Susceptibility Artifacts in Ruptured Intracranial Dermoid Cysts: A Poorly Understood but Important Phenomenon

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Key words: intracranial dermoid, rupture, SWI, chemical shift artifact, blooming artifact

SUMMARY – *Rupture of intracranial dermoid cyst is a rare event. The classical imaging feature is described as a fat-containing lesion with tiny fat droplets in the subarachnoid or ventricular system. The lesion and the fat droplets show susceptibility blooming artifact on susceptibility-weighted images (SWI). Knowledge of this fact is important because these lesions mimic the blooming artifact of haemorrhage on SWI. The cause of the susceptibility artifact in intracranial dermoids has not been reported in the literature to date. We describe two cases of ruptured intracranial dermoids in the basifrontal region and review the clinical and imaging features and possible causes of susceptibility artifacts in intracranial dermoid cysts.*

Introduction

Intracranial dermoid cysts are slow-growing benign lesions lined by keratinized squamous epithelium with ectodermal elements like hair, sebaceous, sweat glands, teeth, nails and fat in varying proportions.

Rupture of intracranial dermoid cyst is a rare event. Imaging with CT/MRI shows a fat-containing extra-axial lesion with fat droplets in the subarachnoid/ventricular system. This can lead to clinical symptoms ranging from headache and seizures to serious complications like chemical meningitis, vasospasm and cerebral infarction. The lesion as well as the fat droplets show susceptibility blooming artifacts on susceptibility-weighted images (SWI) mimicking haemorrhage.

The cause of blooming artifacts in intracranial fat-containing lesions like lipomas remains speculative and has not been described in intracranial dermoids. Two cases of ruptured intracranial dermoids in the basifrontal region are described in this report with a review of clinical, imaging features and possible causes of susceptibility artifacts in intracranial dermoids.

Case 1

A 48-year-old man presented to the emergency room with two episodes of new onset seizure since morning. Seizures were of complex partial type involving the upper limbs. The patient also complained of dull headache for many months. On examination the higher mental functions were normal and there were no motor, sensory or visual disturbances. MRI showed a well-circumscribed extra-axial mass lesion in the midline basifrontal region located anterior to the optic chiasm. The lesion showed a mixed hypointense and hyperintense signal on T1 and hyperintense signal on T2-weighted images with complete homogeneous suppression of the signal on fat-saturated T1 and FLAIR images [Figure 1A-D]. Multiple small T1 hyperintense fat droplets were seen in the subarachnoid CSF spaces along the cortical sulci and sylvian fissures with complete signal suppression on fat-saturated FLAIR images [Figure 1A,C,D]. On SWI the lesion appeared hypointense with blooming artifacts most prominent along its peripheral margin. The fat droplets in the subarachnoid space also appear hypointense showing complete blooming [Fig-

ure 1E,F]. CT scan showed a mass of heterogeneous density with areas of fatty attenuation and peripheral calcification with disseminated fat attenuation droplets in the subarachnoid

space [Figure 1G,H]. A diagnosis of midline basifrontal dermoid cyst with rupture into the subarachnoid CSF spaces was suggested. The patient was operated with total tumour resec-

Figure 1 A 48-year-old man with ruptured intracranial dermoid presenting with seizures. A) T1-weighted axial non-fat-saturated MRI shows an extra-axial mass lesion in the midline basifrontal region with heterogeneous hyperintense signal. B) The mass is hyperintense on coronal T2-weighted images with curvilinear low intensity areas within. C) Multiple small T1 hyperintense fat droplets are seen along the subarachnoid CSF spaces in sylvian fissures on T1-weighted axial non-fat-saturated MRI. D) Complete homogeneous suppression of the mass as well as the fat droplets on axial fat-saturated FLAIR images. E,F) On SWI the lesion and the fat droplets in sylvian fissures appear hypointense with blooming artifacts. Blooming is predominantly peripheral in the lesion. G,H) Corresponding contrast CT scan in axial and coronal planes shows a mass of heterogeneous density with areas of fatty attenuation and disseminated fat droplets in the subarachnoid space. There were also areas of peripheral and central calcification. The areas of central calcification correspond to areas of hypointensity seen on coronal T2-weighted MRI (B).

