

## Traumatic rupture of an intracranial dermoid cyst

Raksha Ramlakhan, BMedSc, MBBCh; and Sally Candy, BSc, HED, MBChB, FCRad (D) (SA)

Intracranial dermoid cysts are congenital tumors of ectodermal origin. Rupture of these cysts can occur spontaneously, but rupture in association with trauma is reported infrequently. The diagnosis of rupture is made by the presence of lipid (cholesterol) droplets in the subarachnoid spaces and ventricles. Nonenhanced CT of the head demonstrates multiple foci of low attenuation that correspond with hyperintense signal on T1-weighted MRI. We present a case of an adult patient with rupture of an intracranial dermoid cyst, precipitated by minor trauma.

### Case report

A 19-year-old male presented to the trauma unit following minor trauma (a low-speed collision with the side mirror of a stationary car while riding a bicycle). He did not complain of headaches; however, he reported pain in his neck, right arm, and leg. On further questioning, he admitted to using illicit substances (methamphetamines, marijuana, mandrax) the night before. On physical examination, he was found to have no external injuries but was combative and assessed to have a GCS of 14/15. A secondary survey revealed no neurological deficits. The only abnormal finding on his initial blood workup was an elevated white cell count of 14.66. Noncontrast CT scan of the head (Fig. 1a) showed multiple locules of low density, with a distribution in the suprasellar and prepontine cisterns and in the lateral and third ventricles. The largest locule was found in the left paracavernous area, measuring 17mm x 8mm, with an attenuation of -80HU (consistent with fat) (Fig. 1b). Anterior to this was a 10mm focus of clumped calcification (Fig. 1c). Limited T1-weighted MRI of the head (Figs. 2a and 2b) demonstrated a corresponding high signal in all locations, confirming the presence of chole-

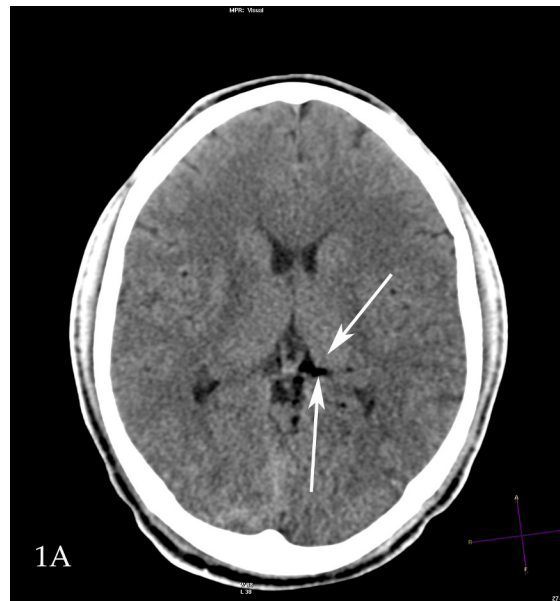


Fig. 1A. Noncontrast CT of the brain shows multiple small foci of low density (HU -80), distributed in the lateral ventricles and basal cisterns (white arrows).

**Citation:** Ramlakhan R, Candy S, Traumatic rupture of an intracranial dermoid cyst. *Radiology Case Reports*. (Online) 2015;10(1);1053

**Copyright:** © 2015 The Authors. This is an open-access article distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivs 2.5 License, which permits reproduction and distribution, provided the original work is properly cited. Commercial use and derivative works are not permitted.

Dr. Ramlakhan is Registrar/Resident, and Dr. Candy is Principal Specialist/Consultant, both at Groote Schuur Hospital (Division of Radiology), Cape Town, South Africa. Contact Dr. Ramlakhan at [docrakshar@gmail.com](mailto:docrakshar@gmail.com).

DOI: 10.2484/rcr.v10i1.1053

terol. The CT and MRI findings were therefore pathognomonic of a ruptured intracranial dermoid cyst.

The patient was admitted to the neurosurgical unit for observation. His hospital admission was complicated by an increased agitated state, likely secondary to drug withdrawal and by an episode of acute bronchospasm, which responded to nebulization and intravenous corticosteroids. The chest radiograph did not demonstrate features of non-

## Traumatic rupture of an intracranial dermoid cyst

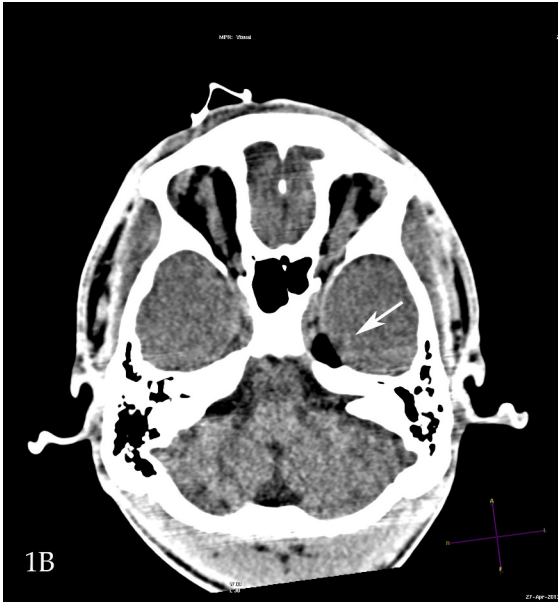


Fig. 1B. Ovoid left paracavernous lesion (white arrow) represents dermoid cyst.

cardiogenic pulmonary oedema. The neurosurgical team elected to manage the patient conservatively.

