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Sylvian Fissure Lipoma Associated with Middle Cerebral Artery Aneurysm – Report of a Rare Case Highlighting Imaging Pitfalls

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Authors' contributions

This work was carried out in collaboration between all authors led by author AKK and first drafted by author NS. All authors read and approved the final manuscript.

Case Study

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ABSTRACT

Aim: We describe a 60 year old lady who had a Sylvian fissure lipoma associated with an underlying middle cerebral artery saccular aneurysm which is a rare association and describe its imaging features.
Presentation of Case: Due to its rarity and MRI appearances simulating blood products, this case offered an initial diagnostic dilemma to the reporting radiologists, when an incidental lesion like lipoma adjoining an aneurysm was initially misinterpreted as blood products.
Discussion and Conclusion: This case highlights potential pitfalls in imaging interpretation even with a newer neuroimaging technique to accurately identify the nature of the lesion that can lead to misdiagnosis with the potential of inappropriate clinical management, at least in some cases.

Keywords: Aneurysm; lipoma; susceptibility-weighted imaging.

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1. INTRODUCTION

Intracranial lipomas are common incidental findings on brain MR scans. However, intracranial aneurysms can be incidental but are often significant, especially, if there is evidence of bleed that indicates rupture. These two entities are rarely seen together, when, due to similarities of lipomas and certain blood products on MRI, an incidental aneurysm may be viewed as a ruptured aneurysm that can potentially raise false alarm and inappropriate management. This case highlights these similarities and intends to raise awareness of this co-incidence and the potential limitation of various MRI techniques in this regard.

2. PRESENTATION OF CASE

Our patient is a 60-year old female, previously fit and healthy, presenting to the acute medical unit with a week's history of bilateral hand and toes paraesthesia associated with mid-thoracic back pain following a short flu-like illness. There was no headache. Clinical examination revealed a left-sided lower motor neuron facial palsy with generalised areflexia, though the rest of her neurological examination was normal. A clinical diagnosis of Miller-Fisher variant of Gillian-Barre syndrome was made, pending specific antibody results, however, subsequent anti-GQ1b antibodies were negative. During her neurological work-up, she underwent an MRI brain scan. This revealed the unexpected finding of a left middle cerebral artery saccular aneurysm surrounded by an area of high signal on T1, T2 and FLAIR initially thought to represent subarachnoid haemorrhage appearing as a localised haematoma with late subacute blood products (Fig. 1).

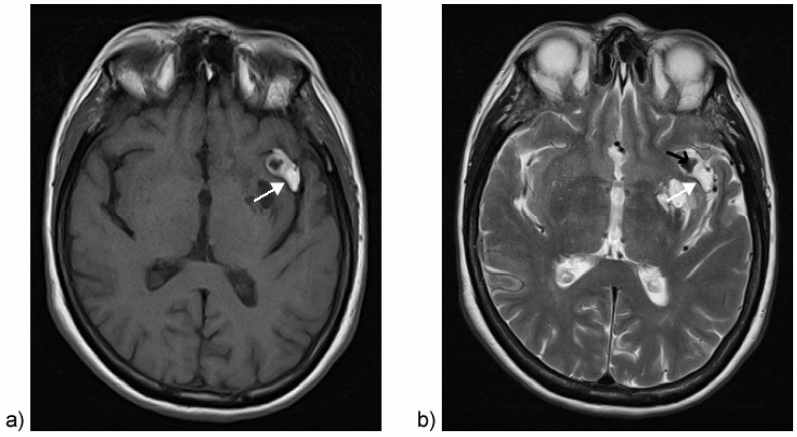

Fig. 1. T1 weighted (a) and T2 weighted (b) axial non-contrast MR brain demonstrating a lesion in the left Sylvian fissure demonstrating high T1 and T2 signal intensity (white arrow) surrounding a central flow void (black arrow in b) corresponding to a middle cerebral artery aneurysm. The signal characteristics of lipoma on these images are similar to that displayed by late subacute blood products containing extracellular methaemoglobin

Susceptibility weighted imaging (SWI) was also performed that showed lower signal on the periphery of the lesion, somewhat resembling blood products, although the center of the lesion remained bright (Fig. 2). Time of flight (ToF) angiogram and diffusion-weighted

images at b0 (Fig. 3) also demonstrated features usually seen in blood products. She subsequently underwent CT brain with CT cerebral angiography for further assessment of the left MCA aneurysm, which revealed that the abnormal signal seen on MRI in the left Sylvian fissure was due to a lipoma rather than haematoma (Fig. 4). The patient was, however, asymptomatic for the aneurysm as well as lipoma and was treated conservatively.

