Journal of Clinical Neuroscience 19 (2012) 352–359

Contents lists available at SciVerse ScienceDirect

Journal of Clinical Neuroscience

journal homepage: www.elsevier.com/locate/jocn

Review

Intracranial neuroenteric cysts: A concise review including an illustrative patient

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ARTICLE INFO

Article history:
Received 1 October 2011
Accepted 9 October 2011

Keywords:
Endodermal cyst
Enterogenous cyst
Epidermal cyst
Neurenteric cyst
Neuroenteric cyst
Surgery
Surgical treatment

ABSTRACT

Neuroenteric cysts (NC) are rare, benign lesions lined by mucin-secreting cuboidal or columnar epithelium of an intestinal or respiratory type. They are regarded as ectopic endodermal cysts, and tend to be found in the spine rather than an intracranial location. Advances in neuroimaging have led to an increased frequency of diagnosis of NC, especially as an incidental finding, although such cysts may be confused radiologically with other lesions such as epidermoid and arachnoid cysts. We undertook a PubMed search of the literature using the search terms "neuroenteric cyst" and its many pseudonyms, including "endodermal cyst", "enterogenous cyst", "neurenteric cyst", "epithelial cyst", "intestinome", "teratomatous cyst", "gastrocytoma", and also "enterogenic", "foregut", "respiratory", and "bronchogenic cyst". Only reports in English and those containing histopathologically-confirmed NC were considered for this review. In total, 140 patients with intracranial NC were found, including the patient reported in the present review. This review describes the classification, epidemiology, embryology, clinical presentation, radiology, histopathology, and surgical treatment of NC, and includes an illustrative patient.

1. Introduction

Neuroenteric cysts (NC) are rare congenital entities arising from displaced elements of the alimentary canal and are most frequently encountered in the posterior mediastinal region. Although they can occur in any region of the central nervous system (CNS), where they account for 0.01% of CNS tumours,[1–3] NC are more commonly found in the spine and represent 0.3% to 0.5% of spinal tumours.[4,5] When located in the cerebellopontine angle (CPA), NC are often misdiagnosed as arachnoid or epidermoid cysts.[6–10] Owing to their disputed pathogenesis, historically the nomenclature for these lesions has varied, and has included terms such as "teratomatous" by Kubie and Fulton[11] in 1928, "intestinome" by Puusepp[12] in 1934, "neurenteric" by Holcomb and Matson[13] in 1954, "gastrocytoma" by Knight et al.[14] in 1955, and "enterogenous" by Scoville et al.[15] in 1963. Other terms used to describe them include enterogenic, foregut, respiratory, and bronchogenic cysts,[10,13] due in part to observed associations with bronchopulmonary foregut malformations.[2] However, the use of the term neuroenteric cyst has gained increasing support in the neuropathology literature,[1,16] despite the World Health Organization's adoption of the term "enterogenous" in the revised 1993 classification of tumours of the CNS.[17]

To our knowledge, 140 intracranial NC, confirmed by histology, including the patient presented in this review, have been reported since 1952.[1,3–8,18–100] All cysts described were symptomatic and required surgery and among those that were partially excised, a symptomatic recurrence occurred in approximately one-third.[76] Recent advances in neuroimaging have led to an increased frequency of diagnosis of these rare lesions. This article reviews the literature pertaining to NC and, in particular, clarifies their diagnosis and management, and includes an illustrative patient.

2. Literature search

The PubMed search engine of the National Library of Medicine and National Institutes of Health (http://www.ncbi.nlm.nih.gov/pubmed/) was utilised to conduct an Internet Medline literature search based on the following search terms: "neuroenteric cyst", "endodermal cyst", "enterogenous cyst", "neurenteric cyst", "epithelial cyst", "intestinome", "teratomatous cyst", "gastrocytoma", and also "enterogenic", "foregut", "respiratory", and "bronchogenic cyst". Only reports in English involving histopathologically-confirmed intracranial NC were considered, no date limitations were imposed in the search criteria, and spinal NC were excluded. Cases from bibliographies of retrieved references were included. In total, 139 reports of intracranial NC were found.

