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LETTER TO THE EDITOR

Nasal chondromesenchymal hamartoma: correlation of typical MR, CT and pathological findings

Alice Yao-Lee · Maura Ryan · Veena Rajaram

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Sir,

We read with great interest the case report on nasal chrondromesenchymal hamartoma (NCMH) by Johnson et al. [1]. They presented the spectrum of CT findings of this rare, benign pathological entity that can be locally aggressive and mimic malignancy. We would like to highlight the MR appearance of NCMH, which correlates with known CT and pathological features, as well as findings seen with intraorbital and intracranial extension.

The described CT findings include internal calcifications, bony remodeling, variable enhancement, cystic components, local extension into the orbit, paranasal sinuses and, rarely, intracranial compartment [1–5] (Fig. 1). The imaging spectrum reflects the histology of NCMH, which is composed of different mesenchymal elements, the most prominent being sheets/fascicles of spindled cells in a fibrous or myxoid background. Other components include well-demarcated islands of cartilage and prominent vessels often with perivascular fibrosis or prominent collagen deposition. Reactive bone may be seen in some of them, which is mainly reactive bone remodeling.

A. Yao-Lee (✉)
Department of Radiology, Northwestern Memorial Hospital,
676 N. St. Clair St., Suite 800,
Chicago, IL 60611, USA
e-mail: alice-yaolee@fsm.northwestern.edu

M. Ryan
Medical Imaging, Children's Memorial Hospital,
Chicago, IL, USA

V. Rajaram
Department of Pathology, Children's Memorial Hospital,
Chicago, IL, USA

Areas of mineralization, within the matrix of the cartilaginous components of the lesion, are better visualized on CT but correlate with areas of T2 hypointensity and low gradient signal seen on MRI. Additionally, areas of fibrosis would have expected hypointensity on T2-weighted images. Cystic and myxoid components are poorly discernible as areas of low density on CT but are more clearly defined on MRI by characteristic, discrete areas of marked T2 hyperintensity (Fig. 2). A companion case of NCMH confined to the nasal cavity demonstrates similar MR findings (Figs. 3 and 4). As

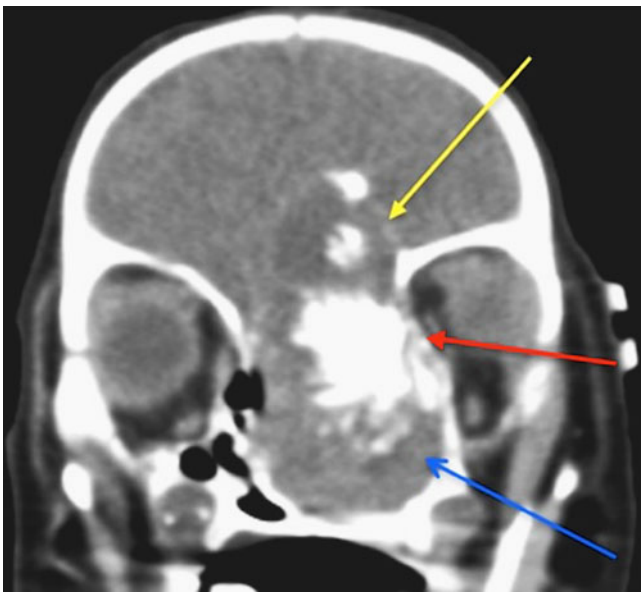


Fig. 1 Non-contrast CT soft-tissue algorithm in the coronal plane reveals a large heterogeneous mass centered within the left nasal cavity with bony remodeling of the sinonasal walls. There is intraorbital extension through the thin and eroded medial left orbital wall (*red arrow*), as well as intracranial extension through the ethmoidal roof *(yellow arrow*). Internal coarse calcifications are well demonstrated by CT, but focal areas of low attenuation are subtle (*blue arrow*)

