RESEARCH PAPER

Acute Urinary Retention with Bilateral Hydroureteronephrosis as the Heralding Feature of Myelin Oligodendrocyte Glycoprotein Antibody-Associated Disease in a 10-Year-Old Boy: A Case Report

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Abstract

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is an inflammatory demyelinating disorder distinct from multiple sclerosis and from aquaporin-4 (AQP4)-IgG seropositive neuromyelitis optica spectrum disorder (NMOSD), and accounts for a substantial proportion of acquired demyelinating syndromes in children. We report a 10-year-old boy admitted with three days of lower abdominal pain and one day of difficulty in passing urine, ten days after a febrile illness treated elsewhere as meningitis. He had a tense, palpable bladder, exaggerated knee jerks, bilaterally extensor plantar responses and absent superficial abdominal reflexes, with normal power in all four limbs and no sensory level. Ultrasonography showed a pre-void bladder volume of 310 mL with a post-void residual of 260 mL and bilateral mild hydroureteronephrosis. Magnetic resonance imaging demonstrated T2 hyperintensity with cord oedema from C3 to T1 and subtle hyperintensity over both caudate nuclei. Cerebrospinal fluid (CSF) showed 50 cells/cumm with 85% lymphocytes and protein 44.8 mg/dL, with negative stains, culture and nucleic acid amplification testing. On a working diagnosis of NMOSD he received intravenous methylprednisolone 30 mg/kg/day for five days followed by oral prednisolone. A paired NMO-MOG panel showed serum AQP4-IgG negative and serum MOG-IgG strongly positive, with both CSF antibodies negative, and the diagnosis was revised to MOGAD. Bladder recovery lagged behind: the catheter required reinsertion twice before a graded trial of micturition succeeded on the fourteenth day, and he was discharged on the eighteenth day voiding spontaneously on a tapering steroid course. Acute urinary retention with hydroureteronephrosis may herald MOGAD myelitis in a child; serum rather than CSF is the specimen of choice, and an NMOSD label should be withheld until antibody results are available.

Keywords: Myelin oligodendrocyte glycoprotein antibody-associated disease; Longitudinally extensive transverse myelitis; Acute urinary retention; Neurogenic bladder; Hydroureteronephrosis; Paediatric demyelinating disease.

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Introduction

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is an antibody-mediated inflammatory disease of the central nervous system, defined by serum immunoglobulin G directed against myelin oligodendrocyte glycoprotein in the presence of a core demyelinating event such as optic neuritis, myelitis, acute disseminated encephalomyelitis (ADEM) or cerebral cortical encephalitis.[1] It is disproportionately a disease of childhood, and in paediatric cohorts of acquired demyelinating syndromes MOG-IgG is detected in a sizeable minority of children, often with phenotypes that differ from those seen in adults.[2,3] Because the clinical and radiological phenotypes overlap considerably with those of neuromyelitis optica spectrum disorder (NMOSD), a child presenting with longitudinally extensive transverse myelitis (LETM) is frequently labelled as having NMOSD before antibody results return.[4,5] The distinction is not academic: the two diseases differ in natural history, in relapse risk and in the threshold for long-term immunosuppression, and an incorrect early label tends to follow a child through subsequent discharge summaries and referrals.[5,6]

Myelitis in MOGAD has a particular tendency to involve the lower cord and conus medullaris, and bowel or bladder dysfunction is reported in a large

majority of patients at presentation.[7] A child may therefore reach hospital not because of weakness or visual loss but because he cannot pass urine, and in that setting the spinal cord is considered only when something in the neurological examination refuses to fit the urological story. We report such a child, in whom acute retention of urine with bilateral hydroureteronephrosis led, by way of an unexpected pair of extensor plantar responses, to a long cervicothoracic cord lesion and ultimately to a diagnosis of MOGAD.

Case Presentation

History

A 10-year-old boy was admitted in April 2026 with lower abdominal pain of three days' duration — sudden in onset, gradually progressive, intermittent, largely suprapubic and worse in the early morning. For one day before admission he had had difficulty in passing urine with frequency, urgency and terminal dribbling, but no burning micturition and no haematuria.