3. Epidemiology

NC are rare benign neoplasms most commonly located within the spine. Intracranial NC are particularly rare and only comprise

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doi:10.1016/j.jocn.2011.10.005

Table 1
Reports of intracranial neurenteric cysts in the literature [a,1,3–8,18–100]

	N	Percentage [b]
Median age (years)	34	
Age range (years)	0–78	
Male	61	51
Female	48	49
Location		
Posterior fossa	47	34
Craniocervical	61	44
Supratentorial	19	14
Other	12	9
Lateralisation		
Midline	87	63
Lateral	41	29
Not specified	11	8

[a] Including the illustrative patient in this review.
[b] Percentage does not add to 100% due to rounding.

of 10% to 17.9% of all NC.[96] Including the patient in this review, only 140 patients with intracranial NC have been reported since 1952.[1–3,5,22,25,26,29,35,46,49,54,77–79,83,96,100–105] Due to the paucity of patients it is difficult to make an assessment of the true incidence or prevalence of NC. Two retrospective studies in China have suggested that intracranial NC make up approximately 0.15% to 0.35% of all intracranial neoplasms,[96,106] but these finding were based on retrospective audits conducted at specialty neurosurgical centres and covered surgically referred patients only and not the general population. The true incidence remains unknown.

The patient demographics and features of the intracranial NC in the published reports is shown in Table 1. Our review of these reports has demonstrated a median age at presentation of 34 years (range 0–78 years). These reports suggest that intracranial NC occur more often in the adult population in comparison to intraspinal NC, which are more common in the paediatric setting (0–10 years of age).[2,4] To date, only 18 patients with intracranial NC have been reported in patients younger than 14 years.[4,27,31,33,39,56,64,77,79,91,93,94,99,100] The youngest patient reported to date is that of a prenatal diagnosis of a large NC occupying the sella and parasellar areas extending to both the right anterior and middle cranial fossa identified at 26 weeks' gestation on routine ultrasonography.[27]

Of the published reports, there does not appear to be any significant gender preponderance. This is in contrast to previously published series which have suggested either male or female predominance.[77,95,96,107] The age at presentation of intracranial NC is later than that of intraspinal cysts, possibly due to the lower tolerance of local mass effect on the spinal cord. A total of 61 (44%) NC in reported patients were located within the posterior fossa with 47 (34%) extending from the posterior fossa to the cervicomedullary junction. Only 19 (14%) of these reports described supratentorially located NC.[1,3,20,21,27,29,32,37,65,66,77,84,87,88,93] This was in keeping with current knowledge of intracranial NC. Multiple NC in both the supratentorial and infratentorial compartments have been documented rarely.[1,3,29,108,109]

Midline-located NC were described in 63% of patients and 29% were located laterally, with reports of 11 (8%) patients not providing sufficient details on cyst location. Although NC are most commonly located in the midline, 61% of those located laterally were found on the left side and 39% on the right side. No bilateral localisation of NC has been reported to our knowledge.

4. Clinical features

The most common presentations are related to the local mass effect of the NC and its specific location. NC located within the posterior fossa present most frequently with vertigo or imbalance (50%), hearing loss (14%) and tinnitus (11%). Less common symptoms have been associated with specific cranial nerve deficits including blurred vision (4%), lower cranial nerve deficits (2%), and sensory loss or trigeminal neuralgia (3%). These presentations appear to be more common in NC located within the CPA.[1,4,7,29,53,76,110] Two small series of posterior fossa NC have demonstrated such symptoms.[22,96] Other specific cranial nerve deficits have been reported secondary to localised NC compressing cranial nerves such as the oculomotor nerve (diplopia),[75] and optic nerve (visual failure).[80]

In the rare supratentorial NC, presenting symptoms and signs are most commonly related to increased intracranial pressure. These patients commonly present with headache, nausea and vomiting. Patients may also present with focal and generalised seizures and associated focal motor and sensory deficits depending on the cyst location,[3,20,21,29,88] or with symptomatic hydrocephalus in patients with intraventricular cysts.[77,93] Some reports have documented recurrent presentations with fever and meningism related to aseptic meningitis. This has been postulated to occur secondary to intermittent leakage or a patent fistula causing drainage of cyst contents into the subarachnoid space leading to an inflammatory reaction.[22,103]

Patients with NC often present with symptoms that been have recurrent over several years. This is presumably secondary to the slow growth of these lesions.[10,29,46,95,96,111–114] This growth may be related to ongoing secretions from the epithelial cells,[14,49,53] osmotic retention of cerebrospinal fluid (CSF)[10,25,115] or intracystic haemorrhage.[12,76,77,88,90,116] The mechanism of transient symptom regression is unknown, but may involve intermittent compression of neural structures through the mass effect of the NC followed by spontaneous leakage, relieving the compression and subsequent later reaccumulation of cyst contents.[76] This phenomenon has been reported in patients with spinal NC.[26,49,78,79,102]

