CASE REPORT

A mistaken identity: rhabdomyosarcoma of the middle ear cleft misdiagnosed as chronic suppurative otitis media with temporal lobe abscess

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SUMMARY

A 5-year-old girl presented with a 3-month history of left side facial palsy, followed sequentially by purulent ear discharge, complete external ophthalmoplegia and blurred vision. On clinical examination she was febrile with left-sided conductive hearing loss. She was clinically diagnosed to have chronic suppurative otitis media of the unsafe type with petrous apicitis, middle cranial fossa abscess and cavernous sinus involvement. Preliminary CT scan findings were reported as a large left temporal lobe abscess and left otitis media with cholesteatoma. MRI of the brain obtained later corroborated the abnormalities detected on the CT scan. Ten days after admission, a mass was seen protruding from the external auditory canal. A biopsy of the mass was obtained and sent for histopathological examination. Meanwhile, review of the MRI suggested an aggressive neoplasm such as sarcoma/rhabdomyosarcoma. Histopathology clinched the final diagnosis of an anaplastic type of embryonal rhabdomyosarcoma of the middle ear cleft.

BACKGROUND

Rhabdomyosarcoma is the commonest soft tissue sarcoma in children.¹ The head and neck account for 35% of rhabdomyosarcomas in children.² When it originates from the middle ear cleft, presenting symptoms of ear discharge, hearing loss and a mass in the external auditory canal closely mimic manifestations of chronic or refractory otitis media.^{3, 4} Presence of facial palsy, though highly evocative of rhabdomyosarcoma, is also a complication of chronic suppurative otitis media (CSOM).³ As the symptoms involve the ear, the majority of patients would seek consultation with otorhinolaryngology services. Therefore, the diagnosis of rhabdomyosarcoma is very often elusive among paediatricians when awareness may be suboptimum, as we experienced with a child having embryonal rhabdomyosarcoma of the middle ear cleft that was misdiagnosed as complicated CSOM. The consequence of late diagnosis is delayed treatment. Since timely referral and treatment impacts prognosis, early diagnosis is critical.¹

CASE PRESENTATION

A 5-year-old Indian girl presented to the emergency paediatric service with recurrent fever and vomiting, deviation of the angle of the mouth to the right side and incomplete closure of the left eyelid of 3 months' duration. She had developed head tilt

to the right side for the past 1 month and purulent, foul smelling left ear discharge and redness of the left eye for 10 days, with headache and blurred distant vision starting 3 days prior. Her immunisation status was age appropriate.

On examination she was febrile. Her weight (12 kg) was below the 3rd centile, and height (105 cm) was between the 15th and 50th centiles (WHO growth charts). There was a foul smelling, purulent, blood-stained discharge from the left ear. ENT examination on admission revealed the left ear external auditory canal filled with pus. Left side conductive hearing loss was detected by Rinne and Weber's tests. The left eye had mucopurulent discharge, ptosis, incomplete closure of the eyelids, Bell's phenomenon, conjunctival congestion, exposure keratitis of the inferior cornea, and absence of corneal and conjunctival reflexes. There was complete left ophthalmoplegia with left pupillary dilation and left side lower motor neuron facial palsy. Examination of other cranial nerves and the rest of the central nervous system was normal. Signs of meningeal irritation were absent. There was no lymphadenopathy.

The initial clinical diagnosis was left unsafe CSOM with left side III, IV, VI and VII cranial nerve palsies probably caused by left petrous apicitis/middle cranial fossa abscess with cavernous sinus involvement.

INVESTIGATIONS

Blood investigations on admission showed a haemoglobin level of 131 g/L, leucocytosis (white cell count of $14.8 \times 10^9/L$, (83% neutrophils, 16% lymphocytes and 1% eosinophils) and platelet count of $400 \times 10^9/L$). Her liver and renal function tests were normal. Mantoux test was negative. Chest radiograph was normal.

