A cerebral mass in a patient with Churg Strauss syndrome

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Abstract

Background Differential diagnosis of mass brain lesions with surrounding peripheral-ring enhancement includes infections, tumours, demyelinating diseases, and vascular lesions such as infarcts or haematomas.

Methods This paper is the case report of a 72-year-old Caucasian female patient who presented with a subacute onset neurologic deficit and a heterogeneous cerebral mass, an imaging finding worrisome for malignancy.

Conclusion More specific brain imaging is necessary to differentiate between different diseases, especially malignant CNS tumours and abscesses. Specific risk factor identification is important but cannot replace stereotactic aspiration of pus for accurate microbiologic diagnosis and initiation of targeted antimicrobial treatment of cerebral abscesses.

Differential diagnosis of mass brain lesions with surrounding peripheral-ring enhancement includes pyogenic brain abscesses, other infections (e.g. toxoplasmosis), malignant tumours, demyelinating diseases, and vascular lesions such as infarcts or haematomas. More specific brain imaging is necessary to differentiate between different diseases, especially malignant central nervous system (CNS) tumours and abscesses.

Specific risk factor identification such as otitis media, mastoiditis, paranasal sinusitis, dental infection, cyanotic heart disease, bacterial endocarditis, pyogenic lung disease, T-cell deficiency and trauma is important but cannot replace stereotactic aspiration of pus for accurate microbiologic diagnosis and initiation of targeted antimicrobial treatment of cerebral abscesses. It can guide empiric treatment though in patients too ill to undergo any form of intervention.

Case report

A 72-year-old Caucasian female with past medical history of Churg Strauss syndrome on steroids and methotrexate, aortic stenosis, asthma, achalasia, atrial fibrillation on anticoagulation, presented with subacute onset of left-sided weakness. She denied headache, loss of consciousness, or seizures. She did not report any fever, sinus or ear pain, vomiting. She had no recent change in her numerous chronic medical conditions.

On admission, vital signs were significant for tachycardia and physical examination revealed left facial droop and left-sided hemiparesis. The remainder of the examination was normal.

CT scan of the brain revealed a large area of low density in the right frontal lobe suggestive of a mildly heterogeneous underlying mass lesion. Further workup with
Gadolinium-enhanced magnetic resonance imaging of the brain revealed a conglomeration of ring enhancing lesion in the right frontal lobe (Figure 1) that demonstrated diffusion restriction (low signal in apparent diffusion coefficient-ADC-map and high signal in diffusion-weighted imaging (Figure 2 and 3), with an exuberant amount of surrounding oedema (high signal in FLAIR imaging) (Figure 4), findings suggestive of a brain abscess.

Right frontal craniotomy with use of frameless stereotaxy was performed to evacuate the intracranial abscesses. Culture of the pus yielded *Nocardia asteroides* and *Streptococcus viridans*.

Postoperative antibiotic treatment with trimethoprim-sulfomethoxazol and ceftriaxone was started.
Three months after initiation of treatment, there was minimal residual neurologic
deficit and repeat imaging showed marked interval improvement in the size of the
surgical cavity and in surrounding oedema.

Discussion

In the United States, it has been estimated that 500–1000 new cases of *Nocardia*
infeciton occur annually. Cerebral nocardiosis is rather uncommon, representing only
2% of all cerebral abscesses.

Our patient presented with a subacute onset neurologic deficit and a heterogeneous
cerebral mass, an imaging finding worrisome for malignancy. Differential diagnosis
between cerebral abscesses and tumours is greatly improved by the adjunct of
diffusion-weighted imaging (DWI) and perfusion-weighted imaging (PWI); yet there
is still overlap. Proton MR spectroscopy is useful in discriminating abscesses from
cystic tumours or even identifying the nature of the abscess—whether it has a
pyogenic, tubercular, or fungal origin. The definitive microbiological diagnosis,
though, is made by culture.

In our case the patient had impaired cellular immunity secondary to chronic
methotrexate and corticosteroid use, an established predisposing risk factor for the
development of nocardial brain abscess. Treatment of nocardial brain abscess includes
a combination of antibiotics, surgical debridement and improvement of immune
function. First line antibiotics are the sulfonamides, mainly TMP-SMX, and the
duration of treatment is 12 months.

Second-line medical treatment is amikacin, ciprofloxacin, imipenem, minocycline,
3rd-generation cephalosporins. Some authors have recommended medical
management alone in immunocompetent patients with a brain abscess less than 2 cm
in diameter and an established diagnosis of *Nocardia* spp. from an extraneural
source. The source of the infection should be treated surgically or medically to
prevent recurrence of the abscess, though the primary focus, mostly pulmonary,
cannot be always identified.

Mortality reaches 75–90% for immunocompromised patients, and patients with
disseminated nocardiosis or with multiple brain abscesses. Mortality is also higher
(50%) for patients undergoing needle aspiration compared to patients undergoing
open craniotomy (24%).

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