5. Definition and classification

NC have been defined as benign cystic lesions that are lined by mucin-secreting epithelium similar to that found in the gastrointestinal tract.[117] However, this definition may not be entirely correct as publications documenting malignant transformation of NC exist, suggesting this entity may not be as benign as originally thought.[3,32,78,97]

Defining and classifying NC has been problematic due to the lack of consistency in terminology regarding NC and the paucity of reports. There are two classifications of intraspinal NC in use, each relying on histological findings and topography.[96,118] The first was developed in 1976 by Wilkins et al. – a classification system for intraspinal NC (Types A–C) based on histological features:[107]

- Type A cysts have an epithelial lining composed of pseudostratified cuboidal or columnar epithelial cells mimicking the respiratory or gastrointestinal epithelium.
- Type B cysts in addition to the above structure, may be arranged in complex invaginations and have associated glands producing mucinous or serous fluid. These cysts may be composed of a range of associated tissue including smooth muscle, striated muscle, fat, cartilage, bone, elastic fibers, lymphoid tissue, nerve fibers, ganglion cells, or Vater Pacini corpuscles.
- Type C cysts, in addition to the findings in Type B, may be associated with glial elements such as ependymal cells of the wall.

The second classification system for spinal NC is based on the location of the lesion within the spinal column.[4,5] The spectrum of anomalies according to this classification includes alimentary

diverticula, post-enteric prevertebral cysts, vertebral body malformations with intraspinal cyst, through to spina bifida and dermal sinus.[13,16,40] There is no such classification tool available for intracranial NC due to their rarity. As the number of reports continues to increase, a classification tool may be developed to guide the diagnosis and management of this condition.

6. Embryology

The precise aetiology and pathogenesis of NC remains unclear, with several hypotheses proposed. The most common hypothesis is that NC develop from failure of the dissolution of the neurenteric canal between the foregut or the respiratory buds and the notochord.[1,7,53,76] During the third and fourth weeks of embryogenesis, a transient communication, the neurenteric canal, exists between the primitive neural tube (ectoderm) and the enteric tube (endoderm). During the formation of the notochord the embryonic ectoderm and endoderm separate.[29] If the endodermal cells become separated from the normal gut and emerge intra-axially, a NC develops (Fig. 1).[96] NC are composed of an epithelial lining of endodermal origin along the neural axis, a finding which supports the theory that they arise from ectopic remnants of the primitive endoderm caught in the developing neuroectoderm during embryogenesis.[29,40,53] This theory is supported by intracranial NC occurring within the posterior fossa and their predominantly anterior midline location as the rostral closure of the notochord by the mesenchyme that subsequently forms the clivus.[1]

The developmental disturbance at the origin the NC is also reflected by the reported occurrence of vertebral body anomalies,[4,5,13,16,116,119,120] although spinal NC without associated vertebral or other congenital abnormalities have been described.[121] Additionally, intra-abdominal or intrathoracic cysts are frequently[4,5] encountered in association with NC.

Although this theory supports the formation of NC in both the posterior fossa and the spinal cord, it does not explain the origin of supratentorial NC. A further theory has proposed that supratentorial NC may originate from a remnant of Seessel's pouch, an outpouching of the oropharyngeal membrane, composed of endoderm that gives rise to the adenohypophysis.[112] This unsupported hypothesis further suggests that both Rathke's cleft cyst and colloid cysts may represent a form of NC that arises from Seessel's pouch. This theory, however, does not adequately explain the origin of laterally located supratentorial NC, which remain poorly understood.[1,3,20,21,29,32,37,66,77,80,82,84,87,88]

Other hypotheses have also been proposed to explain the formation of NC and have suggested adhesions causing an abnormal development of the notochord,[122] abnormal duplication or split-notochord,[123,124] disorders of gastrulation,[125] and persistence of the neurenteric canal.[126] No current theory can explain entirely the formation of NC.