Ten days earlier he had developed low-grade intermittent fever with frontal headache and had been admitted for five days to a hospital in a neighbouring district, where he was treated as meningitis with intravenous meropenem, vancomycin and dexamethasone. Magnetic resonance imaging (MRI) of the brain with contrast and a lumbar puncture had been performed there, and he was discharged on oral levetiracetam, which the parents continued for five days and then stopped on their own; no seizure was ever witnessed or documented. Periorbital and facial swelling had been noted during that admission. The cerebrospinal fluid (CSF) culture from that hospital was subsequently traced and showed no growth at 48 hours; no other records were retrievable.

There was no history of trauma, limb weakness, visual disturbance, difficulty in swallowing or walking, constipation or faecal incontinence. He was born at term by normal vaginal delivery at home, weighed 2.8 kg and required no neonatal admission; development was age-appropriate and he was studying in the fourth standard. Immunisation was complete only up to nine months of age and the BCG scar was absent. The parents were second-degree consanguineous relatives, a 13-year-old sibling was well, and there was no family history of seizures, tuberculosis or neurological illness. The family belonged to socioeconomic class III with poor environmental sanitation, and dietary recall showed an intake of about 747 kcal and 10.5 g of protein daily against an expected 1,000 kcal and 13 g.

Examination

He looked unwell. The pulse was 128/min, blood pressure 110/70 mmHg (approximately the 90th centile for age, sex and height), respiratory rate 28/min, temperature 99.1 °F and capillary refill time 2 seconds. He weighed 22 kg against an expected 32.5 kg (67% of expected, below the 3rd centile), measured 130 cm against an expected 137 cm, and had a body mass index of 13.0 kg/m², also below the 3rd centile; head circumference was 50.5 cm and chest circumference 60 cm. Periorbital puffiness and facial swelling were present; there was no pallor, icterus, clubbing or lymphadenopathy. The abdomen was soft with lower abdominal tenderness and a tense, palpable bladder reaching above the symphysis pubis, without organomegaly. Respiratory and cardiovascular examinations were unremarkable.

He was conscious and oriented. Higher mental functions and all cranial nerves were intact, pupils were equal and reactive, and there was no facial asymmetry or bulbar weakness. Tone was normal and power was 5/5 in all four limbs. The knee jerks were exaggerated, deep tendon reflexes were graded 3+, and the plantar responses were extensor bilaterally. The superficial abdominal and cremasteric reflexes were absent. There was no sensory loss, no demonstrable sensory level, no spinal tenderness and no meningeal sign. The combination of an overfilled, atonic bladder with brisk reflexes, bilateral Babinski signs and absent superficial reflexes in a child with entirely normal limb power was the finding that redirected the evaluation from the urinary tract to the spinal cord.

Investigations

The principal laboratory, CSF and imaging findings are summarised in Table 1. The admission haemogram showed a neutrophilic leucocytosis of 20,900/cumm that settled progressively over the following week; the erythrocyte sedimentation rate was raised while C-reactive protein was only marginally raised on a single occasion. Renal function was normal throughout, with mild hyponatraemia on admission and transient hypokalaemia corrected with oral potassium chloride. Urine routine examination was normal on two occasions and both blood and CSF cultures were sterile. Because of the periorbital oedema, raised anti-streptolysin O titre and borderline-high blood pressure, serum complement C3 was measured and was normal, making post-streptococcal glomerulonephritis unlikely; serial urine albumin was trace to negative and a nephrotic chart maintained for three days showed no evidence of nephrotic syndrome.

Ultrasonography on the day of admission showed a distended bladder with internal echoes and dependent debris, a pre-void volume of 310 mL and a post-void residual of 260 mL, with bilateral mild hydroureteronephrosis attributed by the reporting radiologist to the over-distended bladder rather than to structural obstruction. Both kidneys were of normal size with preserved cortical thickness, and the remaining abdominal viscera were normal. A urinary tract infection was suggested radiologically but was not supported by urinalysis.

The contrast MRI of the brain performed at the referring hospital twelve days before admission had shown a small left retrocerebellar arachnoid cyst and suspicious bilateral leptomeningeal enhancement for which meningitis could not be excluded, with no diffusion restriction and normal brainstem and deep nuclei; the spine had not been imaged. MRI of the brain and whole spine on the second hospital day showed T2 hyperintensity with cord oedema extending continuously from C3 to T1, subtle T2 hyperintensity over both caudate nuclei and a prominent cisterna magna.

