

Toxoplasma encephalitis in a young immunocompetent man with ischemic stroke as initial presentation

A. Romano¹, M. Distefano¹, P. Profice¹, M. Fantoni², C. Colosimo³, G. Della Marca¹

¹Institute of Neurology, Fondazione Policlinico Universitario A. Gemelli, Catholic University of the Sacred Heart, Rome

²Institute of Infectious Diseases, Fondazione Policlinico Universitario A. Gemelli, Catholic University of the Sacred Heart, Rome

³Institute of Radiology, Fondazione Policlinico Universitario A. Gemelli, Catholic University of the Sacred Heart, Rome

Introduction

Toxoplasmosis is a worldwide spread infection caused by *T. gondii*. In immunocompetent hosts, acute acquired *T. gondii* infection is usually asymptomatic or oligosymptomatic with self-limited course. In immunocompromised patients, it's possible a reactivation of a latent infection, which typically involves CNS, characteristically in the form of multiple abscess-like round processes with ring enhancement.

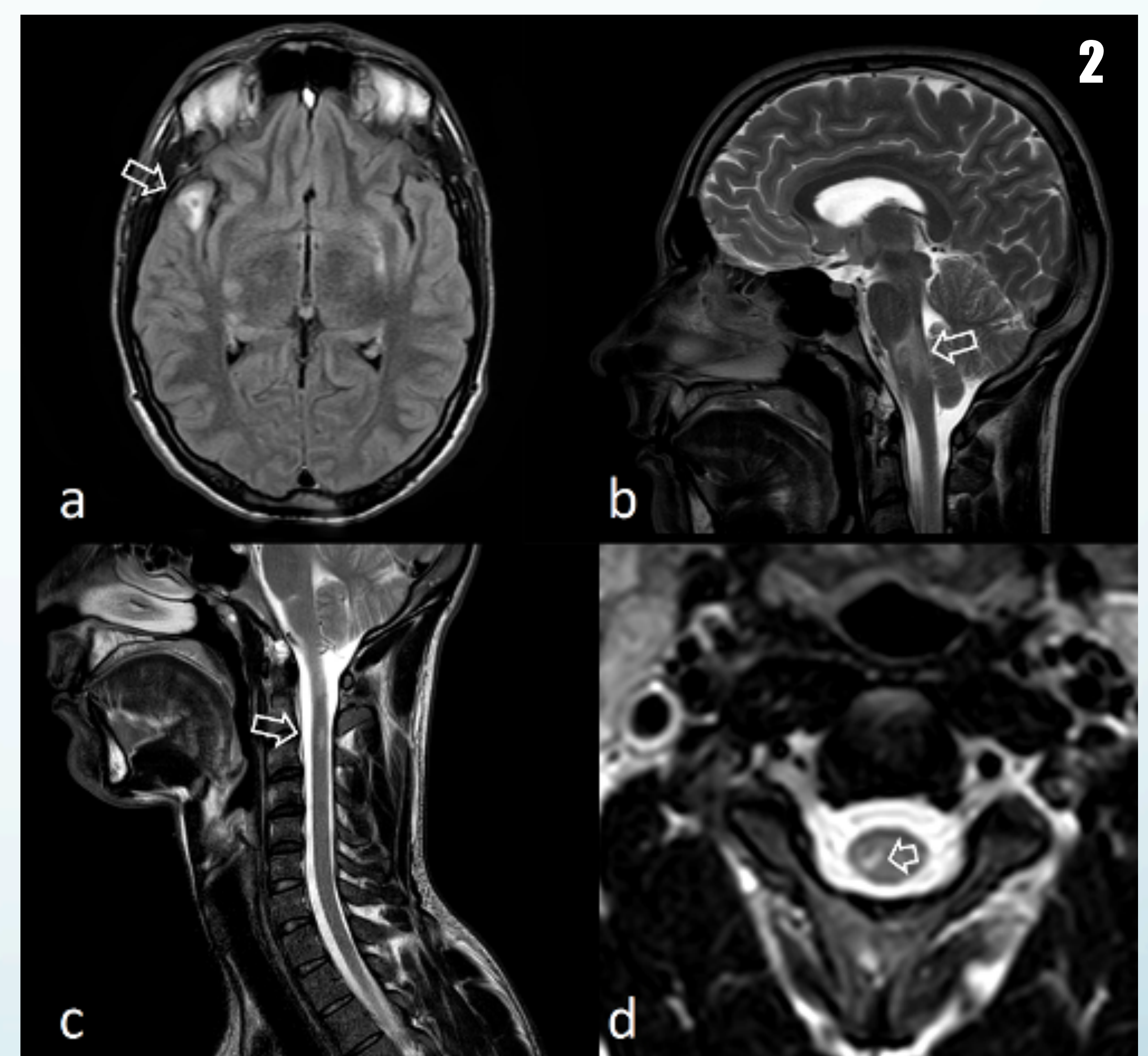
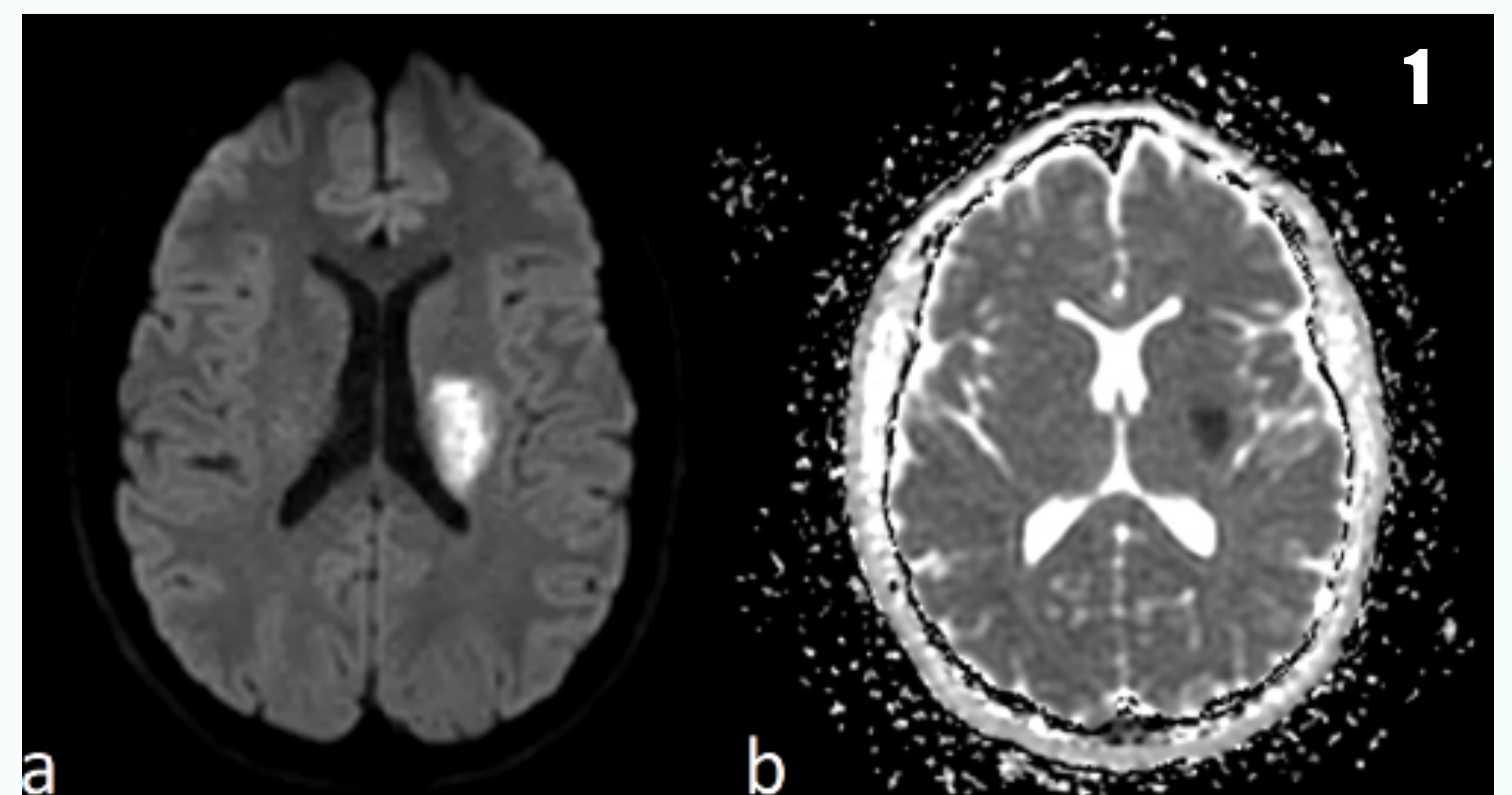
While neurotoxoplasmosis is well documented in immunocompromised individuals, it is rare and often undetected in immunocompetent hosts.