There was no further neurological deterioration, and the patient was discharged after four days, with a plan to follow

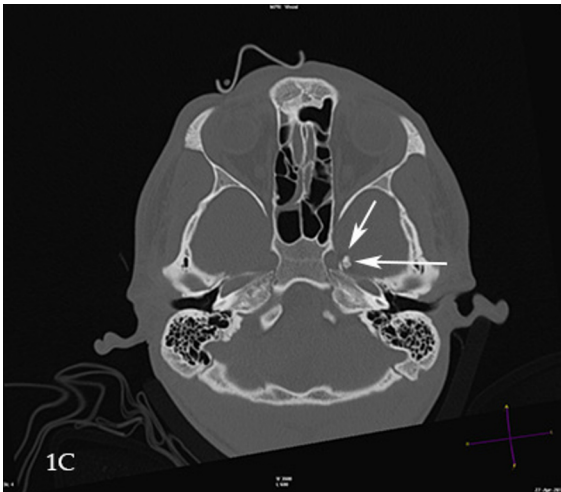


Fig. 1C. On bone settings, the left paracavernous lesion (white arrows) demonstrates fat and clumped calcification.

up at the neurosurgical outpatients' clinic. The patient deferred followup, to this date.

### Discussion

Dermoid cysts are rare, congenital cystic tumors that compose < 0.5% of primary intracranial tumors (1). The

cysts occur mostly near the midline, at either end of the neuraxis, and are found in the sellar, parasellar region, frontonasal region, and posterior fossa (2).

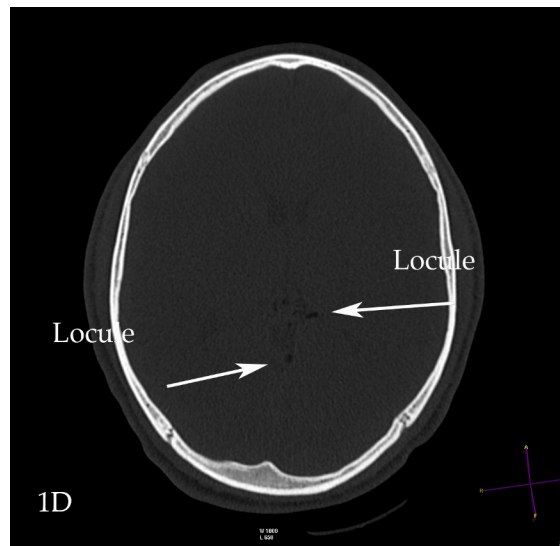


Fig 1D. Bony window confirms that the low-density locules in the basal cisterns (black arrows) represent fat and not air.

They arise from the incorporation of ectodermal cells, due to defective separation of the neuroectoderm at the time of neural tube closure. This process usually occurs during the 3rd to 5th week of embryogenesis (1, 3, 4). The cysts contain ectodermal derivatives that may include

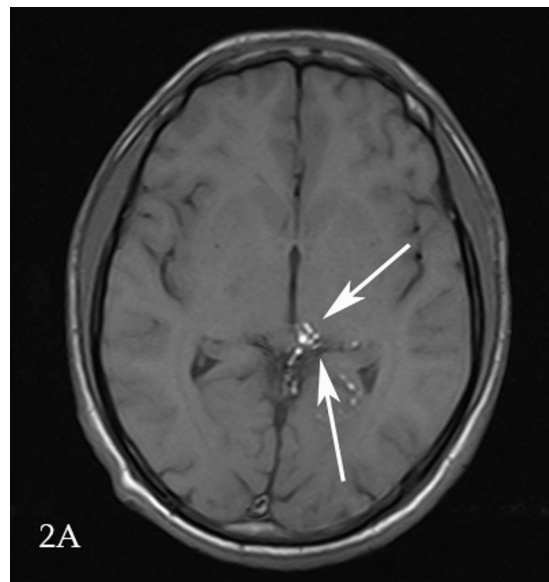


Fig. 2A. T1-weighted axial MRI of the brain demonstrates multifocal high signal in a subarachnoid distribution (white arrows) confirming the presence of cholesterol secondary to rupture.

## Traumatic rupture of an intracranial dermoid cyst

squamous epithelium; sebaceous, apocrine or sweat glands; hair follicles; and even sometimes teeth (4, 5). The capsule consists of simple epithelium supported by collagen, and contains plaques of calcification (1). The cyst contents are due to the secretion of desquamated epithelium, and sebaceous secretions (5). Enlargement of the cysts is due to the accumulation of the above products.

