Ruptured intracranial dermoid cyst: a case report

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Abstract

Intracranial dermoid cysts are congenital, usually nonmalignant lesions with an incidence of 0.5% of all intracranial tumors. They tend to occur in the midline sellar, parasellar, or frontonasal regions. Although their nature is benign, dermoid cysts have a high morbidity and mortality risk, especially when rupture occurs. A 40 year old woman presented with head injury after she experienced sudden loss of consciousness. She had a history of headache, loss of consciousness; her past medical history was not remarkable. The patient had no complaints of nausea, vomiting, or seizures. Vital signs were stable, neurologic deficit was not identified. Computed tomography (CT) and magnetic resonance imaging (MRI) showed right temporobasal zone with fat droplets within right fissure Sylvii and interhemispheric fissure indicating a rupture of a dermoid cyst. Craniotomy and cyst resection were done, and diagnosis was confirmed with pathological examination following surgery. After surgery the patient did not recover. Cerebral ischemia from chemical meningitis was fatal for our patient. Headache as a symptom has many causes. It is rarely due to chemical meningitis arising from a ruptured dermoid cyst. This case report illustrated the importance of investigating a cause of the headache, CT and MRI being diagnostic methods. In this way, mortality as well as morbidity from complications such as chemical arachnoiditis can be significantly reduced if imaging is done early in these patients.

Keywords: dermoid, intracranial, rupture, chemical meningitis

Introduction

Intracranial dermoid cysts are rare, congenital, usually benign lesions. They are usually detected accidentally but often become symptomatic after rupture. The presence of fat droplets in the subarachnoid space and ventricular system is typical finding in computed tomography (CT) and magnetic resonance imaging (MRI). Rupture leads to aseptic chemical meningitis, vasospasm, cerebral ischemia and coma (1, 2). Chemical meningitis may lead to transient cerebral ischemia secondary to vasospasm with complicating infarction and the death of the patient (3), as happened in our case. In this report, we present CT and MRI findings of a ruptured intracranial dermoid cyst with postoperative complications. Cerebral ischemia due to chemical meningitis was fatal for our patient.

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Case Report

A 40 year old female presented with a head injury after she experienced sudden loss of consciousness. She had a history of headache and loss of consciousness, but other than that her past medical history was not remarkable. The patient had no complaints of nausea, vomiting or seizures. Vital signs were within normal limits and neurologic deficit was not evident. CT scan of the head showed right temporobasal, well defined lobular hypodense zone with calx density zones within it (Figure 1). CT also showed tiny, partially confluent, low attenuation areas of fat density within right fissure Sylvii and interhemispheric fissure (Figure 2). The appearance of fat droplets usually follows rupture of a dermoid cyst. Magnetic resonance imaging was requested, which revealed right temporobasal cystic lesion returning a high signal intensity on T1-weighted imaging (T1W) (Figure 3) and heterogeneous signal intensity on T2-weighted imaging (T2W) (Figure 4). The appearance of fat intensity areas within right

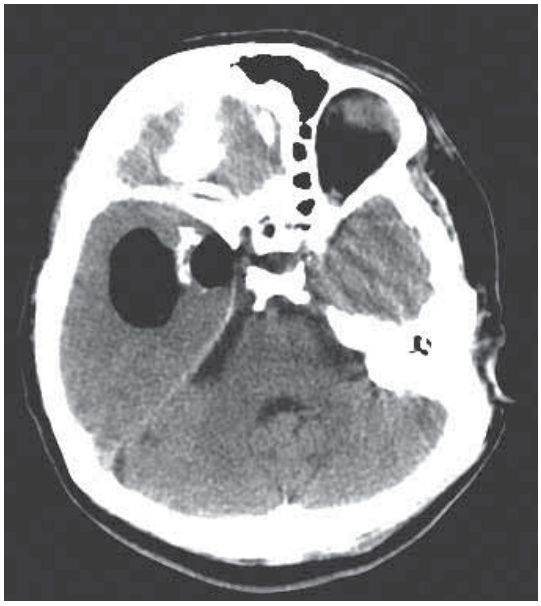

FIGURE 1. Brain CT of dermoid cyst in right temporabasal region.

