Convexity subarachnoid haemorrhage

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Aim

1. To review the causes of convexity subarachnoid haemorrhage (SAH).

2. To outline the varying clinical presentations of convexity SAH and to illustrate the imaging findings of convexity SAH, with particular focus on reversible cerebral vasoconstriction syndrome and cerebral amyloid angiopathy.
Methods and materials

Background

SAH occurring at the cerebral convexities is thought to be an under-recognised entity, comprising an estimated 7% of all cases of SAH. It is most commonly seen in association with trauma however it occurs in the context of amyloid angiopathy in patients over 60 years of age and as a sequelae of reversible vasoconstriction syndrome in patients under the age of 60. Other causes of non-aneurysmal convexity SAH include cortical vein or dural vein thrombosis, infective endocarditis, coagulopathies and posterior reversible encephalopathy syndrome (PRES). This poster will focus on non-traumatic causes of convexity SAH.

Imaging Findings

Convexity SAH is defined as bleeding restricted to the hemispheric convexities. Acutely, there is hyperattenuation in the sulci of the convexities on CT or T2 hyperintensity on FLAIR sequences on MR. In reversible cerebral vasoconstriction syndrome, the convexity haemorrhage is usually mild. Segmental cerebral vasoconstriction, often with a 'string of beads' appearance is demonstrated in the intracranial internal carotid arteries, basilar artery or major arteries of the circle of Willis, which resolves spontaneously within 3 months. In amyloid angiopathy, haemorrhage occurs most frequently in the perirolandic region. On GRE T2 weighted sequences, multiple lobar and subcortical haemorrhages which classically spare the deep grey matter are seen. Superficial siderosis may extend beyond the site of acute haemorrhage suggesting previous occult convexity SAHs.
Results

SAH is typically defined by location as diffuse, perimesencephalic and convexity, based on the epicenter of the haemorrhage.

*Practice point:* location should ideally be determined within 3 days of symptom onset as substantial redistribution of blood occurs after this time.

**Cerebral convexity SAH**

- Intrasulcal bleeding restricted to the hemispheric convexities [3]
- Causes:
  - Trauma - most common cause (Fig 1)
  - Non-traumatic -
    - < 60 years of age: reversible cerebral vasoconstriction syndrome (RCVS). *Presentation:* sudden onset of severe headache +/- neurological deficits
  - No reported cases of ruptured circle of Willis saccular aneurysms causing isolated convexity SAH [2]

**Imaging of convexity SAH**

**CT:** hyperattenuation in the sulci (acute) (Fig 2 - 6)

**MRI:** haemorrhage is T2 hyperintense on FLAIR sequence and hypointense on gradient echo T2*; prior episodes of convexity SAH may appear as low signal intensity haemosiderin filling a sulcus (subarachnoid siderosis) or staining the underlying cortex (superficial cortical siderosis) [4]

*Practice points:*

- MRI is at least as sensitive as CT in the detection of acute SAH and is more sensitive in the detection of subacute SAH;
- gradient echo T2* is the most sensitive MRI sequence to detect SAH [5]

**REVERSIBLE CEREBRAL VASOCONSTRICTION SYNDROME (RCVS)**
• An uncommon but increasingly recognised condition presenting with sudden headache and neurological deficits
• Due to segmental constriction of cerebral arteries that resolves spontaneously within 3 months [6]

Clinical Considerations

• most commonly seen in women aged 20-50 years [7]
• seen in pregnancy, the post partum period or following exposure to adrenergic or serotonergic drugs, tobacco, marijuana or exercise [2]
• "thunderclap headaches": severe pain, peaks in seconds, lasts 1-3 hours (c.f. aneurysm headache) and recurs for 1-2 weeks. Pain is usually bilateral and posterior. Nausea and vomiting common.
• uniphasic clinical course: no new symptoms occurring >2 months after clinical onset. Typically self-limiting however haemorrhagic and ischaemic strokes [6] may result from prolonged vasoconstriction, most commonly in watershed areas and within the posterior circulation [7].
• nonaneurysmal SAH may occur
• calcium channel blockers such as nimodipine decrease headache. Intraarterial verapamil has been used to reverse vasoconstriction but the effect on haemorrhagic or ischaemic complications is questionable [7]

RCVS Imaging Findings

• imaging is usually normal in early stages
• angiography may only be suggestive of RCVS when maximal vasoconstriction occurs (~ 2-3 weeks after clinical onset) [6].