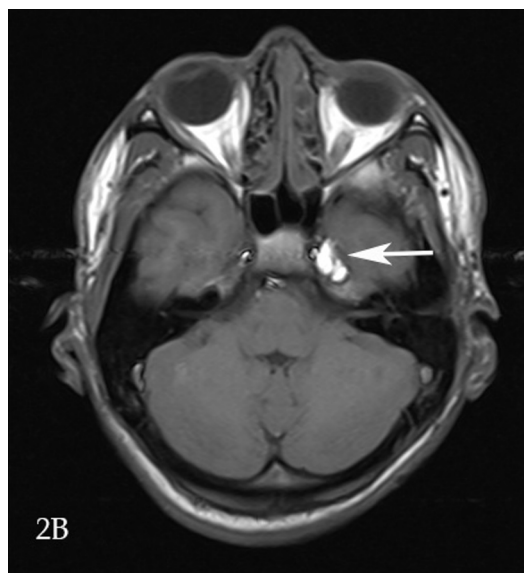


Fig. 2B. T1-weighted MRI of the brain shows the left paracavernous dermoid cyst (white arrow).

These lesions are often asymptomatic unless they rupture, which may occur spontaneously, or in relation to (often minor) trauma. Dissemination of the cholesterol debris from the cyst contents into the subarachnoid and intraventricular spaces, may cause chemical meningitis (2, 3). This can be further complicated by obstructive hydrocephalus, vasospasm, infarction, and even death. The neurological symptoms are ascribed either to mass effect on adjacent intracranial structures, or as a complication of the rupture (4, 5). Postrupture neurologic symptoms include headaches, seizures, nausea, visual loss, neck pain, meningeal signs, and an altered level of consciousness (2, 3).

The CT imaging findings include well-circumscribed, hypodense lesions (Fig. 1b), with the imaging characteristics of fat, due to the predominant lipid content (liquid cholesterol) (2). After initial rupture, fat droplets may be distributed in the subarachnoid spaces and ventricles (Figs. 1a, 1d, and 2a).

The cyst may have a heterogeneous appearance, if there is a mixture of hair, calcification, and epidermal debris (3). Dermoid cysts do not demonstrate enhancement with the administration of contrast (2).

MRI is superior to CT in characterizing the extent of lipid dissemination, and for operative planning (2). The cysts appear hyperintense on T1-weighted imaging (Fig. 2b). The presence of lipid droplets in the subarachnoid cisterns, sulci, and ventricles is pathognomonic of a ruptured

cyst (Fig. 2a). On T2-weighted imaging, dermoid cysts have heterogeneous signal intensity (1). Depending on the different components, they may appear hypo- or hyperintense (1). If the internal fat content is low, the cyst may have fluid signal. If there is associated chemical meningitis, there may be pial and ventricular ependymal enhancement after the administration of contrast (1).

The differential diagnosis of fat-containing intracranial cysts includes epidermoid cysts, arachnoid cysts, cranio-pharyngomas, teratomas, and lipomas (1).

Management involves treatment of the complications, including intravenous steroids for the chemical meningitis, and ventriculo-peritoneal shunting for hydrocephalus. The goal is to aim for complete surgical resection, while weighing against the risk of causing damage to adjacent structures (2). The recurrence rate following resection is low; however, a cyst that is incompletely resected will require routine followup. The ruptured, disseminated fat lesions usually remain stable in position, without consequences (2).

### Conclusion

Intracranial dermoid cysts are rare. Only a handful of cases in the literature describe a relationship between trauma and ruptured cysts. The patient in our case fortunately had an uncomplicated recovery. Early recognition of these pathognomonic imaging findings by radiologists is crucial to the management of these cysts in the event of life-threatening complications.

### Acknowledgment

Thanks to Lyle Eyssen for assistance with the images.

### References

1. Osborn AG, Preece MT. Intracranial cysts: radiologic-pathologic correlation and imaging approach. *Radiology*. 2006;239(3):650-64. [<http://dx.doi.org/10.1148/radiol.2393050823>] [[PubMed](#)]
2. Esquenazi Y, Kerr K, Bhattacharjee MB, Tandon N. Traumatic rupture of an intracranial dermoid cyst: Case report and literature review. *Surgical neurology international*. 2013;4:80. [[PubMed](#)]
3. Park SK, Cho KG. Recurrent intracranial dermoid cyst after subtotal removal of traumatic rupture. *Clinical neurology and neurosurgery*. 2012;114(4):421-4. [<http://dx.doi.org/10.1016/j.clineuro.2011.11.006>] [[PubMed](#)]
4. Ray MJ, Barnett DW, Snipes GJ, Layton KF, Opatowsky MJ. Ruptured intracranial dermoid cyst. *Proceedings*. 2012;25(1):23-5. [[PubMed](#)]
5. Skovrlj B, Mascitelli JR, Steinberger JM, Weiss N. Progressive visual loss following rupture of an intracranial dermoid cyst. *Journal of clinical neuroscience : official journal of the Neurosurgical Society of Australasia*. 2014;21(1):159-61. [<http://dx.doi.org/10.1016/j.jocn.2006.10.028>] [[PubMed](#)]