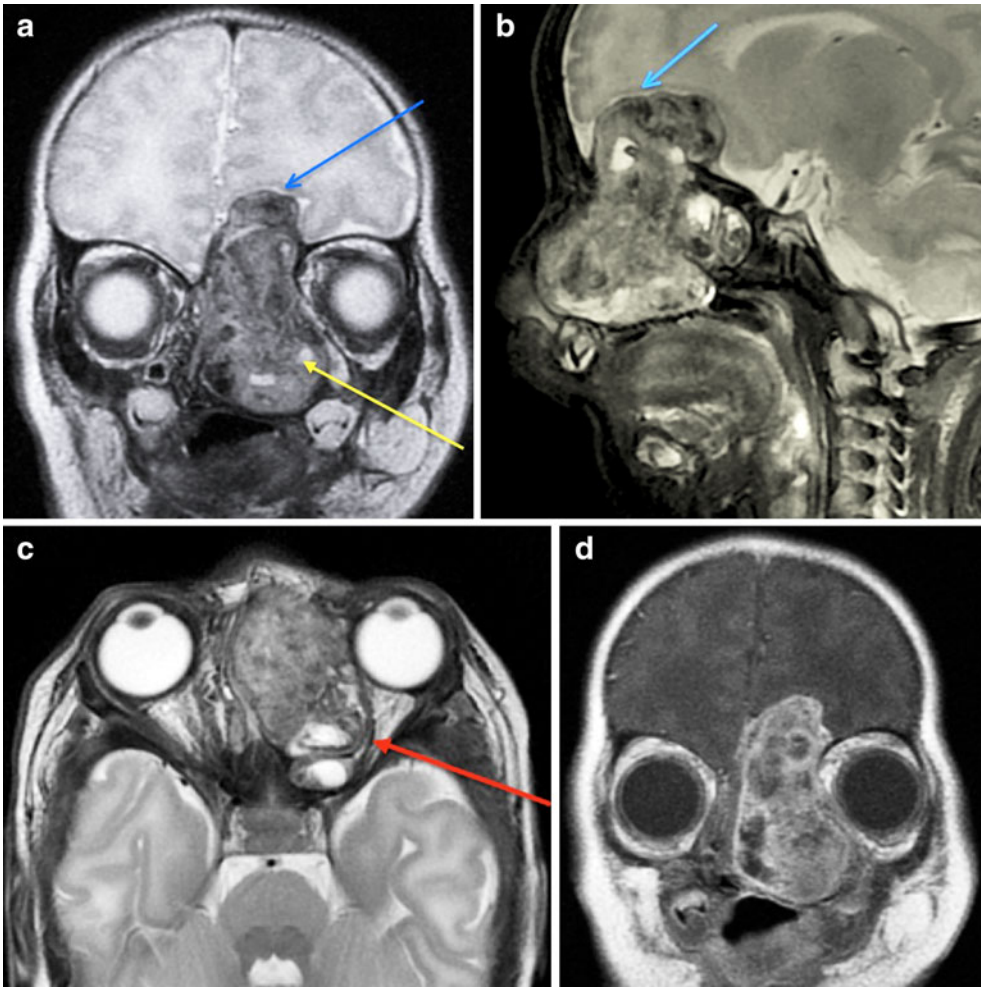
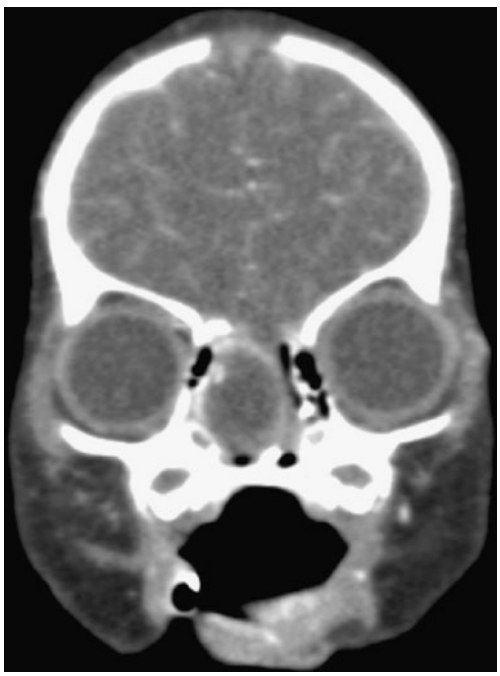


Fig. 2 MRI coronal (**a**), sagittal (**b**) and axial (**c**) T2-W sequences show areas of T2 hypointensity that correspond with coarse calcifications visualized on CT. Other areas of T2 hypointensity reflect fibrosis. Areas of well-defined T2 hyperintensity, reflecting cystic and myxoid components CT (*yellow arrow*), are much more clearly defined than the corresponding low-density regions on CT. Coronal T2 and sagittal fat-saturated T2-W sequences (**a**, **b**) better demonstrate intracranial extension through the non-ossified cribriform plate. The mass is extradural and extends through the floor of the anterior cranial fossa, displacing the frontal lobe superiorly, with a thin rim of T2 hyperintense CSF separating the dural mass component from the brain parenchyma (*blue arrows*). Intraorbital extension through the medial left orbital wall causes mass effect upon the posterior left orbital contents and apex (*red arrow*). **d** A post-contrast coronal T1-W image demonstrates variable but mild enhancement



on CT, the enhancement pattern is variable, but generally mild, despite the presence of vascularity commonly seen on pathology.