Lumbar puncture was performed on the third hospital day at the L4–L5 interspace after written informed consent; the child tolerated the procedure well. A paired serum and CSF NMO-MOG panel was requested the same day and sent to a national reference laboratory, which recorded sample collection on the fifth hospital day. The report, received on the seventh hospital day, showed serum anti-aquaporin-4 (AQP4) IgG negative and serum anti-MOG IgG strongly positive, with both antibodies negative in the CSF. The report was qualitative; neither the assay platform nor an antibody titre was stated.

Differential diagnosis

The differential diagnosis entertained by the treating unit included tuberculous meningitis with lateral cord involvement, acute transverse myelitis, post-infectious immune-mediated myeloradiculitis, optic neuritis, ADEM and a brainstem syndrome. Acute flaccid paralysis syndromes, including Guillain-Barre syndrome and poliomyelitis, were formally considered and a surveillance notification completed, but both were improbable in a child with exaggerated knee jerks, bilaterally extensor plantar responses, retained limb power and a longitudinally extensive cord lesion rather than areflexia and ascending weakness.

Treatment and Hospital Course

A urethral catheter was passed on admission and the bladder decompressed. Because of fever, neutrophilic leucocytosis and CSF pleocytosis, empirical intravenous ceftriaxone 100 mg/kg/day was started on admission and continued for ten days; intravenous vancomycin 20 mg/kg/dose six-hourly was added on the fifth day when fever spikes persisted and was given for ten days, with amikacin held in reserve should fever recur or the urine culture become positive. Once the MRI and clinical findings had been reviewed, the neurology opinion was that the picture was one of transverse myelitis, and the working diagnosis recorded was NMOSD pending antibody results. Intravenous methylprednisolone 700 mg (30 mg/kg) once daily over one hour was begun on the fourth hospital day and continued for five consecutive days. The catheter was clamped and removed on the sixth day, after three doses of methylprednisolone, but retention recurred and the bladder had to be drained again.

On the eighth day the blood pressure rose to 122/80 mmHg with headache and dizziness, and he was transferred to the paediatric intensive care unit; tamsulosin was withheld and an infusion of 3% saline at 0.5 mL/kg/hour was started for presumed raised intracranial pressure. He remained haemodynamically stable, the headache settled, and he returned to the ward the following day; the hypertonic saline was tapered and stopped on the twelfth day. The reference laboratory report, available on the seventh day, showed a strongly positive serum MOG-IgG with a negative AQP4-IgG, and the diagnosis was formally revised from NMOSD to MOGAD. Oral prednisolone 20 mg twice daily (2 mg/kg/day) was started on the ninth day, and tamsulosin 0.2 mg once daily was added for the neurogenic bladder. Intravenous immunoglobulin was planned on two occasions, contingent on a follow-up MRI showing lesion progression and on obtaining cover under a state health-financing scheme; neither condition was met before the child began to improve, and immunoglobulin was not administered. Weekly cholecalciferol 60,000 IU, oral lansoprazole, oral glycerol and oral mecobalamin for the low serum vitamin B12 were added.

Bladder recovery was the slowest element of the illness. After the second episode of retention the catheter was reinserted on the ninth day on neurological advice. From the twelfth day the catheter was clamped intermittently and a graded trial of micturition instituted. It was finally removed on the fourteenth day, after which the child voided spontaneously at three- to four-hourly intervals with a urine output of 3 to 5 mL/kg/hour and no clinically demonstrable residual volume. The complete chronology is set out in Table 2.

Ophthalmological assessment on the day of admission was entirely normal, with clear media, pink discs, well-defined margins and a cup-to-disc ratio of 0.3 in both eyes. Review on the fifth day, prompted by the headache, showed bilateral disc oedema, right more than left, consistent with papilloedema. A further review on the seventeenth day showed resolving papilloedema in both eyes, with visual acuity improving from 6/9 to 6/6 and normal colour vision and contrast sensitivity.