The initial clinical impression was corroborated by a contrast enhanced CT of the brain performed on admission that showed a peripherally enhancing lesion reported as a large left temporal lobe abscess and left otitis media with cholesteatoma causing cavernous sinus, sigmoid sinus and internal jugular vein thrombosis and extending into the left parapharyngeal space (figures 1 and 2). The lesion was also seen extending into the left cerebellopontine angle. *Pseudomonas* spp and *Staphylococcus aureus* were cultured from the ear discharge. Treatment with intravenous amikacin, metronidazole, vancomycin and ceftazidime was started in accordance with culture sensitivity reports. However, there was

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Figure 1 A peripherally enhancing lesion involving the left medial temporal lobe and petrous apex, suggesting likelihood of an abscess.

no improvement as ear discharge and constitutional symptoms persisted after 6 days of therapy, prompting MRI of the brain. The preliminary reporting of the MRI brain suggested a hyperintense heterogeneously enhancing lesion on T2-weighted images with predominant peripheral postcontrast enhancement reported as an abscess which was centred in the left petrous temporal bone with extension in adjacent areas as reported on the CT scan.

Ten days after admission, a mass was seen protruding through the left external auditory canal (figure 3). This mass rapidly enlarged over the next few days. Langerhans cell histiocytosis (LCH) was an alternative diagnosis proposed following otolaryngology consultation. A biopsy of the mass was taken for histopathology and *Mycobacterium* growth incubator tube (MGIT) test for tuberculosis. Meanwhile, the MRI was reviewed by a consultant radiologist (SS, coauthor). The pathology was now reported as a lytic, expansile petrous temporal bone lesion, probably a high-grade sarcoma or a round cell tumour. The

Figure 2 Axial high resolution CT of left temporal bone showing permeative destruction of the left-sided petrous apex. Left middle ear ossicles are surrounded with soft tissue and medially eroded, but there is absence of complete ossicular destruction/disruption.

Figure 3 Clinical photograph of the child showing a mass visible in the left external auditory canal.

lesion extended anterosuperiorly extra-axially to the left temporal lobe, medially to the left cavernous sinus, posteriorly to the cerebellopontine angle and laterally into mastoid air cells and the external auditory canal (figures 4 and 5). A few flow voids were also seen, suggesting high vascularity of the lesion. Review of the CT revealed that the mastoid cell trabeculations and middle ear ossicles were not completely eroded as would usually be expected with a large invasive cholesteatoma.^{5 6}

The biopsy report clinched the diagnosis as primary embryonal rhabdomyosarcoma of the anaplastic type (figures 6 and 7). MGIT was subsequently negative.

DIFFERENTIAL DIAGNOSIS

Ear discharge, hearing loss and a mass in the external auditory canal is highly suggestive of rhabdomyosarcoma, particularly when accompanied by ipsilateral facial palsy.^{3 4 7} The characteristic symptom of a mass in the external auditory canal sometimes occurs late in the course of the disease, as we experienced.⁴ In our case, as the symptoms evolved during the ward stay, several differential diagnoses could be considered at each stage.

Figure 4 T2-weighted axial section of MRI brain shows a heterogeneous, hyperintense lesion measuring 4.7x4 cm (anteroposterior x transverse) in the left petrous temporal bone. This lytic, expansile lesion extends anterosuperiorly to the left temporal lobe region, medially to the left cavernous sinus, posteriorly to the left cerebellopontine angle and left middle cerebellar peduncle, laterally to mastoid air cells and the left external auditory canal.

Figure 5 Coronal and sagittal T1 fat saturated postgadolinium-enhanced MRIs showing heterogeneously enhancing mass lesion with central necrosis that engulfs the carotids with involvement of contiguous structures such as cerebellum, skull base, sphenopalatine fossa, infratemporal fossa, parapharyngeal and visceral space of neck. Central flow voids are visible.