Although CT better visualizes osseous erosion and remodeling, MR provides superior tissue characterization and delineation of extension into the adjacent structures. MRI is particularly helpful in evaluating intracranial extension, given the incomplete ossification of the cribriform plate in the infant population. The MR imaging in this case shows that intracranial extension is extradural and along the floor of the anterior cranial fossa. When orbital extension is present, MR is superior to CT in assessing the involvement of the intraorbital contents, including the extraocular muscles, optic nerve, and globe. In this case, intraorbital extension causes mass effect on the posterior orbital contents, including the optic nerve with subsequent optic nerve atrophy.

◀ **Fig. 3** Contrast-enhanced CT of a companion case of a neonate with NCMH confined to the nasal cavity. Areas of CT hyperdensity along the right lateral aspect of the mass are too dense to represent vascular enhancement and represent chondroid/ossified components of the mass. Areas of hypodensity are ill-defined on CT but better delineated on MR

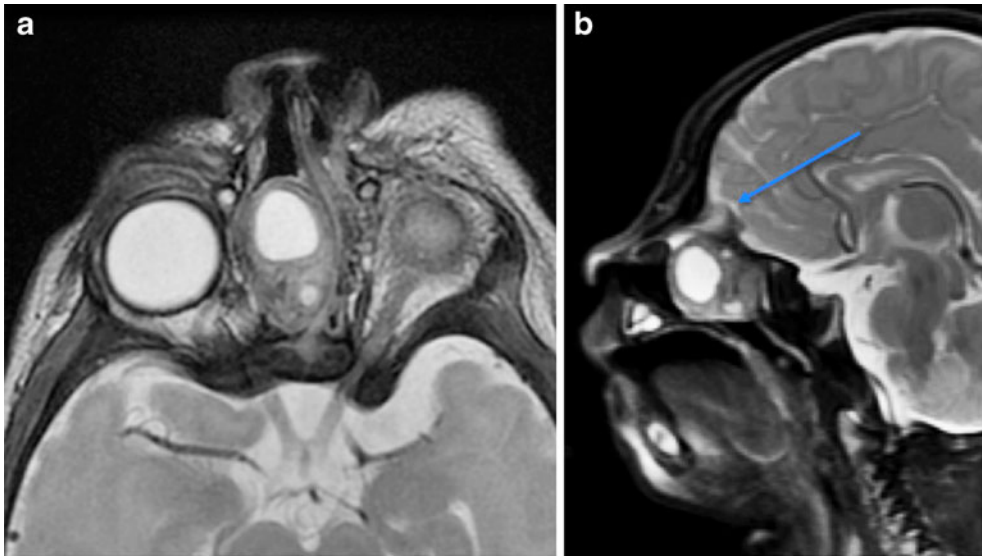


Fig. 4 MRI of a companion case of a neonate with NCMH confined to the nasal cavity. MR axial (**a**) and sagittal (**b**) T2-W images more clearly delineate ill-defined areas of hypodensity on CT as cystic areas of T2 hyperintensity on MRI, representing the myxoid and low cellular components of the mass. Areas of T2 hypointensity represent chondroid or reactive bone components of the mass. There is remodeling of the sinus walls without intraorbital extension. On the MR sagittal T2-W image (**b**), there is CSF separation between the frontal lobe and the ethmoidal roof/crista galli (*blue arrow*). The mass is within the nasal cavity, without evidence of intracranial extension

There have been no reports of malignant degeneration to date. However, growth or regrowth in the setting of subtotal resection has been described and occurred in each of our cases and was identified on follow-up MRI imaging.

MR is increasingly utilized for the evaluation of pediatric sinonasal masses, providing detailed imaging without concerns related to ionizing radiation. The recognition of MR characteristics is important in the diagnosis and follow-up surveillance of this lesion. These cases demonstrate recognizable MR features of NCMH, including well-defined T2 hyperintense myxoid and cystic components, gradient susceptibility and intrinsic T1 shortening related to blood products and mineralization, and T2 hypointensity related to mineralization and fibrosis.

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