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CASE REPORT

MR Spectroscopy-Aided Differentiation: "Giant" Extra-Axial Tuberculoma Masquerading as Meningioma

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SUMMARY: Tuberculosis is common in the developing world and in developed nations secondary to increasing immunocompromise in the population. It commonly causes meningitis and parenchymal tuberculomas. We present a case of an unusual masslike "giant" extra-axial tuberculoma during pregnancy. Unusual morphology and size at imaging made meningioma a close differential. MR spectroscopy served to complement MR imaging, providing diagnostic confirmation and depicted findings characteristic of a tuberculoma.

Tuberculosis, an important public health problem compounded by the upsurge of the human immunodeficiency virus usually results from *Mycobacterium tuberculosis* infection, though *Mycobacterium bovis*, a frequent pathogen of the past, and *Mycobacterium avium* complex (*M avium* and *M intracellulare*) are infrequently encountered. Central nervous system (CNS) tuberculomas typically result from hematogenous dissemination and, upon histologic examination, are granulomas with central caseous necrosis.

Although intra-axial tuberculous granulomata are the more common variety, extra-axial lesions are rarely encountered[1-6] and may present diagnostic dilemmas that need to be resolved by imaging and allied techniques, so as to circumvent unnecessary chemotherapy, biopsies, and surgery. MR spectroscopy is one such technique that we used.

Case Report

A 25-year-old pregnant patient (gravida 1, para 0) presented at 27 weeks' gestation with severe headaches and focal right-sided convulsions of 3 months' duration without fever, papilledema, or elevated blood pressure. Elevated erythrocyte sedimentation rate (ESR) was noted. Postpartum lumbar puncture and CSF analysis after imaging studies revealed mild lymphocytic predominance and positive polymerase chain reaction (PCR) for tuberculosis. Antituberculous therapy was begun after suspending breast-feeding.

Two MR examinations (1.5T EchoSpeed; GE Medical Systems, Milwaukee, Wis) obtained at 27 weeks and postpartum revealed an unchanged 3.8 × 2.75 × 3.0-cm left frontal extraparenchymal mass lesion that appeared hypointense on T1-weighted images (T1WI; Fig 1*A*), with a thick, isohypointense rim. On T2-weighted images (T2WI, Fig 1*B*) and fluid-attenuated inversion recovery (FLAIR), it appeared predominantly hypointense with central hyperintensity, consistent with necrosis. The lesion appeared dark on diffusion-weighted images (DWI; Fig 2*A*). Contrast (gadolinium-diethylenetriaminepentaacetic acid [Gd-DTPA]) enhanced MR (Fig 2*B*) depicted prominent heterogeneous central enhancement with a thick, peripheral nonenhancing wall and peripheral rim-enhancement. Adjoining meningeal enhancement ("dural tail"), adjacent CSF cleft, mass-effect, and vasogenic edema were noted. MR angiography and venography revealed no abnormal vascularity or occlusion. MR perfusion revealed no changes in cerebral blood volume or flow. A differential diagnosis of inflammatory granuloma (with leptomeningitis and cerebritis) versus meningioma was considered.

A volume of interest of 8.0 mL was selected from the center of the lesion on T2WI and image-guided, single-voxel, point-resolved MR spectroscopy (PRESS; repetition time [TR]/echo time [TE] = 1500ms /35ms) was used, because with this technique, lipid contamination in volumes abutting fat structures is negligible. Multivoxel MR spectroscopy was obtained as well. Findings were consistent with necrotic inflammatory granuloma/tuberculoma, revealing elevated lipid-lactate and choline (Cho) peaks with barely detectable *N*-acetylaspartate (NAA) and creatine (Cr). No abnormal peak was detected corresponding to alanine. Follow-up MR spectroscopy (Fig 3*A*,*B*) remained unchanged. Long TE (144 ms; Fig 3*B*) MR spectroscopy sequence obtained from an identical location did not suppress the 1.33-ppm peak, confirming predominance of lipid. Findings were more conspicuous with multivoxel spectroscopy.

Based on imaging, spectroscopic, and clinical features, a diagnosis of giant extra-axial tuberculoma was made. Surgical excision biopsy confirmed the diagnosis.

Discussion

Although intraparenchymal tuberculomas are common, solid extra-axial tuberculous masses are *extremely* uncommon.[1] We believe our case is unique in that our patient was pregnant and immunocompetent, MR findings mimicked those of a similarly located isolated meningioma, and we were able to successfully use spectroscopy to confirm our diagnosis.