1. Haemorrhage

• localized cortical SAH is seen in 20-25% cases, usually mild and early in the clinical course[2] (Figures 7 - 13)
• rarely diffuse SAH or perimesencephalic SAH
• subdural haemorrhage occurs occasionally
• differential diagnosis for convexity SAH in RCVS is amyloid angiopathy however this affects an older population and is not associated with headache
• focal parenchymal haemorrhage is usually solitary and lobar. It is more common in females and in people with a history of migraine [6]

2. Cerebral infarction

• occurs in 7% cases [8]
• ischaemic or haemorrhagic strokes can occur a few days after initial normal imaging [6]
3. Oedema

- reversible cerebral oedema usually occurs early, within a few days of the onset of RCVS symptoms
- resolves within 1 month, usually prior to the resolution of vasoconstriction
- resembles PRES (Fig 14 - 17)
- Both RCVS and PRES may be part of the same process which results in reversible grey and white matter lesions often in the setting of hypertension [7].

4. Vasoconstriction

- segmental cerebral vasoconstriction of internal carotid, basilar, major arteries of the circle of Willis [7] (Fig.11-13, 8-21) giving arteries a "string of beads" appearance [8]
- vessel irregularity is most often bilateral, proximal and diffuse [6] (Fig. 22-23)
- arterial narrowing may fluctuate: repeat angiography after a few days can show resolution in some vessels and new constrictions in other vessels [7]
- RCVS is associated with vertebral artery dissection: seen in12% of patients with RCVS [6, 9]. (Fig. 18-21)

CEREBRAL AMYLOID ANGIOPATHY (CAA)

- common cause of cerebrovascular disorders that primarily affects elderly patients [10]
- diagnosis is based on histopathological finding of deposition of amyloid protein in cortical and leptomeningeal vessels [2]

Clinical Considerations

- degenerative angiopathy characterized by deposition of beta amyloid peptide in the walls of medium and small cortical and meningeal arteries
- preferentially affects the occipital regions
- incidence is age dependent: 33% at 60-70 years of age and 75% over the age of 90 years [10]
- Boston criteria describes the diagnosis of "possible " or "probable CAA" in patients over 50 years with lobar haemorrhages, microhaemorrhages and multiple white matter lesions on imaging [2]
- underdiagnosed cause of cerebrovascular disease as patients are often asymptomatic
- when symptomatic in the elderly, intracerebral haemorrhage is the most well known manifestation [10]
- clinical presentation varies including sudden neurological deficit, seizures, transient symptoms and progressive cognitive decline rather than headaches [1]
CAA Imaging Findings

1. Intracerebral Haemorrhage
   - haematoma with subcortical/cortical distribution generally sparing the deep white matter, basal ganglia and brainstem [10] (Figs 24-28)

2. Microhaemorrhages
   - multiple, lobar, cortical and subcortical haemorrhagic foci lesions best seen on GRE T2 (SWI) (Fig 2-6)
   - haemorrhages classically spare the deep grey matter [1] (Fig 29)

3. Subarachnoid haemorrhage
   - convexity SAH may be due to disruption of the leptomeningeal vessels by beta amyloid protein or by direct extension of a cortical-subcortical haemorrhage into the subarachnoid space [10]
   - frequency of CAA related convexity SAH may be under estimated as this has only been recently been described as a distinct disease category [3]
   - often recurs and is potentially a warning of subsequent intracranial haemorrhage [1] (Fig.26-28)
   - CAA related SAH and superficial siderosis can be present without any other haemorrhagic lesions including intracranial haemorrhage and microbleeds [10]
   - clinically, no headache is present and there may be transient focal neurological deficit mimicking a TIA
   - SAH is most frequently seen in the perirolandic region [1] (Fig 2-6)

4. Superficial Siderosis
   - CAA is an increasingly recognized cause of superficial siderosis [1]
   - repeated convexity SAH leads to haemosiderin deposits in the subpial layers of the supratentorial brain, appearing as gyriform hypointensity on gradient echo sequences [10] (Fig.30)
   - superficial siderosis tends to have a supratentorial distribution, often extending beyond the site of the acute bleed, suggesting there has been previous occult convexity subarachnoid haemorrhages[1] (Fig 2).