7. Radiological features

Although NC are more commonly located within the spinal cord, they may develop intracranially.[5] Most intracranial NC develop within the posterior fossa and account for approximately 70% to 90% of all intracranial NC.[1,3,5,7,22,53,76,88,95,101,105,110] In a small retrospective case series of posterior fossa NC, it was reported that these lesions are located most commonly within the midline and anterior to the brain stem (23.1–51%).[96] The second most common location of NC in the posterior fossa is the CPA (17–51%)[96]; however, these lesions can also occur within the fourth ventricle.[6]

Supratentorial NC are even rarer and have been reported in such locations as suprasellar,[103,112] parasellar,[22,127] along the optic nerve [80] or the oculomotor nerve,[68] superior orbital fissure,[128] or as intraparenchymal or durally based lesions,[21,105,110,115,129]

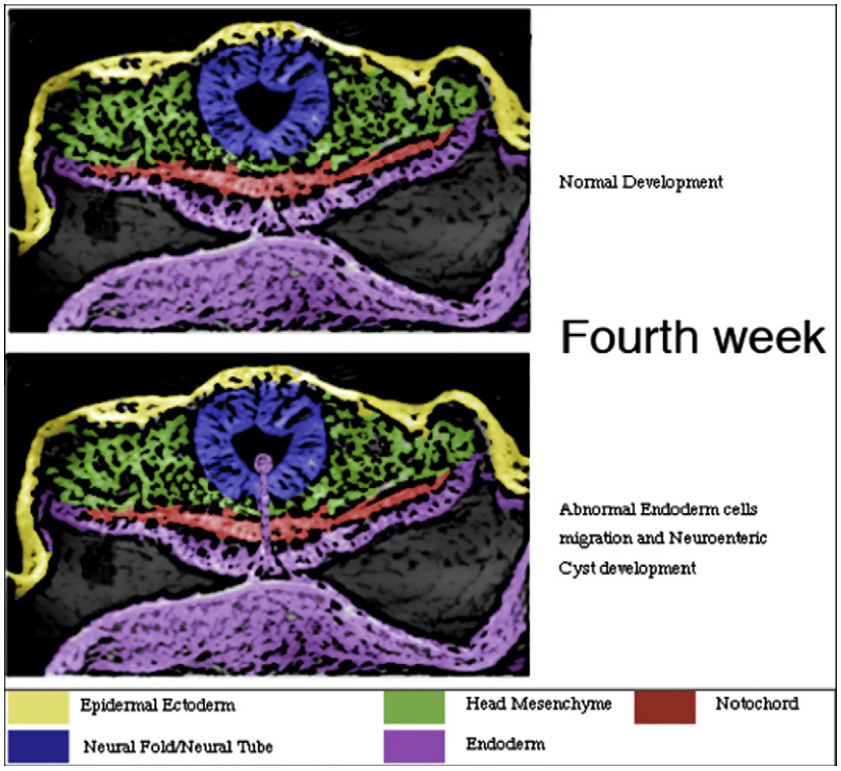

Fig. 1. Embryology of neuroenteric cysts (NC). (Upper) Normal development; and (lower) abnormal development showing abnormal migration of endoderm cells during the fourth week of embryogenesis, which is postulated to underlie NC formation. Blue = neural fold/neural tube, green = head mesenchyme, purple = endoderm, red = notochord, yellow–green = epidermal ectoderm. (Figure modified from[144] Rao MS, Jacobson M. *Developmental neurobiology*. 4th ed. New York, NY: Springer-Verlag 2005, reproduced with kind permission of Springer Science+Business Media B.V.)

intraventricular lesions,[6,71,89,90,111] and those at the craniocervical junction.[35,55,64] Given that NC can appear both histologically and radiologically similar to Rathke's cleft cyst and colloid cysts, location can provide an important distinguishing feature.

The radiographic findings of NC vary significantly with both CT scans and MRI. This has been ascribed to variability of cyst protein content.[7,9,77,83,130–133] NC are usually demonstrated as hypodense lesions with no contrast enhancement using CT; however, they can occasionally appear as hyperdense lesions that are compartmentalised with some enhancement of the cyst wall.[22,77] NC have appeared isodense with surrounding brain parenchyma on CT scan, posing a challenge in diagnosing the cyst radiologically.[7,9] Intracranial NC, unlike their spine counterparts, only very rarely demonstrate associated bony abnormalities with one report demonstrating bony erosion of the petrous temporal bone adjacent to the cyst.[22,77,96]

