Case series.

**Results:** We describe two patients who presented with recurrent stereotyped transient neurological symptoms in the setting of acute cSAH identified on MRI. In one patient, SAH occurred secondary to cerebral venous sinus thrombosis. In the other, SAH was due to extension of a traumatic subdural haematoma.

**Conclusions:** Conditions other than CAA can cause the clinicoradiological syndrome of cSAH with recurrent TIA-like events. Gradient echo or susceptibility-weighted imaging should be included in the diagnostic work-up of patients presenting with such events. When cSAH is detected, the full differential diagnosis for this should be considered. Aetiologies other than CAA can cause this syndrome and management can vary greatly depending on the underlying cause.

## Keywords

Aura, subarachnoid haemorrhage, cortical spreading depression, TIA mimic, cerebral amyloid angiopathy

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## Introduction

Transient ischaemic attack (TIA) mimics are important to identify, as misdiagnosis can lead to inappropriate management. An important TIA-mimic syndrome has recently been recognised in which convexity subarachnoid haemorrhage (cSAH) causes flurries of transient aura-like sensory or sensorimotor deficits (1,2). This syndrome may be a relatively common cause of “late life migraine accompaniments” (3,4), as well as transient neurological symptoms in patients with cerebral amyloid angiopathy (CAA) (5), a condition which has been associated with atraumatic cSAH (6,7), particularly in those aged over 60 years (7).

However, there are many possible causes of cSAH (8), and CAA should not be assumed as the cause of TIA-mimic syndrome in the elderly. We describe two patients over the age of 60 years who presented with the clinicoradiological syndrome of cSAH with recurrent

TIA-like events, not due to CAA. In one case, the cause of cSAH was cerebral venous sinus thrombosis (CVST); in the other, a traumatic subdural haematoma had extended to the subarachnoid space.

## Patient I

An 81-year-old woman with preceding hypertension, dyslipidaemia and breast cancer presented with recurrent transient episodes of right-sided numbness and

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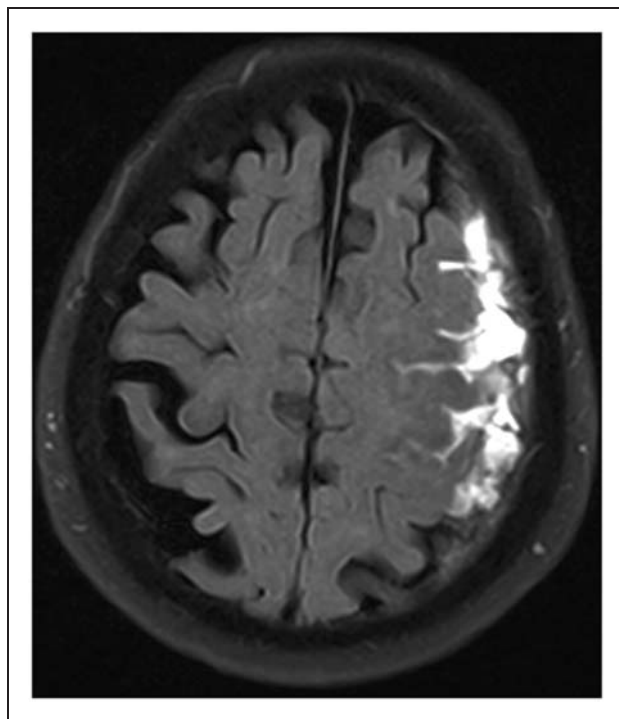
weakness. She had tripped and fallen two weeks previously, sustaining a right orbital floor fracture with associated retrobulbar haemorrhage, orbital compartment syndrome and altitudinal field loss.

She presented to the hospital after repeated episodes of numbness which spread gradually over minutes to involve the right shoulder, arm and then leg. There was associated mild-moderate right-sided weakness. Symptoms resolved in 10–20 minutes. She experienced four episodes over as many days and had another event shortly after admission to the hospital. Neurological examination was unremarkable except for the pre-existing altitudinal field loss. Computed tomography (CT) scan of the head demonstrated a left fronto-parietal subdural haematoma, which was not present on the scan from two weeks prior. MRI confirmed the subdural haematoma, but also demonstrated extension into the subarachnoid space around the central sulcus (Figure 1). Electroencephalogram showed focal slowing in the left temporal region but no epileptiform activity. The patient was initially loaded with phenytoin, and then commenced on sodium valproate. Episodes did not subsequently recur.