Extra-axial tuberculomas simulating meningiomas may be located in the frontoparietal areas[2] and pontine, pericallosal,[4] and suprasellar cisterns. Cystic[1] and en-plaque meningioma[5] mimics and those caused by *M avium* (noted in patients with systemic lupus erythematosus[3]) can be encountered. Most appear hypointense to isointense to gray matter[1-6] on T1WI and T2WI and variable on DWI, often with a hyperintense rim; those with hyperintense centers on T2WI are usually hyperintense on DWI.[7] Meningiomas typically appear isointense to gray matter on T1WI and T2WI *and* DWI, unless atypical or aggressive.[8] Signal intensity is a function of intralesional lipids, macrophages, fibrosis, and cellular infiltrates.[9]

In vivo proton MR spectroscopy has been studied extensively in this context. Tuberculomas are characterized by a prominent decrease in NAA/Cr and slight decrease in NAA/Cho.[10] Lipid-lactate peaks are usually elevated (86% of tuberculomas[11]). Paradoxically, lipid/Cr may occasionally be *decreased* relative to normal cerebral parenchyma, probably resulting from small dimensions of most tuberculomas rela-

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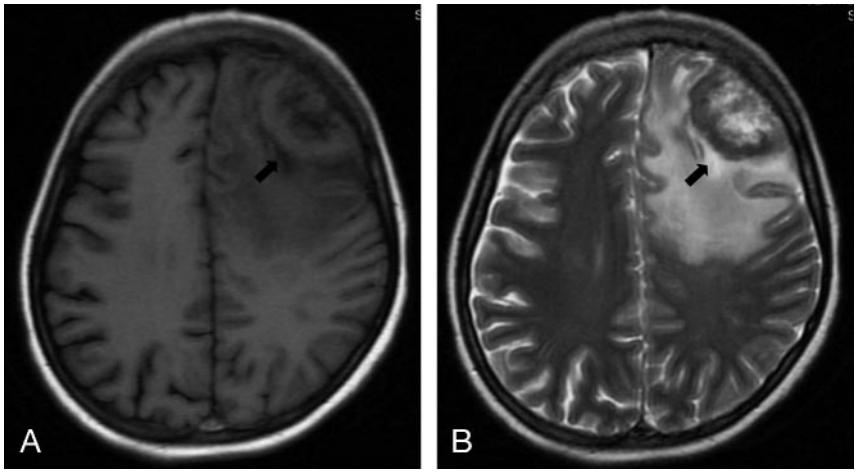

Fig 1. Axial T1WI (*A*; fast spin echo; TR/TE = 2015ms/9.7ms) and T2WI (*B*; fast spin echo; TR/TE = 4360ms/83ms) depicting a lobulated, dura-based lesion in the left frontal convexity that is predominantly hypointense on T1WI with a thick, iso-hypointense rim. On T2WI, it is of mixed intensity but predominantly hypointense with a hyperintense center. Displacement of the underlying parenchyma and a CSF cleft are identified (*arrows*). A large component of vasogenic edema is visualized.

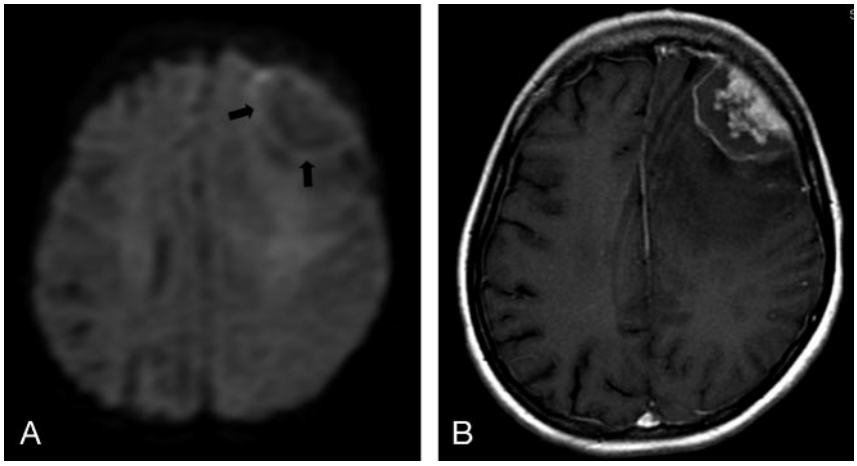

Fig 2. Axial DWI (*A*; TR/TE = 3000ms/11ms) depicts a dark-appearing lesion, with a hyperintense rim (*arrows*). Axial contrast-enhanced T1WI (*B*; Gd-DTPA enhanced fast spin echo; TR/TE = 2015ms/9.7ms) depicts prominent central enhancement, a thick peripheral nonenhancing component and rim-enhancement beyond this. There is thick enhancement of adjoining meninges ("dural-tail" sign, arrow) with mild enhancement of meninges in the anterior interhemispheric region and the right cerebral convexity (not shown).