MRI is the radiological investigation-of-choice for NC. Most NC are proteinaceous and have a T1-weighted signal that ranges from isointense to slightly hyperintense to CSF and classically appears hyperintense on T2-weighted images.[77] NC appear as hyperintense on fluid-attenuated inversion recovery (FLAIR) sequences with an absence of surrounding oedema and usually demonstrate a mild restriction on diffusion-weighted imaging (DWI).[77] Enhancement on MRI is rare, similar to CT.[77,83,132,133] To our knowledge, NC associated with calcification have been described in two patients only.[77,88] Other variants of NC exist such as a solid nodule with strong enhancement from a xanthogranulomatous reaction that simulated cholesterol granuloma[15,83] or a posterior rim enhancement at the interface between the cyst and the adjacent neural parenchyma.[9,77,95,112] Associated hydrocephalus can also occur secondary to the location and mass effect of the NC.[6,76,95,103,111]

8. Radiological differential diagnosis

Although the imaging characteristics and location of the cystic lesion may assist in forming a preoperative diagnosis of NC, other intracranial cystic lesions must also be considered in the differential diagnosis. These lesions include arachnoid cysts,[134] epidermoid or dermoid cysts,[135] choroidal cysts,[73,103] colloid cysts,[111] Rathke's cleft cyst,[73,103] ependymal cysts,[73,103] cysts of parasitic or larval origin,[136,137] and cystic tumours (both primary and metastatic).[29,103] Due to the lack of specific imaging features, NC may be misdiagnosed as any of the above lesions preoperatively. Lesion location can assist with the consideration of an NC and cystic lesions located in the posterior fossa ventral to the brainstem should be considered suspicious for a NC. NC, epidermoid cysts, and dermoid cysts are often highly variable in location, radiological appearance, and manner of growth, making definitive radiological diagnosis difficult.

Arachnoid cysts have similar morphology to NC and generally the content of the cyst is consistent with CSF appearance on both MRI and CT scan, a finding that is common to NC as well. An arachnoid cyst, unlike an NC, commonly has a suppressed signal with MRI FLAIR sequence and does not restrict on DWI.[7,9,64,77,96] This finding of cystic content with the same signal characteristics as CSF is also helpful in distinguishing NC from choroidal cysts and ependymal cysts.[103]

Epidermoid and dermoid cysts may differ from NC in terms of location as they are more likely to be situated in lateral locations such as the CPA, but the NC is more likely to be midline.[96] However, in our review, we found it not uncommon for NC to occur laterally within the CPA.[5,46,69,76,90,96] Epidermoid and dermoid cysts usually appear more lobulated compared to the regular rounded appearance of NC and may demonstrate calcification in 10% to 15% of patients.[88] Dermoid cysts usually demonstrate hyperintensity on both T1-weighted and T2-weighted MRI, with epidermoid cysts typically demonstrating hypointensity on T1-weighted MRI, and hyperintensity on T2-weighted MRI, and with a degree of mild contrast enhancement. Both epidermoid and dermoid cysts demonstrate a moderate to quite significant restriction on DWI which may assist in differentiating these lesions from NC.[113] However, diffusion restriction has been noted in histologically proven NC.[77] In addition to these findings, the rare "white" epidermoid cyst may appear very similar to NC given its hyperintense appearance on T1-weighted MRI and in midline lesions, they may be indistinguishable from NC.[130]

Endodermal derived cysts such as Rathke's cleft cysts and colloid cysts may be differentiated from NC based on location of the lesion.[138] Cystic neoplasms such as schwannoma, pleomorphic xanthoastrocytoma and metastasis can be generally excluded from the differential diagnosis given their rare midline appearance, association with cranial nerves and contrast enhancement.[77]

Parasitic infections such as racemose neurocysticercosis appear as large cystic lesions and may occur with a signal similar to CSF on both CT scans and MRI.[88] These lesions, however, are commonly multiple and located within the basal cisterns and Sylvian fissure. There may also be a degree of associated contrast enhancement of leptomeninges which does not occur with NC.[139]

In the context of highly variable radiological and intraoperative appearances in conjunction with a variety of pathologies that may be indistinguishable with current imaging techniques, a definitive diagnosis of NC can only be made after histological and subsequent immunohistochemical analysis of the cyst wall.