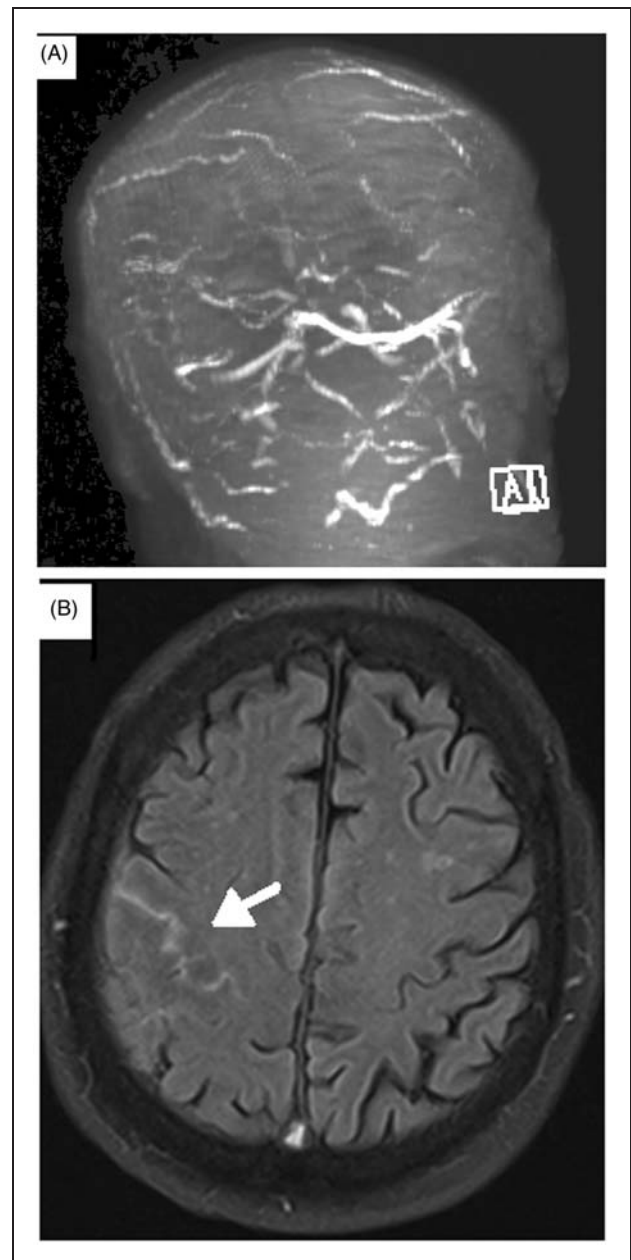
## Patient 2

A 70-year-old man with hypertension, dyslipidaemia and type II diabetes mellitus presented with

recurrent transient episodes of left-sided weakness. A fronto-temporal headache had occurred three weeks previously, lasting for three days. Two weeks after the initial headache, he experienced an episode of left facial and arm weakness, and slurred speech, which resolved after 15 minutes. The following day he had a similar episode which lasted 10 minutes. Approximately one week later, he had the third episode, which consisted of left facial droop with tingling sensation spreading from the left arm to the left leg over 10 minutes. This latter episode lasted for 30 minutes,



**Figure 1.** Fluid attenuated inversion recovery (FLAIR) image showing left subdural haematoma and left central sulcus subarachnoid haemorrhage.



**Figure 2.** (A) Magnetic resonance venogram showing sagittal sinus thrombosis, (B) FLAIR image showing right central sulcus subarachnoid haemorrhage.

was associated with a mild occipital headache and precipitated his presentation to the hospital.