On admission

CSOM with complications is the closest differential diagnosis as the symptom complex of a febrile illness with vomiting, facial palsy, otorrhoea and conductive hearing loss, evolving to complete ophthalmoplaegia due to III, IV and VI cranial nerve palsies, bears a striking resemblance to symptoms of CSOM with associated mastoiditis and cholesteatoma resulting in extracranial and intracranial complications.

Facial palsy results from spread of infection through a congenital dehiscence of the facial nerve canal; it is erosion by an expanding cholesteatoma (extracranial complication) or from petrous apicitis (intracranial complication).⁸ Petrous apicitis would manifest with Gradenigo syndrome characterised by otorrhoea, reduced hearing, pain along the first and second divisions of the trigeminal nerve and VI nerve palsy.⁶ Contiguous or haematogenous spread of infection to the cavernous sinus could explain the III and IV cranial palsies. However, this is a rare complication of unsafe CSOM.^{9 10} Symptoms of fever and vomiting could be attributable to an intracranial complication like a temporal abscess in this setting.

In India, symptom of chronic otorrhoea would be highly suspicious of tuberculosis of the middle ear or mastoid as India has the highest incidence of tuberculosis in the world.¹¹ Tuberculous infection of the middle ear is often primary by inoculation from the external auditory canal through a tympanic membrane perforation. However, 50% of cases of ear tuberculosis in children are secondary and a primary focus of infection

may be apparent elsewhere.¹² Signs of ear tuberculosis include granulations in the external auditory canal, hearing loss and facial palsy.^{4 11 12} Retrograde spread of infection from the mastoid could involve the petrous apex.¹¹ Intracranial complications like meningitis, multiple cranial nerve palsies and tuberculous brain abscess may ensue.¹²

Following appearance of a mass protruding from the external auditory canal

Cholesteatoma can present as a polyp originating in the middle ear or mastoid and prolapsing in the external auditory canal.^{8 13} In a histopathological study by Xenellis *et al*,¹³ the commonest aetiology of external auditory canal mass in 65 out of 75 cases (86%) was a cholesteatoma. Nonetheless, tumours, pseudotumours and inflammatory disease manifesting with a polypoidal mass in the external auditory canal have symptoms similar to a cholesteatoma.¹³ Though most of these conditions occur in adults, aural polyps due to chronic otitis media, tuberculosis of the middle ear and LCH would be common in children. The external auditory canal lesion of glomus jugulare tumour may resemble rhabdomyosarcoma, but this tumour has not yet been reported in children <16 years of age.⁷

LCH involves the temporal bone in 15–61% of cases.¹⁴ Manifestations include mastoid swelling, otorrhoea, deafness, vertigo and aural polyp.¹⁵ Facial palsy has also been reported with LCH involving the temporal bone.¹⁶ Extensive disease

Figure 6 Photomicrograph of tumour cells stained with myogenin (immunohistochemical stain) showing focal positivity, graded+ (×100).

Figure 7 Focal, nuclear positivity for MyoD1 (immunohistochemical stain) in rhabdomyoblastic tumour cells, graded+ (×100).

could involve the petrous apex leading to VI cranial nerve palsy. Cavernous sinus may be rarely involved.¹⁵

On neuroimaging

On CT, a cholesteatoma would appear as a nodular soft tissue mass within the tympanomastoid cavity.¹⁷ It is usually accompanied by bone erosion involving the wall of the epitympanic recess, ossicles (especially long process of the incus), tegmen tympani and lateral semicircular canal.^{6 17} The mastoid air cell trabeculations disappear.⁵ The mastoid is usually sclerotic in chronic infectious or inflammatory disease.⁶ A cholesteatoma on MRI is isointense on T1-weighted images with variable enhancement on contrast. On T2-weighted images, the signal is isointense. The distinguishing feature is a hyperintense signal on non-echo-planar imaging diffusion-weighted imaging.¹⁷