9. Histopathology

On gross anatomical inspection intraoperatively, NC usually appear as yellow or milky-white, thin-walled cysts containing a gelatinous typically transparent fluid, although mucoid or xanthochromic cyst contents have been described.[6,20,21,88,96,140] Upon histological examination NC are benign lesions with cyst walls composed of simple or pseudostratified cuboidal or columnar epithelium with a basement membrane that resembles gastrointestinal or respiratory epithelium.[17,95,116] Approximately 50% of cysts are lined by gastrointestinal epithelium, with 17% by ciliated respiratory epithelium and the remainder having a mixed appearance of the above types as well as containing pancreatic and squamous epithelium.[1,4,6,11,12,20,65,88,102,114,116] The finding of respiratory epithelium is in keeping with the embryological formation of the respiratory tract from buds of the neuroenteric canal.[69,88,103] NC epithelium may also contain mucin-secreting goblet cells consistent with a gastrointestinal epithelium.[1]

Although these lesions are most commonly benign, rare instances of both *de novo* malignancy and malignant transformation of NC in the setting of recurrence have been reported.[3,32,78,97] Malignant transformation of the NC wall into an adenocarcinoma appears to have been the cause in these patients and this information is supported through careful exclusion of a potential primary as a source of metastasis and a noted increase in the Ki-67 immunohistochemical marker for cellular proliferation.[32,78,97,141]

In addition to histopathological appearance using light and electron microscopy, immunohistochemistry (IHC) has assisted in the diagnosis of NC. NC epithelial cells usually stain positive with anti-carcinoembryonic antigen antibody, anti-cytokeratin (CK) monoclonal antibody, and anti-epithelial membrane antigen (EMA) antibody suggesting an endodermal origin of the epithelium.[1,26,29,54,102,103,114,115] NC are reported to stain negative for glial fibrillary acidic protein, neuron specific enolase and vimentin, these markers being positive in ectodermal cysts.[58,96,142] Additional special stains are also useful in the work-up of NC including

periodic acid Schiff (PAS) staining, an indicator of the presence of secretory granules in goblet cells, signifying an endodermal origin of the cyst.[1,46] Carbohydrate antigen (CA) 19-9 has been demonstrated to stain positively in both NC of benign origin and those having undergone malignant transformation.[102,143] IHC and light microscopy appear essential in establishing a diagnosis of NC, particularly in the setting of non-specific findings on radiology.

10. Management

Due to the lack of sufficient reports, the management of intracranial NC remains poorly understood. The current recommendation in the literature suggests that surgical excision is the only treatment to be advocated in patients presenting with a symptomatic NC. The most favourable outcomes have been associated with complete resection of the cyst wall through aspiration and ideally marsupialisation.[103] The surgical approach must adequately expose the cyst and its interface between the surrounding tissue. In some instances the cyst wall may adhere strongly to critical neural structures such as the brainstem and total resection may not be possible. The authors of a surgical series of seven patients with NC suggested that the degree of adhesion between the lesion and surrounding neural structures is directly proportional to the size of the lesion and thinness of the cyst wall.[96]

Postoperative meningism has been reported with some patients complaining of headache, fever and neck stiffness.[96] It has been postulated the meningism is secondary to chemical meningitis caused by leakage of cyst contents into the subarachnoid space and has been confirmed through a finding of elevated CSF protein content. This was managed successfully with regular lumbar punctures every two to three days.[96] To prevent this complication, it was further suggested that the cyst should be punctured with a syringe and the contents aspirated prior to its incision. The subarachnoid space should be protected during this process with cotton patties surrounding the operative region and copious warm irrigation to remove residual cyst contents.[96]

Avoidance of cyst fenestration, biopsy and partial wall resection have been suggested as management options due to the high risk of recurrence.[26,128] Wang et al. have postulated that in subtotal resection of an NC, the residual cyst wall may be responsible for cyst recurrence. If the remnant walls are sufficient to overlap, the lesion may reform. Thus, care should be taken intraoperatively to identify cyst wall remnants and to electrocoagulate them to avoid NC recurrence.[96] Recurrence in NC, which may occur between four months and 14 years,[26,96] has occurred even in the setting of complete macroscopic resection.[26] Duration of follow-up for postoperative intracranial NC patients with repeat imaging should reflect this potential for long-term recurrence. Recurrence may present with signs of aseptic meningitis or can be associated with increasing levels of CA19-9 isolated in the CSF.[96] This may provide an avenue for surveillance for recurrence with biochemical testing.