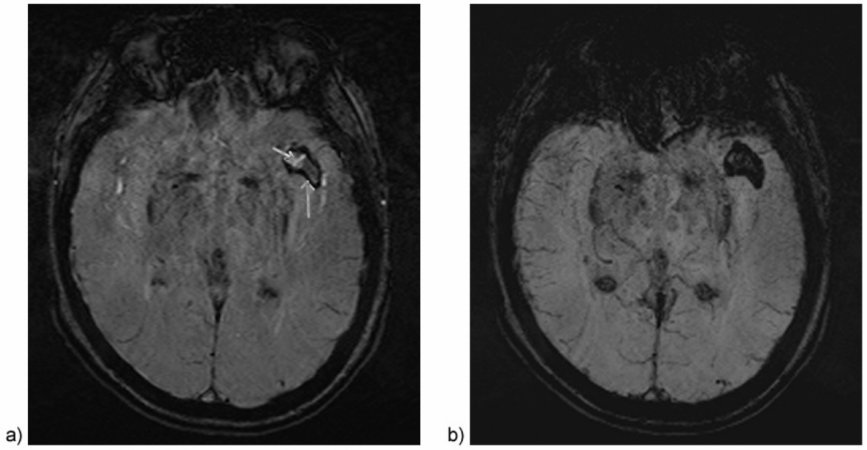

Fig. 2. Susceptibility weighted imaging – SWI images thin slice (a) and minimum intensity projection (MIP) (b) through the lipoma. A rim of low signal is seen around lipoma (long arrow in a) better seen on MIP images that mimics blood products and hence easily falsely interpreted as haematoma in the context of an associated middle cerebral artery aneurysm (short arrow in a)

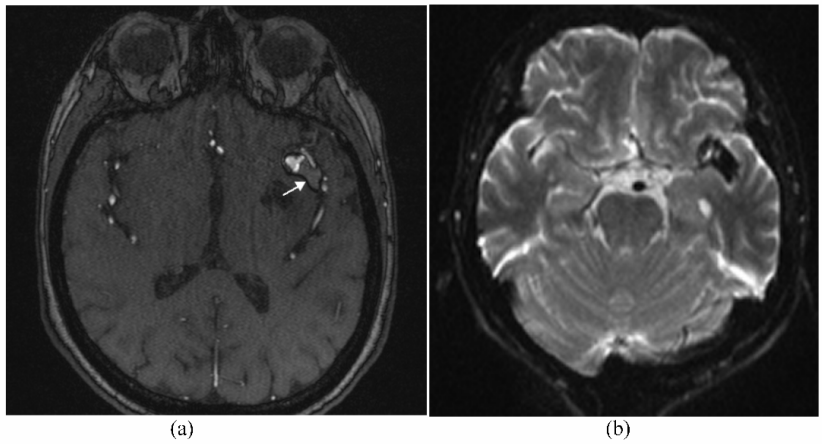

Fig. 3. (a)-Intracranial lipoma demonstrating high signal intensity on Time-of-Flight (ToF) MR angiography sequence due to associated T1 shortening, a feature also shared by subacute haematomas. Dark margin posteriorly around the lipoma is due to chemical shift artifact (arrow). (b) – b0 sequence diffuse weighted image (DWI) shows lipoma as low signal due to fat suppression in these sequences. Several blood products can show low signal on these images due to susceptibility effects and these can also cause difficulty in differentiation

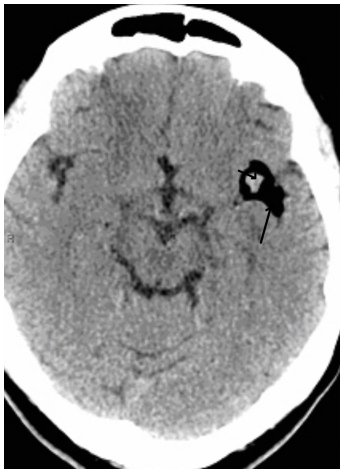

Fig. 4. Axial CT non-contrast of the brain demonstrating that the lesion around the aneurysm (short arrow) is a very well defined area of fat attenuation consistent with lipoma (long arrow) rather than blood products. No rim calcification was present on CT in relation to the lipoma