In tuberculosis of the middle ear, CT would reveal mucosal thickening of the external auditory canal and a soft tissue occupying the entire middle ear-mastoid region, which may extend into the external auditory canal but without bony erosions. The mastoid air cells would be preserved with absence of sclerosis.¹¹ Involvement of the petrous is characterised by loss of aeration and septae within the mastoid, which is occupied by a soft tissue mass on high resolution CT. On MRI, the soft tissue is hypointense on T1-weighted images with heterogeneous contrast enhancement. On T2 or short tau inversion recovery images the mass lesion is hyperintense.¹¹

On the other hand, CT lesions of LCH are destructive. The mastoid is commonly involved, whereas involvement of the squamous temporal bone and middle ear is rare. The typical finding is a lytic, 'punched out' bone lesion.¹⁵ On MRI, a soft tissue mass when present is hypointense to isointense on T1-weighted images, enhances homogeneously with contrast and is hyperintense on T2-weighted images.¹⁵

OUTCOME AND FOLLOW-UP

The child was referred to a regional cancer institute for further management where workup (CT scan of the chest and abdomen, cerebrospinal fluid examination and cervical lymph node biopsy) did not show evidence of metastasis. She was advised neoadjuvant chemotherapy. She completed three cycles of chemotherapy over 3 months; however, CT following chemotherapy showed progressive spread of the tumour to the skull base and parapharyngeal space. She succumbed to the disease approximately 5 months after diagnosis.

DISCUSSION

Among head and neck cancers in children, rhabdomyosarcoma is encountered in 7.8–25.7% of cases.¹⁸ In children, 35–40% of rhabdomyosarcomas occur in the head and neck and involve the orbit, oral cavity, nasopharynx, middle ear and temporal bone.^{2 19}

Rhabdomyosarcoma of the middle ear is a type of parameningeal rhabdomyosarcoma. It accounts for less than 10% of all cases of head and neck rhabdomyosarcomas.²⁰ The anatomical extent of middle ear cleft rhabdomyosarcoma includes tumours arising from the middle ear, eustachian tube and/or the mastoid region.⁷ These are considered unfavourable sites as they are prone to complications of cranial nerve palsies, skull base erosion and intracranial extension.²¹

Symptoms of rhabdomyosarcoma of the middle ear or mastoid are otorrhoea, hearing loss and aural polyp; they are often accompanied by facial palsy.^{3 4 7} In four out of six cases (66%) of temporal bone rhabdomyosarcoma reviewed by Sbeity *et al*,²⁰ symptoms were of chronic otitis media. Suboptimum

awareness of this manner of presentation and resemblance of clinical features to chronic otitis media may delay diagnosis.³ In the present case, constitutional symptoms and facial palsy were the initial complaints, followed by ear discharge progressing to complete ophthalmoplaegia. It was only when a protruding mass was visible at the external auditory meatus late in the course of the disease that a neoplasm was considered as a diagnosis.

Additionally, in India prevalence of ear infections such as CSOM (including mastoiditis and complications such as cholesteatoma) is among the highest in the world, estimated at 7.8% in school children.²² Owing to this high prevalence of CSOM, it would not be surprising that the recognition of rhabdomyosarcoma would be inadvertently delayed by a paediatrician. However, rhabdomyosarcomas involving the middle ear may be more frequent among Indian children as half of the 24 cases of head and neck rhabdomyosarcoma studied by Mehta *et al*²³ involved this site. This emphasises the need for awareness of the manner of presentation of rhabdomyosarcomas in this population.