5. Microinfarctions
   - small round foci of T2 hyperintensity and restricted diffusion in subcortical white matter and cortex [10] (Figs 2-6, 31-32)
6. **CAA inflammation**

- inflammatory CAA arises from an autoimmune response to vascular beta-amyloid deposits resulting in a vasculitis
- clinically there is progressive dementia and seizures
- MRI demonstrates large, confluent areas of T2 hyperintensity predominantly affect the subcortical white matter with some associated cortical involvement (Fig. 33-37)
- lesions tend to be asymmetric and resolve with steroid therapy [2]

7. **Irreversible Leukoaraiosis**

- multiple, relatively symmetrical white matter lesions [10]
- mainly deep white matter with relative sparing of subcortical-U fibers

**OTHER CAUSES OF CONVEXITY SAH**

**Posterior Reversible Encephalopathy Syndrome**

- most commonly associated with pregnancy induced hypertension, eclampsia and severe hypertension
- may also occur in normotensive patients, particularly in the setting of medications such as cyclosporine, after solid organ or allogeneic bone marrow transplantation
- vasogenic oedema involves the subcortical white matter of the parietal, occipital or posterior frontal lobes bilaterally. Less commonly, the cerebellum, basal ganglia, thalamus or brainstem may be affected [4].
- haemorrhages may be seen in 5-17% cases of PRES [1]. These may be convexal or parenchymal in location [4]. Microhaemorrhages (<5mm) may be present.
- haemorrhage is likely to be due to reperfusion lesions or rupture of pial vessels secondary to severe hypertension and impaired cerebral autoregulation [1]

**Cerebral Venous Thrombosis**

- cortical vein or dural venous sinus thrombosis usually affects young adults, mainly women [1]
- clinical presentations are varied and patients may present with headaches, seizures, altered conscious state and signs of raised intracranial pressure [4].
• apart from visualizing the thrombus itself, other findings of CVT include cerebral oedema, parenchymal haemorrhages, infarcts (which may be ischaemic or haemorrhagic) and subarachnoid haemorrhage [4]
• subarachnoid haemorrhage may result from venous hypertension and subsequent rupture of dilated thin walled subarachnoid veins [1]
• subarachnoid haemorrhage in the setting of cerebral venous thrombosis may be convexal in location and usually spares the basal cisterns

Vasculitides and Primary angiitis of CNS

• wide variety of primary and secondary cerebral vasculitides which can cause cerebral haemorrhage including isolated convexity SAH due to inflammation of the wall of blood vessels [1]
• Primary Angiitis of CNS may present with an insidious onset of symptoms
• headaches are common but are not thunderclap in nature
• may be a stepwise deterioration with transient deficits, infarcts and cognitive decline
• on imaging, white matter abnormalities, deep or superficial infarcts of different ages may be seen [6]
• vessel wall enhancement may be seen after contrast administration
• angiography may be normal or demonstrate irregular, eccentric, assymetrical vessel narrowings and dilatations of medium sized arteries [1]

Infective Endocarditis

• SAH complicates 1-2% cases
• this may present with fever, headache, altered mental state and there may be a history of intravenous drug use
• on imaging, embolic infarcts, microhaemorrhages, microabscesses and mycotic aneurysms may be seen
• SAH may result from focal endarteritis or vessel rupture at the site of embolic occlusion or occult septic aneurysm [4]

