



Improved detection of MOG antibody-associated transverse myelitis with 18F-FDG-PET: a case report

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Introduction

Transverse myelitis is a rare disorder that has been associated with vaccination, infections, autoimmune and systemic diseases or malignancies [1]. Diagnosis is based on clinical findings and is supported by neuroimaging and CSF analysis. We describe a patient with myelin oligodendrocyte glycoprotein (MOG) antibody-associated transverse myelitis in which the spinal cord 18F-fluorodeoxyglucose positron emission tomography (18F-FDG-PET) was of significant diagnostic value in the presence of ambiguous MRI findings.

Case description

A 75-year-old male was admitted with progressive gait difficulties and micturition initiation problems for 4 days, accompanied by a tense feeling with back pain at the mid-thoracic level and numbness of his feet. His relevant medical history stated prostatectomy for prostate malignancy

fourteen years earlier. Twelve days before his admission, he received a first dose of ChAdOx1 nCoV-19. Vital parameters were normal with a body temperature of 36.0 °C. Physical examination showed a broad-based gait with flaccid paresis of the right leg. Tendon reflexes were normal. However, plantar reflexes demonstrated extension bilaterally. Sensory testing showed band-like numbness at the level of Th6 and on both soles and lateral sides of the feet. Upper limb and cranial nerve examinations were normal. Rapid clinical deterioration would be observed in the following three days, with the occurrence of left leg paresis and subsequent progression to flaccid paraplegia, as well as mild right hand weakness and bilateral upper limb ataxia. By that time, dysarthria, leftward tongue deviation, first degree horizontal nystagmus on left, right and upward gazing, and diplopia were present.

Cerebral and thoracolumbar spinal CT were normal. Blood examination demonstrated mild leukocytosis (11,500/ μ L, 74% neutrophils) with normal CRP. Serological testing for *B. burgdorferi*, *T. pallidum*, tuberculosis (QuantiFERON) and HIV was negative. Blood cultures remained sterile. SARS-CoV-2 PCR on nasopharyngeal swab was negative. CSF analysis showed pleocytosis (27/ μ L, 92.6% lymphocytes), elevated protein (79 mg/dL), normoglycorrhachia (69% of serum glucose) and increased lactate (2.6 mmol/L), after which empirical antimicrobial treatment with aciclovir, amoxicillin and ceftriaxone was initiated. IgG index was normal (0.54) and CSF-restricted oligoclonal bands were absent. *L. monocytogenes*, *Herpes simplex virus*, *Varicella-zoster virus* and Enterovirus PCR were negative. Aerobic and anaerobic CSF cultures remained sterile and Gram stain was negative. Paraneoplastic antibodies were absent in the CSF. Histopathological CSF examination showed inflammatory changes.

Brain and spinal cord MRI on the second and third day of admission, respectively, showed no clear signs of inflammation, infection or infarction, nor gadolinium enhancement, although the MRI image at the cervical level was inconclusive due to high noise and movement artifacts. (Fig. 1a–d).

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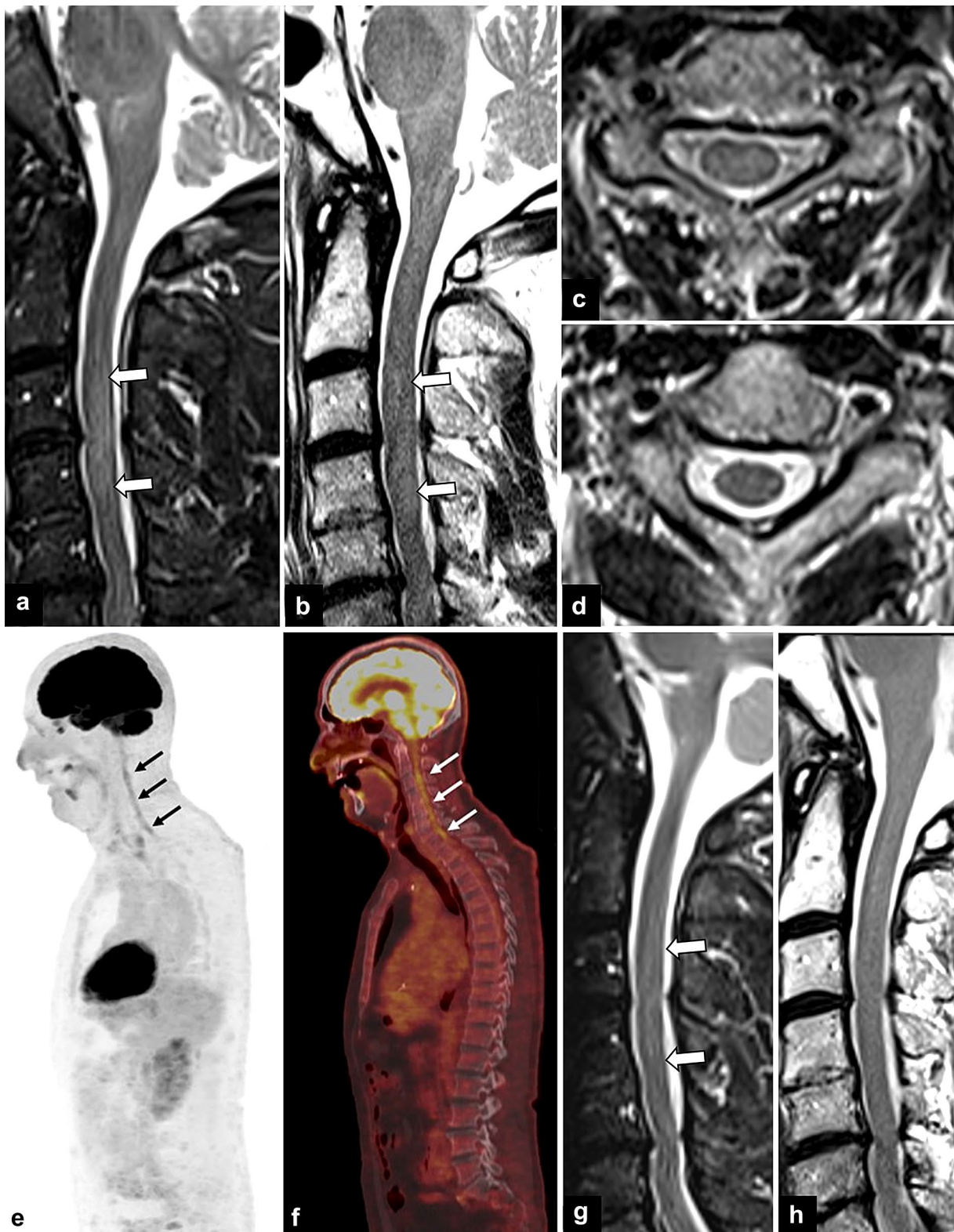
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To exclude an underlying malignancy or occult inflammation, 18F-FDG-PET/CT was performed, which showed a diffusely increased metabolism of the cervical spinal cord (Fig. 1e–f).

