HIV-associated cerebral toxoplasmosis in Ireland:

A case series highlighting diagnostic and therapeutic complexities

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3 adults over 12 months

Advanced HIV, focal neurology, CD4 5–92 cells/μL and positive Toxoplasma gondii IgG.

Atypical imaging spectrum

Calcific foci, haemorrhagic foci, deep thalamic disease, hydrocephalus and variable enhancement.

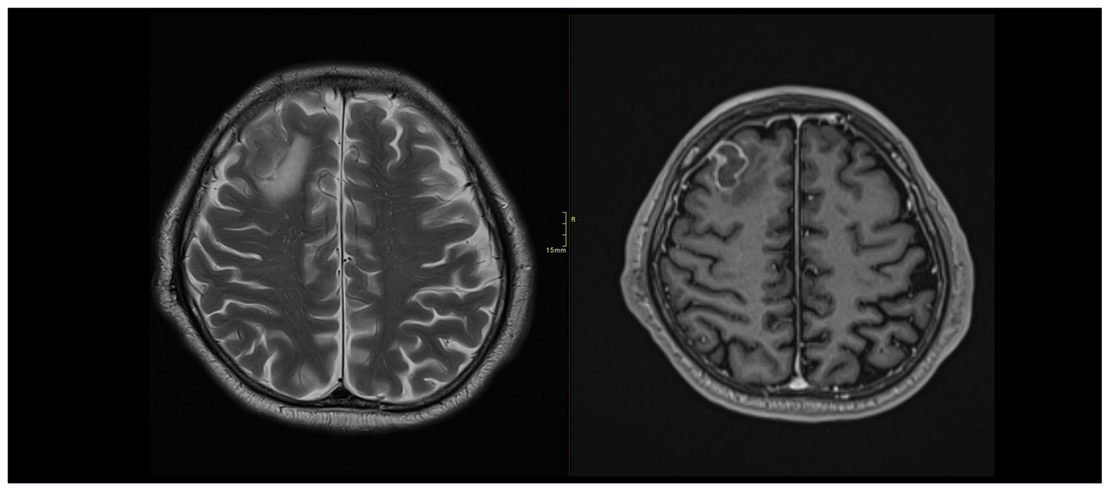
Importance of MRI

Greater lesion burden, improved morphology assessment and more reliable follow-up than CT alone.

IMAGING FINDINGS:

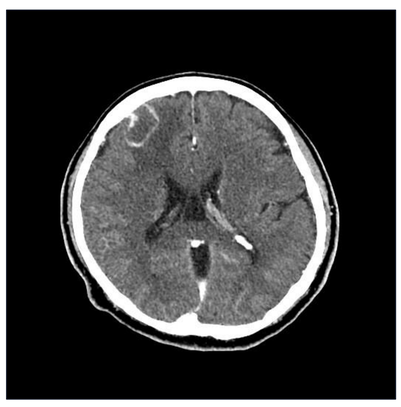
CASE 1 | Irregular hyperattenuating ring-enhancing lesions with oedema and calcific foci

Pre-contrast and post-contrast CT showed multiple irregular hyperattenuating/ring-enhancing lesions at the grey–white junction and basal ganglia with oedema, mass effect and calcific foci.

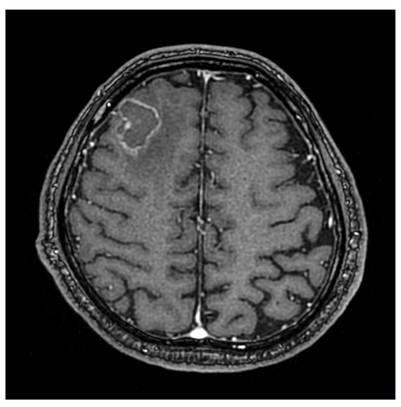


Serial MRI demonstrated only slight reduction in lesion size and surrounding oedema after 6 weeks, illustrating delayed radiological response despite treatment.

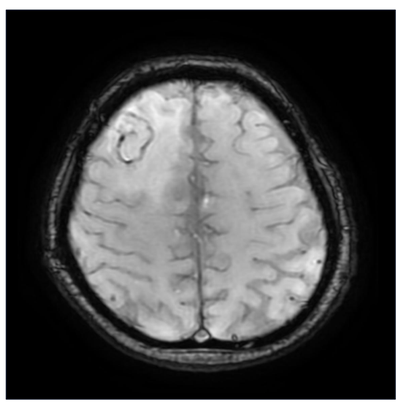
CASE 2 | CT suggested solitary frontal disease; MRI demonstrated additional lesions and haemorrhagic foci

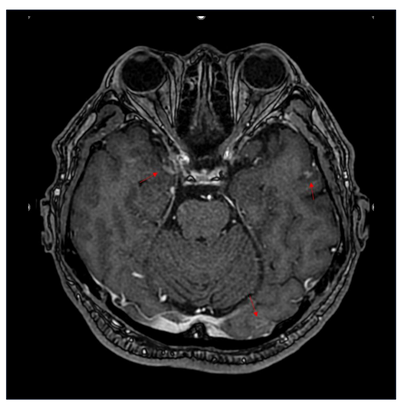


Initial CT showed a 2.3 × 1.7 cm right frontal lesion with vasogenic oedema and mild frontal horn effacement.