Outcome and Follow-up

The child was discharged on the eighteenth hospital day, conscious, afebrile and haemodynamically stable, with a blood pressure of 110/68 mmHg, a weight of 22.2 kg, no headache or dizziness, normal limb power, and spontaneous voiding five to six times a day with a urine output of 4.5 mL/kg/hour. Fundoscopy showed resolving papilloedema. He was discharged on a structured steroid taper advised by the neurologist: oral prednisolone 40 mg/day for four days, then 30 mg/day for seven days, 20 mg/day for seven days and 10 mg/day for seven days before stopping. He also received weekly cholecalciferol

60,000 IU for six weeks, oral lansoprazole, oral mecobalamin and oral glycerol, with a short course of an antihistamine and calamine lotion for a transient pruritic rash. The parents were counselled about review of bladder symptoms, a repeat MRI and repeat MOG-IgG testing, and were advised to report immediately any recurrence of urinary difficulty, visual blurring or limb weakness. Neither investigation had been obtained at the time of this report.

Discussion

The 2023 International MOGAD Panel criteria require a core clinical demyelinating event, a positive serum MOG-IgG tested by a cell-based assay, and the exclusion of better diagnoses; when the antibody result is clearly positive, supporting clinical or MRI features are not additionally required.[1] This child had acute myelitis with a longitudinally extensive cord lesion, a strongly positive serum MOG-IgG, a negative AQP4-IgG and a negative infective work-up, and therefore satisfies these criteria. The 2015 international consensus criteria for NMOSD are not satisfied: he was AQP4-IgG negative, and only one core clinical characteristic was documented, whereas AQP4-IgG-negative NMOSD requires at least two.[8] The early working label was therefore appropriately revised, and the revision matters because the two conditions differ in relapse risk, prognosis and the threshold for long-term immunosuppression.[5,6,10]

The presentation deserves emphasis. In the largest series of MOG-IgG myelitis, conus involvement was seen in 41% of patients and bowel or bladder dysfunction in 83% at presentation, with longitudinally extensive lesions in 79%.[7] In this child the bladder was not merely involved; it was the presenting organ. A post-void residual of 260 mL from a pre-void volume of 310 mL indicates near-complete failure of emptying, and the hydroureteronephrosis was a direct consequence of chronic over-distension rather than of structural obstruction, as confirmed by normal renal parenchyma and normal renal function. An Indian urodynamic series in NMOSD found urinary retention to be the commonest complaint, present in 72% of patients, with an acontractile detrusor in nearly half and no patient having a normal urodynamic study.[11] One feature of this case runs against the usual explanation. Bladder dysfunction in MOGAD is commonly attributed to conus involvement, yet the demonstrated lesion here was cervicothoracic, extending from C3 to T1, with no conus abnormality reported. Severe detrusor failure with a cervicothoracic lesion is best explained by interruption of the descending autonomic pathways that travel in the lateral columns to the sacral micturition centre, producing a suprasacral neurogenic bladder; the alternative explanation, that a distal cord lesion was present but not captured by what was a screening spine sequence, cannot be excluded. Either way, the clinical implication is the same: a normal-looking conus does not exclude a cord cause for retention. The practical lesson is that a child in acute retention without an obvious urological cause deserves a careful examination of the reflexes before being managed as having a urinary tract infection; here, testing the plantar and superficial abdominal reflexes took under a minute and changed the entire diagnostic trajectory.

The behavior of the bladder during recovery is equally instructive. Retention recurred after the first catheter removal on the sixth day despite three doses of high-dose methylprednisolone, and again after a second attempt; spontaneous voiding returned only after a longer course of corticosteroid, the addition of an alpha-blocker and a deliberately graded trial of micturition with intermittent clamping. Autonomic recovery in MOGAD myelitis lags behind motor recovery, and premature decatheterisation risks repeated over-distension injury to a detrusor that is still recovering. Clean intermittent catheterisation and a structured voiding programme, rather than a single trial of removal, are the safer way to protect the upper tracts.[7,11]