3. DISCUSSION

Intracranial (IC) lipomas constitute only 0.1 – 1.3% of all brain tumours of which 47% are seen in the corpus callosum, 20% are found within the ambient, quadrigeminal, chiasmatic and cerebellopontine angle cisterns and only 5% seen within the Sylvian fissure [1,2]. Verga was the first person to describe an IC lipoma in 1929 [3] and hypothesised that IC lipomas are rare congenital malformations resulting from abnormal persistence and maldifferentiation of meninx primitiva during the development of the subarachnoid cisterns. Truwit and Barkovich [1] support this hypothesis. Intracranial lipomas are almost always located in the subarachnoid space since they are formed from the meninx.

Most IC lipomas are asymptomatic and picked up incidentally during brain imaging for unrelated reasons. When symptomatic they usual relate to location of the lipoma and may include persistent headaches, cranial nerve palsies or neuralgia, psychomotor retardation or seizures. The latter are more commonly seen in association with Sylvian fissure lipomas due to their location in close proximity to the temporal lobes [4,5].

IC lipomas are frequently associated with brain malformations of varying degrees [1,6] with intracranial vessels and nerves coursing through 36% of lesions [1]. The most common midline associated anomaly is that of corpus callosum agenesis or dysgenesis [7], though another example is when seen in the intercerebellar fissure associated with vermis hypoplasia.

Angiographic studies have demonstrated abnormalities in contiguous cerebral arteries such as fusiform aneurysms, which are not thought to be rare. Eldevik and Gabrielsen [8] identified fusiform aneurysms in 19 of 22 cases with lipoma of the corpus callosum in their review of the previous literature. While lipomas of Sylvian fissure are uncommon, associated saccular aneurysms in the middle cerebral artery bifurcation are even rarer. While the exact

incidence of this association is unknown, we could find only 4 cases previously described on the literature search, making the current case as the 5th documented case [2,9,10]. However, there have been other reports of aneurysm separate from the site of lipoma [11].

The aetiology of the association of lipomas and aneurysms is obscure [10]. While intracranial lipomas are believed to have a malformative origin due to abnormal differentiation of the meninx primitiva, aneurysms may arise due to a congenital structural deficiency of the blood vessel occurring at the same time as the formation of a lipoma [10]. It is also possible that the lipoma may itself have resulted in degeneration of cerebral arterial wall. It has been suggested that cerebrovascular smooth muscle may receive its nutrition from subarachnoid cerebrospinal fluid via diffusion as the cerebral arteries do not have vasa vasorum, this nutrition could be adversely affected by adjoining lipoma. It has also been suggested that lipomas might inhibit the growth of arterial smooth muscle by secreting some factors, resulting in weakening the arterial wall that may result in aneurysm formation due to hemodynamic stress [12].

Rupture of intracranial aneurysms more often result in generalised subarachnoid haemorrhage but these can also result in localised haematomas in the Sylvian fissure. It is this subgroup that may be relevant with regards to similarity with Sylvian fissure lipomas. Other complications of aneurysms are possible though less common, such as pressure effects and embolism causing stroke. Ischemic stroke secondary to embolization from arterial aneurysm was observed only in 2 of 70 cases in a clinical study of ischemic stroke of unusual etiology [13].

On CT scans, lipomas typically show density that of pure fat ranging between -40HU and -100HU (Fig. 4) and the imaging appearances have no similarity with blood products [2]. Calcification may be present though is more commonly seen in interhemispheric lesions and less commonly seen elsewhere.