Here we describe a case of encephalomyelitis associated with acute *T. gondii* infection in an immunocompetent young man, with an ischemic stroke as initial presentation.

Case Report

M, 25 year-old, acute right FBC hemiparesis

- No remarkable past medical history (no trauma, fever, vaccination or drug abuse)
- Emergency brain MRI (fig. 1): acute ischemic lesion in left corona radiata
 - Thrombolysis with partial benefit
- Laboratory: mild eosinophilia ($0.59 \times 10^9/L$), high serum IgE (1073 UI/mL)
- Epi-aortic US, TT/TE echocardiogram, Holter EKG: normal
- Brain-spine MRI performed 7 days later (fig. 2): new supratentorial, infratentorial and spinal lesions, hyperintense on T2-w/FLAIR images, without diffusion restriction, some of them with slight CE. Peculiar the right temporal lesion (fig. 2a), showing a central hypointense region, surrounded by an hyperintense area, with mild peripheral enhancement
- CSF examination: proteins 46 mg/dl, cells 28/mm³, normal glucose content; no oligoclonal IgG bands
- Microbiology:
 - serological testing and CSF PCR for neurotropic viruses negative (including serology for HIV-1/2)
 - serology for *T. gondii*: serum IgM 8.68 S/CO, serum IgG >250 UI/mL with low IgG avidity (0.206), suggestive of a recent infection; PCR for *T. gondii* on CSF negative
- Treatment: anti-Toxo therapy (sulfadiazine and pyrimethamine plus leucovorin) + high dose IV steroids
- Follow-up MRI performed 5 days after beginning therapy: partial regression of CNS lesions



Conclusions

In this case, clinical course and neuroimaging suggest that CNS involvement is induced with different etiological mechanisms:

- the **direct action of the microorganism** (suggested by the peculiar signal features of the temporal lesion)
- a **vasculitic pathogenesis** (consistent with the stroke presentation at the onset)
- an **inflammatory, post-infectious pathogenesis** (suggested by the distribution of the lesions, the contrast enhancement and the regression after steroid treatment)

References

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