Moya Moya

• collateral lenticulostriate vessels develop after chronic stenosis /occlusion of the supraclinoid ICA and proximal branches of the circle of Willis resulting in the characteristic "puff of smoke" appearance
• may be congenital or acquired after progressive vascular occlusion from any cause [4]
• most haemorrhages are parenchymal however SAH may occur, which may be basal or convexal [1]
• the basal SAH may result from rupture of associated saccular aneurysms whereas the convexal SAH may be secondary to rupture of fragile transdural and transosseous pial collaterals [4]

**Vascular Malformations**

• pial and dural arteriovenous fistulae typically present with parenchymal haematoma and basal SAH and only rarely with isolated cortical SAH
• there have also been case reports of cavernous haemangiomas which are superficial and leptomeningeal that have caused convexity SAH [1]

**High grade atherosclerotic stenoses**

• similar to Moya Moya, high grade stenoses of intra or extracranial cerebral arteries results in formation of by dilated fragile pial vessels which may potentially rupture and cause convexal SAH [1]

**Other Causes of Isolated Convexity SAH**

• coagulopathies [4]
• migraine
• leptomeningeal metastases - usually rapidly progressive multiple cranial
• nerve paresis and no sudden onset headache [1]

**FIGURES**

**Fig.1.** Non contrast sagittal CT images in a 34 year old after a fall from a roof demonstrates subtle acute frontal convexity SAH.
Fig. 1

References: Monash health - CLAYTON/AU

Fig 2 - 6: Amyloid Angiopathy in a 72 year old male with transient facial and left arm parasthesia. (Fig 2-3) Non contrast axial and sagittal CT imaged demonstrate a convexity SAH involving the right frontal sulci and perirolandic region. This is again demonstrated as sulcal T2 hyperintensity on FLAIR sequences (Fig 4). DWI (Fig 5) show small foci of restricted diffusion in subcortical white matter and cortex in keeping with CAA-related microinfarcts. (Fig 6) Gradient echo T2* sequences demonstrate superficial siderosis extending beyond the site of the acute bleed suggesting there has been previous occult convexity subarachnoid haemorrhages.
Fig. 2

References: Monash health - CLAYTON/AU
Fig. 3

References: Monash health - CLAYTON/AU
Fig. 4

References: Monash health - CLAYTON/AU
Fig. 5

References: Monash health - CLAYTON/AU
Fig. 6

References: Monash health - CLAYTON/AU

Fig. 7 - 10: 46 year old female with RCVS presenting with thunderclap headache. Axial non contrast CT (Fig 7-8) demonstrates bifrontal convexity subarachnoid haemorrhages. The initial MRA (Fig 9) demonstrates vasospasm, best observed in the middle cerebral arteries which is seen to resolve on an MRA (Fig 10) three months later.
Fig. 7

References: Monash health - CLAYTON/AU
Fig. 8

References: Monash health - CLAYTON/AU
Fig. 9

References: Monash health - CLAYTON/AU
Fig. 10

*References:* Monash health - CLAYTON/AU

**Fig. 11-13:** RCVS. Non contrast CT images (Fig 11) in a 42 year old presenting with headache demonstrates left parietal convexity SAH. MRA (Fig 12) demonstrates segmental narrowings of the left middle cerebral artery which resolve on a follow up MRA 3 months later (Fig 13).
Fig. 11

References: Monash health - CLAYTON/AU
Fig. 12

References: Monash health - CLAYTON/AU
Fig. 13

References: Monash health - CLAYTON/AU

Fig. 14 - 17: RCVS. Non contrast CT (Fig 14) in a 46 year old female with headache and limb weakness demonstrates a right frontal convexity SAH. FLAIR sequences (Fig 15 - 16) from an MRI performed 4 days later demonstrates oedema in the parieto-occipital lobes bilaterally and right frontal lobe and diffuse vasospasm (Fig 17), both of which resolved on follow up MRI.
Fig. 14

References: Monash health - CLAYTON/AU
Fig. 15

References: Monash health - CLAYTON/AU
Fig. 16

References: Monash health - CLAYTON/AU
**Fig. 17**

**References:** Monash health - CLAYTON/AU

**Fig. 18-21:** RCVS. Axial and reconstructed MRA images (Fig 18-20) in a 36 year old female 2 weeks post partum with severe headache demonstrates a focal dissection and dissecting aneurysms involving the atlantic segment of the left vertebral artery. There is also diffuse vasospasm of branches of the right middle cerebral artery which resolves spontaneously after 2 months (Fig 21). The vertebral dissection is unchanged.
Fig. 18