Lipomas share similar MRI features with late subacute blood products containing extracellular methaemoglobin [4] and appear hyperintense on T1- and T2- weighted images (Fig. 1). Unlike late subacute haematomas, lipomas are hypointense on fat suppressed images and exhibit chemical shift artefacts. However, fat suppressed sequences are not routinely performed, unless presence of fat is suspected in the first instance. Susceptibility-Weighted Imaging (SWI) is a gradient echo technique that generates strong negative contrast in regions where magnetic susceptibility changes and is highly sensitive to accumulations of paramagnetic substances such as blood products and calcium. The appearance of lipomas on SWI has not been well described in literature and only been recently described by Lingegowda et al. [14] for the first time, who have shown lipomas to have low peripheral signal on SWI, as in our patient (Fig. 2) although none of their cases were in sylvian fissure. They have attributed it to microscopic mineralization or chemical shift. We agree that it is probably due to microscopic calcification or the susceptibility gradient across the lipid—tissue boundary. In the case presented here, it is likely to be the latter, as no discernible calcium was observed on CT (Fig. 4) although subtle calcium, undetectable on CT due to averaging with fat may be present. This may have important clinical implications when MRI and not CT is performed when it is coupled with the fact that it is more probable to have blood products associated with an aneurysm. The SWI sequence, routinely performed in such situations, may further add to the suspicion of blood. It can be argued that there may have been an episode of bleed in the past resulting in these appearances. While theoretically, such a possibility cannot be excluded, there was no evidence from the patient's history to suggest that. Moreover, the low signal was limited to

the vicinity of lipoma and not present anywhere else, making such a possibility rather unlikely.

The T1-weighted Time-of-Flight (TOF) angiography sequence, commonly performed in patients with aneurysms, would show lipomas and late subacute blood products as hyperintense (Fig. 3). It was noted by Kemmling et al. [15] that intracranial lipoma in close proximity to a cerebral aneurysm can easily be mistaken for a partially thrombosed aneurysm. They advocate the use of an out-of-phase echo time to produce the “India-ink” artefact the periphery of lipid-containing lesions to help avoid such a potential diagnostic pitfall.

Another common sequence performed in MRI is a Diffusion Weighted Imaging (DWI) sequence, which generates an apparent diffusion coefficient (ADC) map. The appearances of blood products on DWI have been debated in literature with certain controversies and somewhat different appearances and ADC values are described in various stages [16]. Both late subacute blood products and lipomas appear dark on ADC, which does not help to differentiate the two. The b0 DWI (no diffusion weighting applied) are low-resolution echo-planar images with fat-suppressed T2-weighting, where lipomas appear hypointense. The late subacute blood products can appear hyperintense due to T2 relaxation but several blood products are known to be dark on these images due to susceptibility effects (Fig. 3), though less sensitive than T2* images [17]. In certain instances, while it may be possible to resolve the issue of whether an abnormality is lipoma or not, these do not offer a definite way of differentiating blood products and lipomas and these may not be reviewed in the light of evidence from SWI pointing towards blood products.

Therefore when presented with an unusual condition of fat surrounding an aneurysm, the reporting radiologist may be overwhelmed by the appearances of SWI, corroborated by time of flight angiogram and overlook subtle features like chemical shift artefact, raise a false alarm of subarachnoid haemorrhage and seek urgent neurosurgical attention before realising the error. It may result in considerable distress to the patient and, if not identified early, possible inappropriate management. It is important as some neurosurgical centres may not perform a CT angiogram and rather perform a digital subtraction angiography with a view to further assess for coiling, and the diagnosis would remain unknown. It is important to stress that hypointensity seen on SWI does not automatically point towards the presence of blood products in every case. SWI is essentially “Susceptibility” weighted image and not “blood” weighted image in that context. Since SWI is a relatively new technique, radiologists across the world are still in the process of building experience in its appearances in different conditions and limited literature is available.

4. CONCLUSION

We have presented a rare case of Sylvian fissure lipoma associated with saccular aneurysm of middle cerebral artery and the appearances on various sequences including SWI. It is important to realize that most MRI sequences including DWI, TOF and SWI sequences are not necessarily helpful in this situation as these tend to show similarity in signal characteristics between blood products and fat and lipoma can be misinterpreted as blood products, particularly as there is an associated aneurysm. The clinical history should be properly considered and MRI images appropriately interpreted for signal, shape and location, with careful assessment of subtle features such as chemical shift artefact. High-resolution fat-suppressed sequences should be performed or old CT scans should be reviewed if there is doubt about the correct diagnosis. Unless the radiologists are aware of this possible

association and have more experience in use of SWI, the appearances can be mistaken for a ruptured aneurysm with subacute blood products with potential to cause patient distress, unnecessary urgent neurosurgical attention and possibly inappropriate management.

CONSENT

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

ETHICAL APPROVAL

Not applicable.

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COMPETING INTERESTS

Authors declare that no competing interests exist.

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