Figure 2 A 36-year-old man with ruptured intracranial dermoid presenting with headache. A) T1-weighted axial non-fat-saturated MRI shows an extra-axial mass in the midline basifrontal region with a heterogeneous hyperintense signal. B,C) The mass is hyperintense on sagittal T2-weighted images and shows complete homogeneous suppression on sagittal fat-saturated T1-weighted images. D,E) Multiple small T1 hyperintense fat droplets are seen along the subarachnoid CSF spaces in sylvian fissures on T1-weighted axial non-fat-saturated MRI. F) These show complete suppression on fat-saturated FLAIR images. G,H) On SWI the lesion and the fat droplets in sylvian fissures appears hypointense with complete blooming artifacts. Complete blooming is seen in the lesion as opposed to previous case which may be due to its smaller size.

tion. Histopathology showed keratinized squamous epithelium lining the lesion with fat and areas of calcification.

Case 2

A 36-year-old man presented to the outpatient clinic with complaints of a dull boring type of headache for one month. MRI revealed an extra-axial mass lesion in the midline basifrontal region. The lesion had similar signal intensity characteristics as in the previous case appearing heterogeneously hyperintense on T1 and hyperintense on T2-weighted images with complete homogeneous suppression of the signal on fat-saturated T1 images [Figure 2A-C]. Multiple small T1 hyperintense fat droplets were seen in the subarachnoid CSF spaces in the sylvian fissures which also showed complete fat suppression [Figure 2D-F]. On SWI the lesion showed peripheral blooming artifacts while the fat droplets in the subarachnoid space showed complete blooming [Figure 2G,H].

Discussion

Intracranial dermoid cysts are rare non-neoplastic slow-growing masses comprising <1 % (0.04-0.6 %) of all intracranial tumours¹. They are derived from inclusion of ectodermal-derived cells during neural tube closure around three to five weeks of embryogenesis and represent congenital ectodermal inclusion cysts². These cystic masses are thick-walled and lined by keratinized squamous epithelium with ectodermal elements like hair, sebaceous, sweat glands, teeth, nails and contain fat in varying proportions. Intracranial dermoids are slightly more common in males than females. These lesions are more common in the infratentorial compartment usually occurring in the midline either in the cavity of fourth ventricle or vermis. Supratentorial dermoids are commonly located near the skull base close to the midline in sellar/parasellar and front nasal regions^{2,3}. There is often associated dermal sinus with posterior fossa dermoid cysts^{4,5}. Malignant transformation into squamous cell carcinoma has been described².

Dermoid cysts are not true neoplasms and enlarge by desquamation and accumulation of sebaceous secretions by dermal elements². Symptoms are because of mass effect on the

adjacent intracranial structures. Mean duration of symptoms has been reported from three months to 6.87 years³. Posterior fossa dermoid cysts typically present in the first decade due to mass effect on the fourth ventricle and hydrocephalus, while supratentorial dermoid cysts usually present in the second and third decades of life with visual disturbances or headache.

Rupture of intracranial dermoid cysts is a rare event. In a recent report by Liu JK, rupture represents 0.18% of all new CNS tumours operated on during a 12-year period in their institution⁶. Rupture is typically spontaneous although rupture secondary to closed head trauma has been reported^{7,8}. The pathophysiology of spontaneous rupture is not well explained, but it has been related to glandular secretions caused by age-dependent hormones⁹, head movements and brain pulsations³. Following rupture, keratin and cholesterol breakdown products disseminate into the ventricles and subarachnoid space with aseptic chemical meningitis which is reported in approximately 7% of cases¹⁰. Chemical meningitis can present with a wide variety of symptoms including headache (32.6%), seizures (26.5%), cerebral ischaemia secondary to vasospasm with infarction, sensory and/or motor hemisindrome (16.3%) and chronic granulomatous arachnoiditis^{11,12,13}. Hydrocephalus may occur secondary to mass effect, occlusion of ventricular foramina by the intraventricular fat or chronic ventriculitis due to repeated leakage of cystic contents into the subarachnoid or ventricular system¹⁴. No correlation has been found between the distribution of fat and clinical symptoms¹⁵.

On computed tomography dermoid cysts typically appear well-circumscribed hypodense fat attenuation lesions although density varies depending on the proportion of fat, hair, calcification and epidermal debris. The cyst wall may show calcification in 20% of cases¹⁶⁻¹⁸.

MRI shows dermoid cysts of hyperintense signal on T1-weighted images due to high lipid content and variable hypointense to hyperintense signal on T2-weighted images¹⁹. Fine curvilinear hypointense elements on T2-weighted images represent hair. Diffusion-weighted images show pronounced hyperintensity with ADC similar to brain parenchyma²⁰. A chemical shift artifact of the first kind may be seen due to miss registration of signal in the frequency encoding direction¹⁶. FLAIR images show a hyperintense signal compared to CSF which allows differentiation from arachnoid cysts. The presence of high signal intensity fat droplets

on T1-weighted images disseminated along the subarachnoid and ventricular space is diagnostic of dermoid cyst rupture. Fat-fluid levels in the non-dependent portions of the ventricles may also be seen. Dermoid cysts rarely enhance with the administration of contrast and pial and ventricular ependymal enhancement may be seen following chemical meningitis.