References: Monash health - CLAYTON/AU
Fig. 19

References: Monash health - CLAYTON/AU
Fig. 20

References: Monash health - CLAYTON/AU
Fig. 21

References: Monash health - CLAYTON/AU

Fig.22-23: RCVS. MRA images in a 45 year old female performed 5 days after the onset of severe headache demonstrates (Fig 22) diffuse vasospasm, best appreciated in the middle cerebral branches. A repeat MRA (Fig 23) performed 2 months after presentation demonstrates normal caliber vessels.
Fig. 22

References: Monash health - CLAYTON/AU
Fig. 23

References: Monash health - CLAYTON/AU

Fig 24-25: Axial (Fig 24) and coronal (Fig 25) non contrast CT images in a 73 year old with right hemiparesis demonstrates a large acute left frontal subcortical/cortical haematoma likely related to CAA.
Fig. 24

References: Monash health - CLAYTON/AU
Fig. 25

References: Monash health - CLAYTON/AU

Fig. 26-28: Non contrast CT images (Fig 26) in a 75 year old with transient leg paraesthesia demonstrates a right frontal convexity SAH. Gradient echo imaging (Fig 27) demonstrates further areas of superficial siderosis involving the right posterior frontal and parietal sulci, suggesting CAA. The patient represented 2 weeks later with a dense left hemiplegia. A CT study (Fig 28) revealed a large right frontoparietal haematoma. Convexity SAH is potentially a warning of subsequent intracranial haemorrhage.
Fig. 26

References: Monash health - CLAYTON/AU
Fig. 27

References: Monash health - CLAYTON/AU
Fig. 29: Gradient echo T2* sequences in a 75 year old female demonstrate typical the typical pattern of CAA-related microhaemorrhage - lobar, cortical and subcortical haemorrhagic foci with sparing of the deep grey matter.
Fig. 29

References: Monash health - CLAYTON/AU

Fig 30: Axial gradient echo T2* sequences in an 80 year old with confusion demonstrates gyriform hypointensity in the left frontal lobe due to previous unrecognized convexity SAH in the context of CAA.
Fig. 30

References: Monash health - CLAYTON/AU

Fig 31-32: Axial DWI images demonstrate small foci of restricted diffusion in subcortical white matter and cortex in keeping with CAA-related microinfarcts in an 85 year old female with left facial and hand tingling. Multiple microhaemorrhages (not shown) were also seen.
Fig. 31

References: Monash health - CLAYTON/AU
Fig. 32

References: Monash health - CLAYTON/AU

Fig 33 - 37: Inflammatory Amyloid. Non contrast CT (Fig 33) and FLAIR (Fig 34) sequences in an 83 year old with confusion and right homonymous hemianopia demonstrating a confluent area of hypoattenuation/ T2 hyperintensity predominantly affect the left posterior parietal subcortical white matter with associated cortical involvement representing probable inflammatory amyloid. There was no restricted diffusion on MRI (not shown). Several T2 hyperintense white matter lesions are scattered throughout the subcortical and periventricular white matter. GE sequence (Fig 36)
demonstrates multiple microhaemorrhages. FLAIR sequences (Fig 37) from a follow up study after steroid treatment demonstrates resolution of the left parietal T2 hyperintensity.

Fig. 33

References: Monash health - CLAYTON/AU
Fig. 34

References: Monash health - CLAYTON/AU
Fig. 35

References: Monash health - CLAYTON/AU
Fig. 36

References: Monash health - CLAYTON/AU
Fig. 37

References: Monash health - CLAYTON/AU
Conclusion

Convexity SAH is an increasingly recognized manifestation of cerebral amyloid angioplaty and reversible vasoconstriction syndrome. An awareness of these conditions and of other causes of convexity SAH will optimise consequent imaging pathways and management decisions.
References