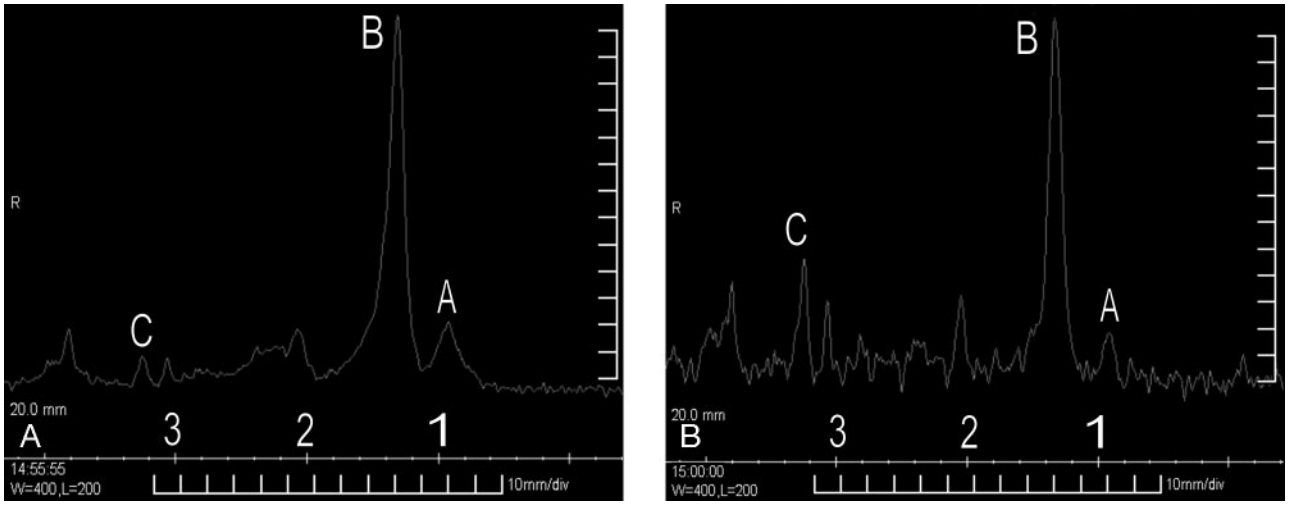

Fig 3. Single-voxel PRESS MR spectroscopy (*A*; TR/TE = 1500ms/35ms). Adequate water suppression has been achieved. Reading from right to left, *peaks A* and *B* (tall and broad) at 0.9 and 1.33 ppm, respectively, represent typical long-chain lipids (lipid/lactate). NAA and Cr are barely detectable. A small Cho peak, *C*, is seen to resonate at 3.2 ppm. Long TE MR spectroscopy (*B*; TR/TE = 1500ms/144ms) depicts persistence of predominant lipid peak at 1.33 ppm.

tive to voxel volume. 3D multivoxel proton spectroscopy with 2D chemical shift imaging interrogates relatively small voxel volumes and overcomes this paradox.[12] Meningiomas invariably have elevated alanine. A high lipid/Cr ratio may also be noted.[10] "Finger-printing" of *M tuberculosis* cell-wall biochemicals in tuberculomas is now possible, facilitating their detection.[13] T1-weighted magnetization transfer MR imaging is a useful adjunct to MR spectroscopy and shows promise in tissue characterization of CNS tuberculomas.[13]

Conclusions

When diagnostic dilemmas present themselves, MR spectroscopy considered in perspective with MR imaging and clinicopathologic features can be useful in certain situations. Rare as

they are, extra-axial tuberculomas may masquerade as meningiomas. To our knowledge, ours is the first report of an extra-axial "giant" tuberculoma that bore a striking resemblance to meningioma and in which diagnostic confirmation was obtained using proton MR spectroscopy that was later corroborated by surgical biopsy and histopathology.

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