The antibody results illustrate a point of laboratory practice that is still not universally appreciated. Serum and CSF were submitted as a paired panel; the serum MOG-IgG was strongly positive while the CSF MOG-IgG was negative. International recommendations are explicit that serum, tested by a live cell-based assay, is the specimen of choice, and that a negative CSF result does not exclude the diagnosis.[1,6] Had CSF alone been tested, this child would have been reported as antibody-negative and would in all likelihood have retained the NMOSD label. It is relevant that the serum was collected on the fifth hospital day, after intravenous methylprednisolone had already been started and after a course of dexamethasone at the referring hospital, and was nonetheless strongly positive; corticosteroid exposure before sampling reduces but does not abolish detectability. The principal limitation of the antibody data here is that the report was qualitative, with neither assay platform nor titre stated — a recurrent problem when samples are referred to distant laboratories, and one that matters because serial titres carry prognostic weight.[9]

Several contributory diagnoses were weighed. Infective myelitis became untenable after negative CSF stains, culture and nucleic acid amplification testing at two hospitals. The CSF adenosine deaminase of 10.2 U/L fell within the laboratory's equivocal band and, given the limited sensitivity and specificity of this assay in isolation, was not regarded as evidence of tuberculous meningitis against otherwise negative microbiology.[12] Vitamin B12 deficiency can produce a myelopathy with dorsal column signal change,[13] but it does not explain an acute longitudinally extensive cervicothoracic lesion with CSF pleocytosis; the low

level was treated as an incidental finding in a severely undernourished child and was supplemented. The raised anti-streptolysin O titre most probably reflected recent streptococcal exposure, and with a normal C3 and negative urinalysis no causal link with the myelitis is claimed. The subtle bilateral caudate signal change is of interest, since deep grey nuclei involvement and ADEM-like features are described more often in paediatric than in adult MOGAD,[2,3,9] and the leptomeningeal enhancement reported on the earlier outside scan may equally have been inflammatory.

Treatment followed accepted practice. High-dose intravenous methylprednisolone followed by an oral taper is the standard first-line treatment for an acute attack, with plasma exchange or intravenous immunoglobulin reserved for incomplete response.[10] Immunoglobulin was planned twice but not given, escalation having been made conditional on radiological progression that was not demonstrated and on financial cover under a state scheme. The boy recovered fully on corticosteroid alone, so the decision was vindicated by the outcome; nonetheless, the episode is a reminder that in resource-constrained settings the availability of second-line therapy, rather than the indication for it, often determines what is given. Because many children with MOGAD follow a monophasic course, long-term immunosuppression is generally reserved for relapsing disease or persistent antibody positivity, with serial MOG-IgG testing and interval imaging guiding that decision.[6,9,10]

This report has limitations. Follow-up is short and ends at discharge; neither a repeat MRI nor a convalescent MOG-IgG titre was available, so the monophasic or relapsing nature of the disease cannot be established. The formal spinal MRI report, the MOG-IgG assay method and titre, and the records from the referring hospital were not retrievable, and the corresponding details are taken from the treating unit's contemporaneous case record. Sensation was recorded as intact at every assessment and no sensory level was demonstrable, which is unusual in transverse myelitis and may reflect either genuine sensory sparing or the difficulty of eliciting a reliable sensory level in an unwell 10-year-old. The spinal study was performed as a screening sequence, so a distal cord or conus lesion cannot be excluded with certainty. A urine culture result was not available, and nerve conduction studies, although requested, were not reported.

Conclusion

Acute urinary retention with bilateral hydroureteronephrosis can be the first and loudest manifestation of MOGAD myelitis in a child. When a child presents in retention without a urological explanation, the reflexes should be examined before a urinary tract infection is assumed, and a cord cause sought early with MRI of the whole spine. In any child with longitudinally extensive transverse myelitis, serum MOG-IgG and AQP4-IgG should be requested together by a cell-based assay and a diagnosis of NMOSD withheld until those results are available, because MOGAD carries a different prognosis and a different long-term management strategy. Finally, the neurogenic bladder of MOGAD recovers more slowly than the limbs and deserves a planned, graded programme of catheter care rather than a single optimistic trial of removal.