In the further etiological work-up, anti-nuclear factor was positive (titer 1:80, nuclear homogeneous pattern [AC-1]). ANCA, serum angiotensin converting enzyme and serum anti-neuronal, autoimmune encephalitis, anti-aquaporin-4

Fig. 1 MRI of the cervical spine and whole body 18F-FDG-PET scan during hospitalization and MRI of the cervical spine six months after treatment. On initial MRI of the cervical spine (a–d), the spinal cord is slightly hyper-intense on the sagittal STIR images (a) and sagittal T2-weighted images (b) at the C3–C4 level (arrows) but also at the level of the pons and medulla oblongata, possibly indicating an artifactual nature of the hyper-intensity at the C3–C4 level. The signal abnormalities are only discretely visible on the axial T2-weighted images (c). Normal axial T2-weighted image of the spinal cord at the C2 level for comparison (d). 18F-FDG-PET scan at diagnosis (e–f), with Maximum Intensity Projection (MIP, e) and sagittal image (f) showing a diffuse increased 18F-FDG uptake in the cervical spinal cord. Follow-up MRI of the cervical spine six months after diagnosis and treatment (g–h), showing discrete signal hyper-intensity on the C3–C4 level (arrows) on the sagittal STIR images (g) but not on the T2-weighted images (h). The spinal cord also appears slightly thinner than on the initial MRI. STIR: short tau inversion recovery

and antiganglioside antibodies were negative or normal. MOG antibodies were detected in the serum by commercially available fixed cell-based assay (Eurimmun) and externally confirmed by live cell-based assay on Fluorescence Activated Cell Sorting (laboratory Sanquin, Amsterdam, the Netherlands).

On day 4 of hospitalization, intravenous immunoglobulins (IVIg, 0.4 g/kg/day for 5 days) were initiated. On day 15, intravenous methylprednisolone (1 g/day for 3 days) was started, without oral tapering. The antimicrobial therapy was discontinued when PCR and cultures revealed no infectious agent.

After initiating IVIg, cranial nerve and upper extremity symptoms fully recovered within a few days. The flaccid paraplegia recovered to a proximal paresis within two weeks. Bladder function did not improve at this time. After 3 months of inpatient rehabilitation, physical examination showed a spastic gait with a range of 500 m without support, decreased vital sensitivity from Th10 downwards and flexor plantar reactions. His bladder function had improved, although the need for manual abdominal pressure to initiate micturition persisted. On MRI of the cervical spinal cord six months after diagnosis and treatment, the equivocal cervical medullary hyper-intensity was no longer visible on the T2-weighted images and had decreased on the short tau inversion recovery (STIR) images (Fig. 1g–h). MOG antibodies remained detectable in the serum.

Discussion

Myelin Oligodendrocyte Glycoprotein antibody-associated disease (MOGAD) is a recently recognized rare neuro-inflammatory disease that can present with transverse myelitis [2]. Spinal cord MRI has been found to be normal in up to 10% of MOGAD patients when performed within 6 weeks of the beginning of a first clinical myelitis [3]. Our patient showed questionable medullary MRI signal abnormalities

confined to the C3 and C4 level, whereas a whole-body 18F-FDG-PET/CT, performed to rule out a paraneoplastic myelopathy, demonstrated a diffuse, slightly increased cervical spinal cord metabolism. This case therefore demonstrates that 18F-FDG-PET/CT may be a promising complementary diagnostic tool, potentially with a higher sensitivity than MRI, in acute MOG antibody-associated myelitis.

Of note, in our patient transverse myelitis developed following administration of the first dose of ChAdOx1 nCoV-19. Five other cases of proven MOG antibody-associated disease with extensive spinal cord lesions occurring after SARS-CoV-2 vaccination have been described [4–6]. In these cases, the spinal cord lesions were visualized on MRI and PET/CT was not performed.

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Declarations

Conflict of interest The authors declare that there is no conflict of interest.

Informed consent Informed consent was obtained from the case patient and is attached to this submission as a separate file.

Consent to publish The participant has consented to the submission of the case report to the journal.

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