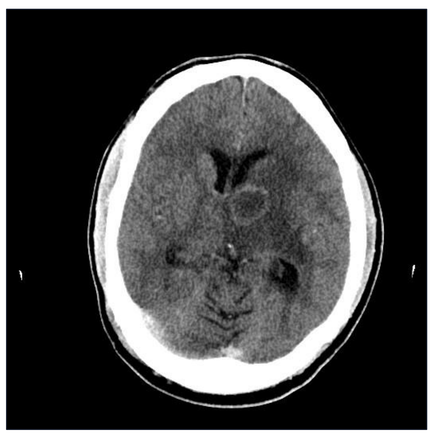
MRI better characterised lesion morphology, including mural/wall nodularity, additional lesions and SWI haemorrhagic foci at the lesion margins.

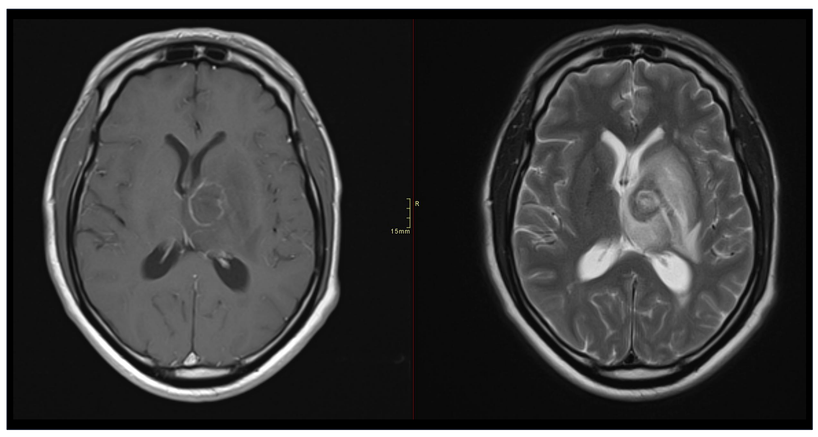


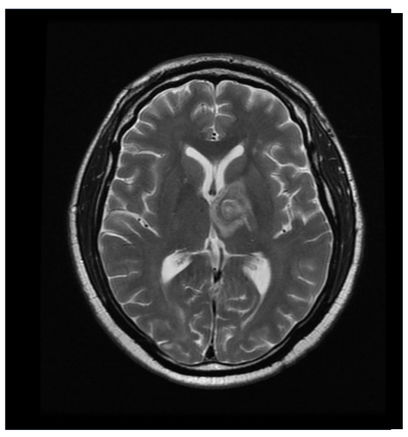


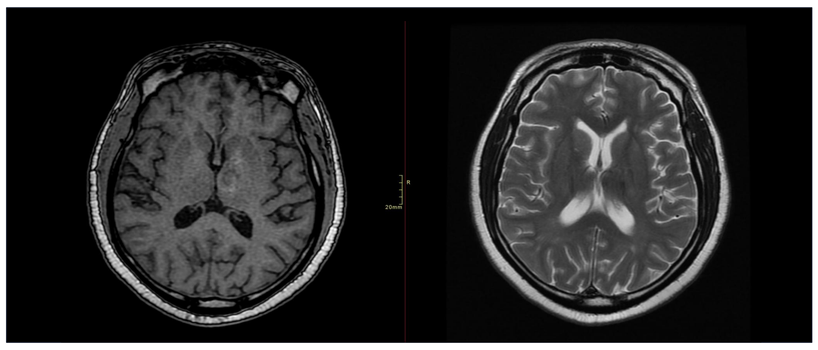
Follow-up imaging subsequently showed interval decrease in lesion size and perilesional oedema, supporting therapeutic response.

CASE 3 | Deep thalamic lesion with mass effect, third-ventricle compression and evolving hydrocephalus









Dominant centrally cystic/necrotic left thalamic rim-enhancing lesion with marked oedema and mass effect caused obstruction at the third ventricle and evolving hydrocephalus.

MRI (right) performed after 3 weeks of therapy demonstrates persistent signal abnormality in a similar distribution to before, particularly involving the left thalamus. However, appearances have significantly improved compared with the previous study

Serial MRI (left) post treatment completion showed significant improvement, resolution of oedema and continued reduction in residual lesion size on later outpatient imaging.

KEY CLINICAL INFORMATION

PRESENTATION

- Case 1: gait ataxia; multifocal lesions on CT/MRI.
- Case 2: headache and foot drop after PJP; CT suggested a solitary frontal lesion.
- Case 3: subacute cognitive change; deep thalamic lesion causing obstructive hydrocephalus.

MANAGEMENT

- All received pyrimethamine–sulfadiazine with folinic acid.
- Corticosteroids were used where mass effect/hydrocephalus was clinically relevant.
- Two developed sulfonamide hypersensitivity and switched to pyrimethamine–clindamycin.
- Additional PJP prophylaxis was required after regimen change.

CLINICAL AND RADIOLOGICAL COURSE

- ART started during acute treatment (day 0–12); no IRIS observed.
- Case 1: delayed radiological response until approximately 6 weeks; psychosis and later status epilepticus.
- Case 2: partial regression by 3 weeks.
- Case 3: rapid clinical/radiological improvement by 3 weeks with hydrocephalus resolution.

ROLE OF MRI

- CT underestimated lesion burden in this series.
- Atypical features broadened the differential to malignancy and primary CNS lymphoma.
- MRI clarified lesion distribution and morphology.
- Serial MRI was most useful when clinical response was subtle or discordant.

CONCLUSION

In people living with advanced HIV and focal brain lesions, cerebral toxoplasmosis should remain a leading differential even when imaging is atypical.

Early MRI, empiric therapy, follow-up imaging, toxicity monitoring and multidisciplinary support are key to optimising outcomes the radiological course may lag behind clinical improvement, so response should be assessed using serial MRI plus clinical context.
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