Learning Points

- Urinary retention with bilateral hydroureteronephrosis may herald MOGAD myelitis in a child; extensor plantar responses and absent superficial abdominal reflexes in a child with normal limb power should prompt urgent spinal imaging.
- Serum, not cerebrospinal fluid, is the specimen of choice for MOG-IgG; a negative CSF result does not exclude MOGAD, and a strongly positive serum result remains informative even after corticosteroid exposure.
- A working diagnosis of NMOSD should not be recorded in a child with longitudinally extensive transverse myelitis until paired AQP4-IgG and MOG-IgG results are available.
- Neurogenic bladder in MOGAD may need prolonged catheter care, alpha-blocker therapy and a graded trial of micturition; premature decatheterisation risks further over-distension injury to the upper urinary tract.

Ethical Approval and Patient Consent

This is a report of a single patient and does not constitute a research study involving human participants; accordingly, formal institutional ethics committee approval was not required. Written informed consent for publication of this case and the accompanying clinical and laboratory data was obtained from the parent of the patient, and the child gave assent. All direct and indirect identifiers, including name, hospital registration number and calendar dates of individual events, have been removed, and no identifiable images are included. The report conforms to the CARE guidelines for case reports.[14] A signed copy of the consent form is held by the corresponding author and will be furnished to the editorial office on request.

Author Contributions

PPP, TVB and BSP collected and verified the clinical, laboratory and imaging data, reviewed the literature and prepared the first draft. RSH conceived the report, supervised data verification and critically revised the manuscript for intellectual content. All authors approved the final version and agree to be accountable for all aspects of the work.

Conflict of Interest

The authors declare that they have no conflict of interest.

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Table 1. Principal laboratory, cerebrospinal fluid and imaging findings

Investigation	Result	Comment
Haemoglobin, red cell indices	11.2–11.8 g/dL; MCV 85.4 fL, MCH 29.3 pg, MCHC 34.3 g/dL	Mild normocytic normochromic anaemia; blood group B positive
Total leucocyte count (serial)	20,900 → 16,200 → 14,000 → 13,500 /cumm	Neutrophils 90% → 74%; progressive fall
Platelet count	2.26–3.13 lakh/cumm	Normal throughout
ESR / C-reactive protein (serial)	70 → 30 → 46 mm/h / 3.20 → 6.74 → 0.72 mg/L	ESR raised; CRP marginally raised on day 5 only (reference 0–6 mg/L)
Blood urea / serum creatinine	16.6–25.1 / 0.5–0.7 mg/dL	Normal throughout admission
Serum sodium / potassium / calcium	133–137 / 3.3–3.9 mEq/L / 8.9 mg/dL	Mild hyponatraemia; transient hypokalaemia corrected with oral potassium chloride
Serum complement C3 / ASO titre	133.78 mg/dL / 375.2 IU/mL	C3 normal; ASO raised (reference <200 IU/mL)
Serum vitamin B12	179.5 pg/mL	Low; oral mecobalamin started
Urine routine (×2), blood culture	Normal; no growth	No pyuria; trace to negative albumin
CSF cell count	50 cells/cumm (lymphocytes 85%, neutrophils 15%)	Smear: predominantly lymphocytes, few neutrophils and red cells
CSF protein / glucose / chloride	44.8 / 42.8 mg/dL / 119 mEq/L	Mildly raised protein
CSF adenosine deaminase	10.2 U/L	Equivocal band (10–12.5 U/L); not diagnostic of tuberculous meningitis
CSF Gram and ZN stains, culture, CBNAAT	No organisms or acid-fast bacilli; no growth; M. tuberculosis not detected	Referring hospital CSF culture also sterile at 48 h
Serum anti-AQP4 IgG / anti-MOG IgG	Negative / strongly positive	Requested day 3, laboratory collection day 5, reported day 7; qualitative report, assay method and titre not stated
CSF anti-AQP4 and anti-MOG IgG	Both negative	CSF testing less sensitive than serum
Ultrasonography, abdomen and pelvis	Pre-void 310 mL, post-void residual 260 mL; bilateral mild hydroureteronephrosis; kidneys 8.7 × 4.2 cm and 8.4 × 4.0 cm	Attributed to bladder over-distension; normal cortical thickness and parenchyma
MRI brain and whole spine (day 2)	T2 hyperintensity with cord oedema C3–T1; subtle T2 hyperintensity over both caudate nuclei; prominent cisterna magna	Longitudinally extensive transverse myelitis