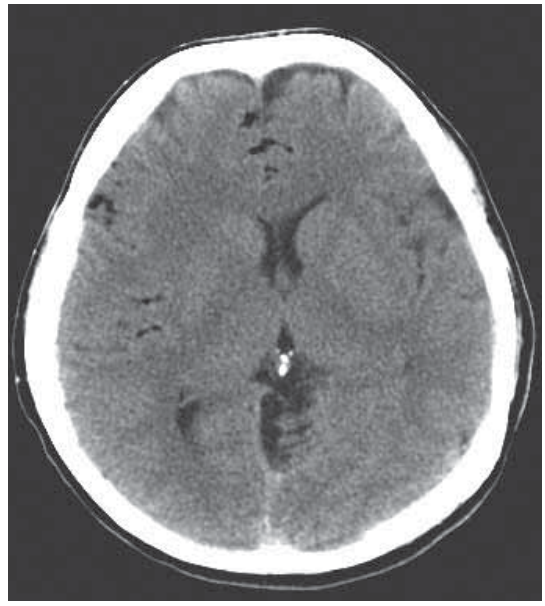

FIGURE 2. Brain CT showing fat droplets within right fissure Sylvii and interhemispheric fissure.

fissure Sylvii and interhemispheric fissure was also apparent. No contrast enhancement was seen. MRI scan confirmed CT diagnosis of a ruptured dermoid cyst, and the patient was referred for a neurosurgical opinion. The decision was made to conduct a craniotomy and cyst resection. Pathological examination, following surgery, determined that histological type of the mass was dermoid cyst. After surgery the patient remained in coma, so CT was performed, and multiple hypodense zones corresponding to ischemic vascular lesions were observed in parietal and occipital area, as well as in the basal ganglia. The final outcome of the patient was fatal.

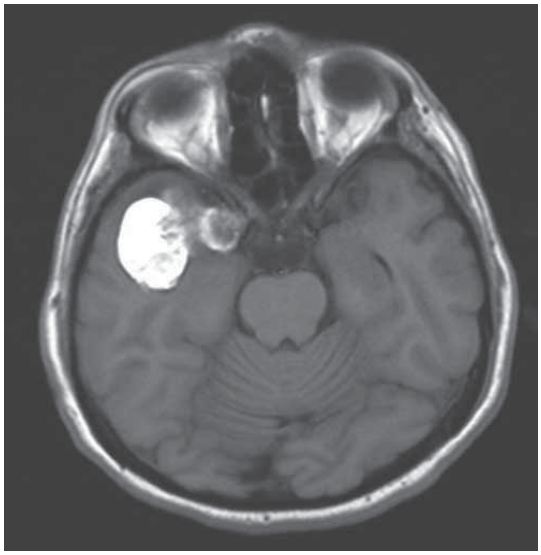

FIGURE 3. Brain T1-weighted MRI of dermoid cyst.

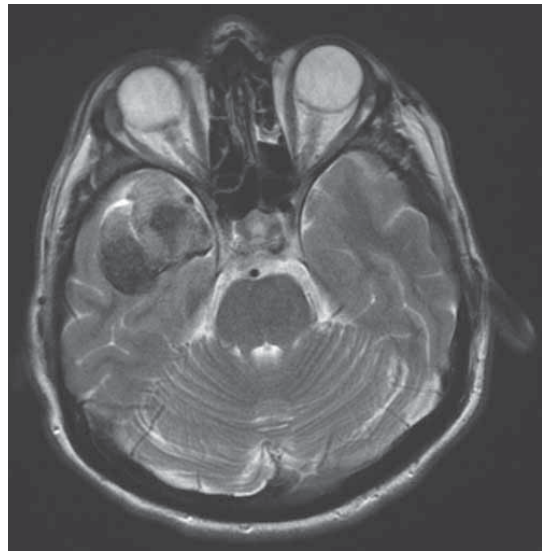

FIGURE 4. Brain T2-weighted MRI of dermoid cyst.