On susceptibility-weighted imaging dermoid cyst and the fat droplets appear hypointense and show blooming artifacts. The blooming artifact was predominantly along the peripheral margins of the dermoid cysts seen as a thin dark rim in both cases with some areas of blooming artifact also seen within the lesions. The small fat globules also showed complete blooming in both cases. Knowledge of this fact is consequential, since this lesion mimics the MRI characteristics of haematoma on SWI. Both haematoma and dermoid cyst appear hyperintense on T1-weighted images and show blooming on SWI images. Fat-suppressed T1-weighted imaging will help to differentiate both the lesions as dermoid cyst shows fat suppression appearing hypointense whereas haematoma remains hyperintense on fat-suppressed T1-weighted images. Similarly, fat droplets in the subarachnoid space may mimic haemorrhage on SWI as they show complete blooming. CT scan is helpful showing characteristic fat attenuation within the lesion as well as the fat droplets. A few reports have described the characteristics of fat-containing intracranial lesions on susceptibility-weighted imaging²¹⁻²⁴. However, to the best of our knowledge there is no report in the literature describing the SWI characteristics of ruptured intracranial dermoid cyst.

The cause of blooming artifacts on SWI in intracranial fat-containing lesions has not been extensively studied and established. Mehemed et al., based on their work using SWI in a lard-water phantom and intracranial lipomas, suggested chemical shift artifacts of the second kind and the phase shift contrast of the high-pass filtered-phase images between fat and water rather than magnetic susceptibility as a cause of the prominent low signal intensity peripheral rim seen in the lipoma on SWI²¹. Firstly, chemical shift artifacts of the second kind are seen in gradient echo sequences (SWI is a 3D gradient echo sequence) when there is a proportion of both fat and water signal in a pixel. At a particular TE when fat and water protons are out of phase their signals cancel each other with signal intensity loss along the

pixels containing both fat and water protons manifesting as sharply defined dark rim. Pixels that contain solely fat do not lose their signal because there is no water signal to cancel. Secondly, the phase-mask component of SWI allows fat to be out of phase from water. In this setting a high phase shift occurs at the fat-water interface on the high-pass filtered-phase images and seen as a low signal intensity rim on susceptibility-weighted images.

In a case series of SWI in intracranial lipomas, the authors postulated microscopic calcifications and mineralization due to foci of osseous metaplasia as a cause for blooming of fat on SWI²². No significant contribution of macroscopic calcification was observed as all the lipomas showed blooming, irrespective of whether or not calcification was identified on CT scan. They also proposed that chemical shift artifact made a significant contribution to the blooming artifact in lipomas on susceptibility-weighted images. Two patterns of blooming were observed – peripheral and central, with peripheral blooming common in larger lipomas and central blooming in smaller lipomas [mean short diameter 4 mm]. It was assumed that the central blooming in small lipomas was due to merging of peripheral blooming from the opposite walls. This hypothesis in part can explain blooming of fat droplets in the subarachnoid space with merging of the peripheral blooming to give an appearance of complete blooming.

Schembri et al. reported sylvian fissure lipoma associated with middle cerebral artery aneurysm which showed peripheral blooming on SWI and could be misinterpreted as haematoma associated with ruptured aneurysm²³. They also hypothesized a susceptibility gradient across the lipid-tissue boundary as a cause of blooming as suggested by Lingegowda et al.²². Sudhir et al. also reported blooming of fat globules on SWI within an arachnoid cyst²⁴.

Though there are no direct reports, these principles can be applied to intracranial dermoids, the chemical shift artifacts of the second kind and blooming related to calcification and ossification explaining the susceptibility artifacts.

Treatment of these lesions is primarily surgical and only indicated in patients with significant mass effect or rupture. The goal is complete excision of the lesion with removal of the tumour capsule²⁵. Recurrence is rare following total surgical excision, but may occur with retained portions of tumour wall following surgical excision.

Conclusions

Rupture of intracranial dermoid is a rare but serious event.

The lesion can be confused with haemorrhage on SWI and correct identification is es-

sential for proper and urgent management. The cause of blooming in intracranial dermoids is likely related to chemical shift artifacts of the second kind due to fat content as well as a contribution from microscopic calcification and ossification.

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