Involvement of cranial nerves is common with parameningeal rhabdomyosarcoma due to direct spread to the central nervous system and indicates advanced tumours.¹⁹ Involvement of all cranial nerves from II to XII has been reported, though the vestibular part of the auditory nerve is spared.⁷ All the 25 cases with neurological manifestations of embryonal rhabdomyosarcoma of the middle ear cleft reviewed by Leviton *et al*⁷ had facial palsy. Facial palsy may be the only presenting feature, often preceding involvement of other cranial nerves.^{4 7} Thus, presence of facial palsy preceding or developing early in the course of 'chronic otitis media' should provoke suspicion of a neoplasm like rhabdomyosarcoma even in the absence of an aural mass. Abducens nerve is also one of the vulnerable cranial nerves in parameningeal rhabdomyosarcoma.^{7 19} Extensive neoplastic invasion of the skull base and cavernous sinus can result in multiple cranial neuropathies including complete ophthalmoplaegia (III, IV, VI) and facial palsy (VII) as observed in our patient.^{7 19} The oculomotor and trochlear nerves could also be involved in neoplastic pathology of the petrous apex due to their proximity.²⁴

In the case being reported, CT findings of bony destruction and a lesion with peripheral postcontrast enhancement were misinterpreted as a temporal abscess. The initial MRI report by a trainee radiologist of a temporal lobe abscess was also misleading. Subsequent review of the MRI showing a lytic, expansile and invasive lesion with intermediate signal intensity on T1-weighted images, heterogeneous signal intensity on T2-weighted images and variable enhancement was highly suggestive of a sarcoma. Thus an experienced radiologist is instrumental for accurate diagnosis. On imaging, the soft tissue component of the lesion was predominantly solid with heterogeneous enhancement while there was a relatively smaller area of central necrosis that showed peripheral enhancement. This favours neoplasm over an abscess. On MRI, an abscess would be expected to have more necrosis than a soft tissue component and peripheral ring of enhancement. On diffusion-weighted imaging, an abscess would show restricted diffusion.¹⁴ An additional imaging clue in our patient pointing away from a cholesteatoma was the presence of flow voids suggestive of a lesion with a vascular component.

Imaging of middle ear cleft rhabdomyosarcoma requires both CT and MRI as complementary investigations for an accurate diagnosis. MRI offers excellent imaging of bony erosion while MRI provides information about the origin, size, extent, invasiveness and spread of the tumour to lymphnodes and the

central nervous system, and post-therapy recurrence.^{1 20 21 25} MRI would also aid in ruling out the other differential diagnoses (cholesteatoma, tuberculosis and LCH).

This case illustrates the long diagnostic journey of a rare head neoplasm with a grave prognosis due to symptoms mimicking a common entity of unsafe CSOM with intracranial complications. The misinterpretation of symptoms was not only clinical, but also reinforced by misreading of neuroimaging findings. On hindsight, some clinical clues pointed to a neoplastic aetiology: (1) facial palsy was the inaugural symptom, which preceded onset of otorrhoea by a few months, (2) cavernous sinus thrombosis with III and IV cranial nerve paralysis is an exceptionally rare complication of unsafe CSOM whereas it is frequently involved with parameningeal rhabdomyosarcoma and (3) despite symptoms of headache and vomiting, there were no objective signs of meningitis, hemiparesis or increased intracranial pressure on physical examination, which would have accompanied chronic otitis media with intracranial complications of brain abscess. Thus a logical analysis of clinical manifestations and correct interpretation of neuroimaging abnormalities is vital for early diagnosis.

Learning points

- ▶ Rhabdomyosarcoma of the middle ear presents with symptoms mimicking chronic suppurative otitis media.
- ▶ Facial palsy is invariably present and could be the presenting symptom of rhabdomyosarcoma. When facial palsy is accompanied by multiple cranial neuropathies, careful examination of the middle ear to detect an aural mass is crucial in children presenting to a paediatric service with otitis media, as this could clinch the diagnosis of rhabdomyosarcoma.
- ▶ Early biopsy of an aural mass is important to clinch the diagnosis.
- ▶ Involvement of an experienced radiologist is critical for correct interpretation of bone destroying pathologies of the petrous bone. MRI with its superior soft tissue resolution complements CT for accurately defining such lesions.

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