Discussion

Intracranial dermoid cysts are congenital ectodermal inclusion cysts. They are usually nonmalignant lesions with an incidence of 0.5% of all intracranial tumors. They tend to occur in the midline sellar, parasellar, or frontonasal regions. They emerge from the inclusion of ectodermal primitive pluripotent cells due to defects in neural tube closure (gestational weeks 3-5) (1). The capsule of dermoid cysts consists of simple epithelium supported by collagen. It contains a dense liquid composed of cholesterol, keratin, lipid metabolites, calcifications, hair and teeth (1-3). They are detected accidentally, but also may give symptoms of seizures and headache, and rarely olfactory delusions. Although their nature is benign and development is slow, dermoid cysts have a high morbidity and mortality risk, especially when rupture occurs (4). They can rupture and release lipid droplets in the subarachnoid space and ventricular system (1, 3). Rupture is usually spontaneous, even though in some cases is due to surgery or head injury (4, 5). The rupture of dermoid cyst and the presence of lipid in the subarachnoid space and ventricular system may cause chemical meningitis, hydrocephalus, vasospasm and cerebral ischemia (6). Clinical symptoms of acute rupture are headache, nausea, vomiting, vertigo, vision problems, aseptic chemical meningitis, hemiplegia, mental changes, and coma (4). Aseptic chemical meningitis is rare complication and is found only in 7% of patients (7). Chemical meningitis may lead to transient cerebral ischemia, secondary to vasospasm with complicating infarction and the death of the patient (3-5). Symptoms usually do not occur at the time of rupture, but may be delayed from 3 months to 6.5 years after rupture, because the irritative effects of the spilled contents require time to develop (3, 8). CT and MRI imaging are diagnostic methods. On CT scans these lesions have internal density characteristics consistent with fat (negative Hounsfield units) and their wall is usually calcified. Occasionally the wall can partially enhance after the administration of CT-iodinated contrast material (3). On MRI scans, dermoids are hyperintense on T1-weighted imaging due to high lipid content, and heterogeneous on T2-weighted imaging because of different components in the cyst such as bones, cartilage and calcifications (3, 4). If the internal fat content is relatively low, the lesion reveals cerebrospinal fluid–like signal intensity. In such cases, fluid attenuation inversion recovery (FLAIR) is useful, because the fat appears hyperintense (bright), while fluid signal is suppressed (dark). When a dermoid cyst ruptures, fat droplets appear hypodense on CT or T1 hyperintense on MRI within ventricular system and/or subarachnoid space (3). Differential diagnosis includes epidermoid cyst, teratomas, lipomas, cystic craniopharyngiomas and occasionally arachnoid cysts (3, 4, 8). Intracranial epidermoids are 4 - 9 times more common than intracranial dermoids (8). Epidermoid cyst and dermoid cyst both usually appear as sharply defined, low density or intensity mass lesions with no contrast enhancement on CT and MRI. Nevertheless the location of epidermoid is more variable than that of dermoid cyst and shows greater deviation from midline (8). Teratomas can be confused with dermoid because they may contain fat, but teratomas cause contrast medium enhancement. Lipomas are similar to dermoids as they both have a hyperintense appearance in T1W and T2W, but they are differentiated from dermoids by their smooth borders and typical midline localization (4). Cystic craniopharyngiomas and arachnoid cyst can be differentiated from dermoids based on signal characteristics and demonstration of fat in dermoids and using FLAIR sequences (8). Patients typically do well after operative intervention. Recurrence is rare but is more common if there are retained portions of the tumor wall. Rare reports describe malignant transformation of dermoid cysts into squamous cell carcinoma (1,3). Prognosis of patients with ruptured intracranial dermoids depends on the spread of the contents and the time period after rupture (5).

Conclusion

Headache as a symptom has many causes. Loss of consciousness as well. Headache due to dermoid cyst or due to chemical meningitis arising from a ruptured dermoid cyst is not so common. This case report illustrated the importance of investigating a cause of the headache, CT and MRI being diagnostic methods. In this way, mortal-

ity as well as morbidity from complications such as chemical meningitis can be significantly reduced if imaging is done early in these patients.

Competing Interests

Authors declare no conflict of interest related to this study.

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