Outcome in terms of recurrence rates remain poorly understood due to the paucity of patients. In addition, there are case reports of apparent *de novo* progression of NC to adenocarcinoma which were subsequently treated with surgical resection.[3,32,78,97] Surgery remains the treatment of choice and neither conventional radiation nor chemotherapy is recommended.[32,97] There is insufficient evidence to support the use of stereotactic radiosurgery in the treatment of NC.[26,96] However, more research, as the number of patients with NC increases, may assist in further delineating the roles of adjuvant treatments in NC, particularly in the setting of malignant progression.

11. Illustrative patient

11.1. Clinical presentation and investigation

A 47-year-old woman presented with an eight-week history of headache and minor, non-specific symptoms. The physical examination was normal. A CT scan showed low-density extra-axial lesions of the midline posterior fossa and the right frontal region. MRI demonstrated non-enhancing extra-axial cystic lesions in the posterior fossa, left cerebellar hemisphere, right frontal and right parietal convexity, all homogeneously hypointense on T1-weighted, and hyperintense on T2-weighted, MRI (Fig. 2). The lesions were initially considered to be arachnoid cysts, but were noted to enlarge over a period of nine months. Other pathologies were considered including neurocysticercosis, as the patient had lived most of her life in South-East Asia.

11.2. Surgery and postoperative course

It was decided to biopsy/excise the right frontal cyst as this was the most accessible and had been observed to grow during the

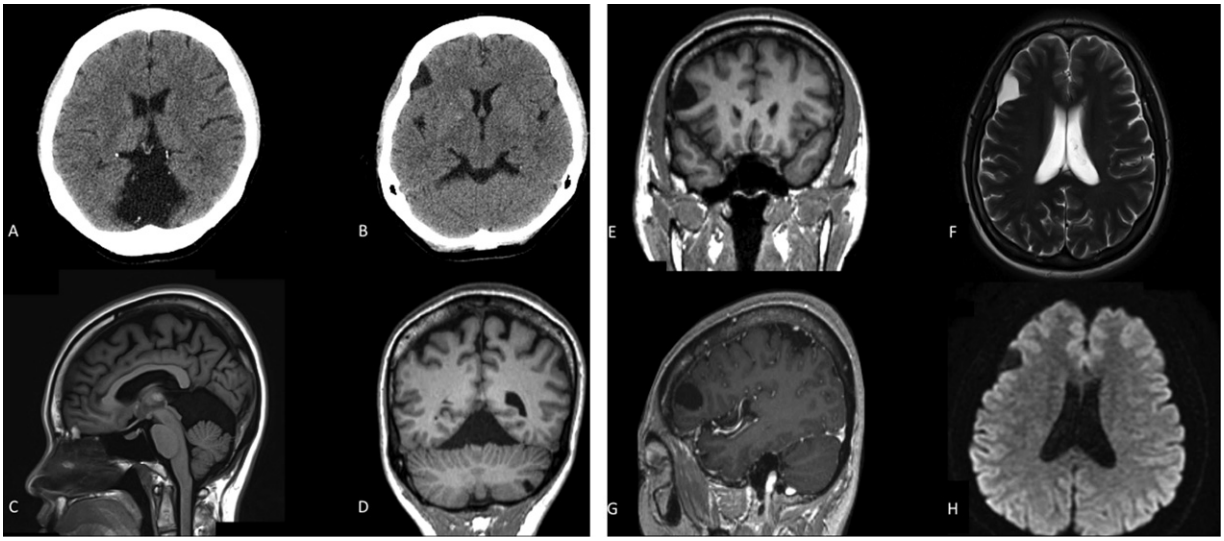

Fig. 2. (A, B) Axial CT scans showing hypodense extra-axial lesions in the posterior fossa and right frontal lobe. (C, sagittal; D, E, coronal) MRI showing lesions that are hypointense on T1-weighted MRI, (F, axial) hyperintense on T2-weighted MRI, (G, coronal) non-enhancing on T1-weighted enhanced MRI and (H, axial) without significant restriction on diffusion-weighted imaging.