Neurological examination was unremarkable. CT head scan showed cSAH in the right central sulcus, as well as a positive Delta sign, suggestive of sagittal sinus thrombosis. MRI of the brain confirmed the sagittal sinus thrombosis (Figure 2A), as well as the right central sulcus SAH (Figure 2B). Thrombophilia and autoimmune screens were negative. The patient was started on heparin and warfarin. During hospital admission he developed three further transient episodes of left facial and arm weakness, which resolved within 10 minutes and were associated with occipital headache. Following commencement of sodium valproate he experienced no further episodes.

## Discussion

We present two cases of cSAH-associated recurrent transient aura attacks secondary to a traumatic subdural haematoma and CVST, respectively. Although it has long been known that CAA can cause recurrent TIA-like symptoms (5), it has only recently been appreciated that cSAH is a common (and perhaps invariable) concomitant (1). CT scanning, while effective at detecting large and hyperacute SAH, becomes less sensitive with time and lesser degrees of haemorrhage; MRI is far superior in these settings (9), especially when fluid-attenuated inversion recovery (FLAIR), gradient echo and susceptibility-weighted imaging sequences are used (8). Because recurrent episodes can occur long after the initial CT signature hyperdensity has resolved, it is only recently, as newer imaging sequences are routinely employed, that the link to cSAH has been appreciated.

Several cSAH cases of unknown aetiology with similar recurrent neurological symptoms can be found in the literature (10), and it has been proposed that cases not meeting diagnostic criteria for CAA (11) may represent a "forme fruste". This suggestion is not unreasonable, as CAA was the probable cause of every case of convexity superficial siderosis discovered incidentally in a recent community-based MRI study (6).

However, CAA is not the only cause of cSAH (8), and it is important to consider other aetiologies, particularly in the absence of convexity cerebral microbleeds. Acute cSAH can occur following trauma, cerebral venous thrombosis, Moya Moya disease, primary CNS angiitis, arteriovenous malformations and fistulas, severe atherosclerotic carotid stenoses and drug- and pregnancy-related vasculopathies. Although "TIA-like" episodes have been occasionally reported in association with cerebral venous thrombosis (12,13), no reports have previously associated this with both cSAH and recurrent transient neurological symptoms.

A recent retrospective study (7) of 29 patients with atraumatic cSAH found that the majority of such

patients over the age of 60 years had evidence of CAA on imaging, whereas those 60 years or younger tended to have imaging findings of reversible cerebral vasoconstriction syndrome (RCVS). Our two patients, both well over the age of 60, demonstrate the importance of considering causes of cSAH other than CAA. Patient 1, in particular, illustrates the necessity of taking a history regarding recent trauma, as she did not develop symptoms from her haemorrhage until almost two weeks post-fall. Kumar et al.'s study (7) also noted that severe headache was a prominent clinical feature in those 60 years or younger, but was rare in those over this age, whereas transient sensory or motor symptoms tended to occur in those greater than 60 (7 of the 8 patients with these symptoms were over 60 years old). In keeping with these findings, severe headache was not a feature in our two patients (although patient 2 had mild headache), and both presented with recurrent transient neurological symptoms. The cause for this apparent age-related difference in clinical presentation remains unclear, and requires further study, but could possibly reflect altered cerebral vascular reactivity with age (14,15).

It has been previously recognised that cSAH-associated transient neurological episodes evolve in a fashion more consistent with migrainous aura than with epilepsy or ischaemia (1). Coupled with recent studies which have demonstrated cortical spreading depression (CSD) in both experimental (16) and human SAH (17), it is probable that CSD induced by subarachnoid blood products is the pathophysiological mechanism and that the reported efficacy of anticonvulsants in this setting is probably from suppression of CSD. Our current report demonstrates that CAA should not be assumed as the cause when this syndrome is encountered in clinical practice. The full differential diagnosis of cSAH should be considered, as treatment aimed at the underlying cause may vary significantly, from (for instance) anticoagulation for cortical vein thrombosis, to immunosuppression for cerebral vasculitis, or embolisation for a dural arteriovenous fistula.

In summary, aetiologies other than CAA can cause the clinicoradiological syndrome of convexity SAH with recurrent TIA-like events. Not only should gradient echo or susceptibility-weighted imaging be performed in all patients presenting with recurrent TIA-like events; for patients in whom cSAH is detected, the full range of underlying diagnostic possibilities should be considered.

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