Investigation	Result	Comment
MRI brain with contrast (referring hospital, 12 days earlier)	Left retrocerebellar arachnoid cyst 22 × 17 × 13 mm; suspicious bilateral leptomeningeal enhancement; no diffusion restriction	Spine not imaged; deep nuclei normal at that time
Fundoscopy (serial)	Day 1 normal → day 5 bilateral disc oedema (R>L) → day 17 resolving papilloedema	Visual acuity improved from 6/9 to 6/6

ASO, anti-streptolysin O; CSF, cerebrospinal fluid; ESR, erythrocyte sedimentation rate; ZN, Ziehl-Neelsen; CBNAAT, cartridge-based nucleic acid amplification test; AQP4, aquaporin-4; MOG, myelin oligodendrocyte glycoprotein; MRI, magnetic resonance imaging.

Table 2. Chronology of clinical events and treatment

Hospital day	Event	Treatment
Day 1	Admission with lower abdominal pain and acute retention; tense bladder, extensor plantars, absent abdominal reflexes; ultrasonography shows post-void residual 260 mL with bilateral hydroureteronephrosis; fundoscopy normal	Urethral catheterisation; intravenous ceftriaxone 100 mg/kg/day; ranitidine; paracetamol as required
Day 2	MRI brain and spine: T2 hyperintensity with oedema C3–T1 and subtle bilateral caudate hyperintensity; working diagnosis of NMOSD	Plan for lumbar puncture, paired NMO-MOG panel and corticosteroid
Day 3	Lumbar puncture at L4–L5; CSF lymphocytic pleocytosis; paired serum and CSF NMO-MOG panel requested	Neurotropic B-complex infusion added
Day 4	Neurology review confirms transverse myelitis	Intravenous methylprednisolone 700 mg (30 mg/kg) once daily commenced
Day 5	Persistent fever spikes; ophthalmology review shows bilateral disc oedema (right > left) and advises contrast MRI of brain and orbit; samples for NMO-MOG panel collected by reference laboratory	Intravenous vancomycin 20 mg/kg/dose six-hourly added, with amikacin held in reserve; oral potassium chloride; weekly cholecalciferol 60,000 IU
Day 6	Catheter clamped and removed after three doses of methylprednisolone; retention recurs	Bladder redrained
Day 7	Reference laboratory: serum AQP4-IgG negative, serum MOG-IgG strongly positive, CSF antibodies negative; diagnosis revised from NMOSD to MOGAD	Tamsulosin 0.2 mg once daily started; fifth and final methylprednisolone dose given on day 8
Day 8	Blood pressure 122/80 mmHg with headache and dizziness; transferred to paediatric intensive care unit	Tamsulosin withheld; 3% saline at 0.5 mL/kg/hour; blood pressure and sensorium monitored
Day 9	Returned to ward, stable and headache-free; retention persists	Recatheterised; oral prednisolone 20 mg twice daily (2 mg/kg/day) started; intravenous immunoglobulin planned pending scheme approval
Days 10–12	Clinically stable; blood pressure normalises; ceftriaxone course completed on day 10	3% saline tapered and stopped on day 12; catheter clamping with graded trial of

Hospital day	Event	Treatment
		micturition begun
Day 14	Catheter removed; spontaneous voiding established at three- to four-hourly intervals	Oral mecobalamin and oral glycerol added; vancomycin course completed
Day 17	Ophthalmology review: resolving papilloedema both eyes; visual acuity 6/6	Supportive treatment continued
Day 18	Discharged — conscious, afebrile, normotensive, voiding spontaneously 5–6 times daily (urine output 4.5 mL/kg/hour)	Prednisolone taper 40 mg/day × 4 days → 30 mg/day × 7 → 20 mg/day × 7 → 10 mg/day × 7; weekly cholecalciferol × 6; lansoprazole; mecobalamin; advised review with repeat MRI and repeat MOG-IgG

NMOSD, neuromyelitis optica spectrum disorder; MOGAD, myelin oligodendrocyte glycoprotein antibody-associated disease; other abbreviations as in Table 1.

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