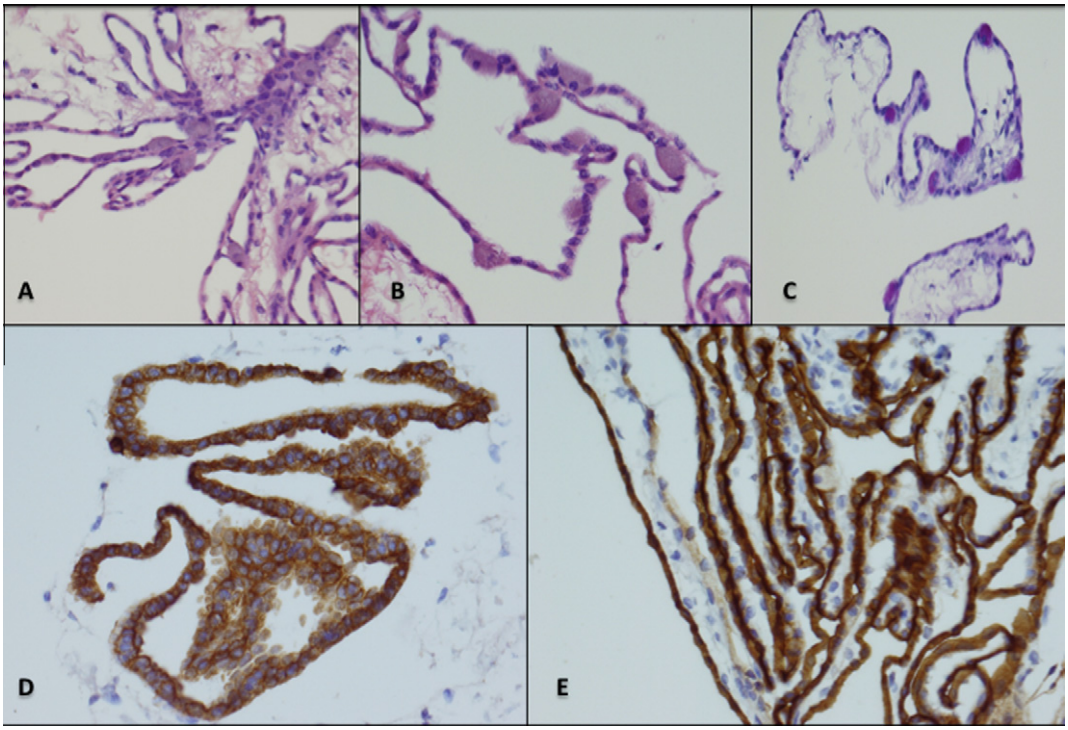

Fig. 3. Histopathological examination of the surgical specimen showing: (A) a single layer of cuboidal epithelium (haematoxylin and eosin [H&E], ×400), (B) scattered goblet cells (H&E, ×600), (C) goblet cells containing periodic acid Schiff (PAS)-positive mucin in the cytoplasm (PAS, ×400), (D) epithelial cells staining positively for cytokeratin (CAM5.2, ×400) and (E) staining positively for epithelial membrane antigen (EMA, x400).

period of observation. The patient underwent a right frontotemporal craniotomy. The cyst had a very thin arachnoid-like wall containing clear fluid, and had indented the adjacent frontal lobe. The wall of the cyst was excised completely and both the cyst wall and the fluid were sent for pathological analysis. The postoperative period was uneventful and the patient was discharged a few days after surgery. This patient, with bilateral localisation of multiple NC in both the supratentorial and infratentorial compartments, illustrates a very rare presentation.[1,3,29,108,109]

11.3. Pathological examination

Light microscopy revealed a single layer of cuboidal epithelium with goblet cells containing PAS-positive mucin in the cytoplasm. IHC showed that the epithelial cells stained strongly for CK and EMA (Fig. 3). The morphological appearance intraoperatively and histological findings were in keeping with the diagnosis of NC.

12. Conclusion

NC are rare, benign and non-neoplastic lesions, but should be considered in the differential diagnosis of cystic lesions involving the CNS. Their radiological appearance may be misleading owing to variable protein content of the cyst fluid and the existence of similar intracranial lesions that are difficult to differentiate from NC. Because of this, histology with IHC is required to make a definitive diagnosis. Due to the high local recurrence rate observed after partial resection, the goal of surgery should be cyst content aspiration followed by complete microsurgical cyst wall resection. As recurrences may be delayed in the order of years, periodic clinical and MRI follow-up is advised for at least 10 years following surgery.

Acknowledgements

The authors thank A/Prof. Kate Drummond (Consultant Neurosurgeon, Royal Melbourne Hospital, Australia) for her advice and assistance in preparing this article.

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