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CASE REPORT

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An atypical case of overlapped anti-NMDA-R and anti-GFAP encephalitis secondary to a teratoma of the fallopian tube: a case report

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Abstract

Background Anti-N-methyl-D-aspartate receptor (anti-NMDA-R) and glial fibrillary acidic protein (anti-GFAP) encephalitis are autoimmune conditions that can occur concurrently, often presenting with severe neurological symptoms. Coexistence of both antibodies has been associated with greater resistance to immunotherapy and a higher risk of underlying neoplasms, particularly teratomas, which are commonly linked to anti-NMDA-R encephalitis. Detecting such tumors is crucial, though standard imaging may fail to identify them, especially in atypical locations.

Case presentation We report the case of a 32-year-old woman presenting with severe overlapping of anti-NMDA-R and anti-GFAP encephalitis. She required long-term admission to the intensive care unit and showed no improvement after receiving both first- and second-line immunotherapies. Despite unremarkable imaging studies, the lack of therapeutic response prompted further investigation for a potential hidden neoplasm. An exploratory laparoscopy was performed and revealed a fallopian tube teratoma. Following surgical removal of the tumor, the patient experienced marked clinical improvement and returned to normal daily activities within six months.

Conclusions This case underscores the importance of investigating occult neoplasms in patients with overlapping anti-NMDA-R and anti-GFAP encephalitis who do not respond to immunotherapy. The coexistence of these antibodies increases the likelihood of an underlying teratoma, which may not be detectable through conventional imaging, especially when located in atypical sites such as the fallopian tube. In such cases, additional imaging studies and early consideration of invasive diagnostic procedures, including laparoscopy, is warranted. Prompt tumor removal can significantly improve clinical outcomes, even when initial imaging is normal.

Keywords Anti-NMDA-R encephalitis, Paraneoplastic, Occult neoplasm, Case report

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Background

Anti-NMDA-R encephalitis is the most common and well-characterized autoimmune encephalitis. It predominantly affects young women, accounting for approximately 80% of cases [1, 2], among whom the frequency of an underlying tumor has been reported to exceed 50%, most commonly ovarian teratomas [3]. The coexistence of anti-NMDA-R antibodies and anti-GFAP antibodies further increase the likelihood of an underlying teratoma [4, 5], underscoring the importance of actively investigating for these tumors.

The treatment of this encephalitis is based on immunotherapy and tumor removal when a neoplasm present. First line treatment includes high-dose intravenous steroids, immunoglobulins, and/or plasma exchange. Around 50–60% of patients with anti-NMDA-R encephalitis require a second line therapy, typically with rituximab or cyclophosphamide, which usually leads to good responses. In refractory cases, other agents, such as bortezonib or tocilizumab have been used in clinical practice. Notably, the presence of concomitant anti-GFAP antibodies has been associated with greater resistance to immunotherapy, making these cases more challenging to treat. Overall, anti-NMDA-R encephalitis generally responds well to immunotherapy combined with tumor resection, with favorable clinical outcomes in about 80% of patients two years after onset [1].

We present a case of overlapping anti-NMDA-R and anti-GFAP encephalitis refractory to immunotherapy due to an occult neoplasm, emphasizing the importance of searching for underlying tumors in treatment-resistant cases.

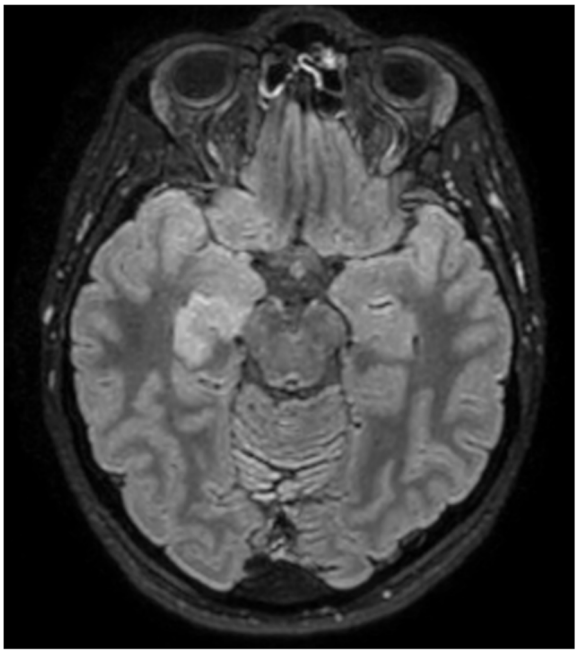

Fig. 1 Brain MRi, Axial-FLAIR. Legend: FLAIR signal enhancement in the right middle temporal lobe

Case presentation

A 32-year-old woman with a history of hypercholesterolemia, active smoking and occasional cannabis use, was admitted in the Emergency Department due to a 24-h history of psychomotor agitation, auditory hallucinations and anterograde memory impairment. Three days prior, she had experienced a generalized tonic–clonic seizure with no clear trigger. She also reported generalized asthenia, anorexia and nausea over the preceding days, without fever or other systemic symptoms. On neurological examination, the patient showed inattention, disorientation, and exhibited anterograde memory deficits. Initial brain CT was normal. Video-electroelectroencephalography showed a normal background activity with bursts of irregular slow waves, accompanied by occasional epileptiform discharges localized in the anterior and lateral region of the right temporal lobe. Antiepileptic therapy with levetiracetam was started. Notably, six months earlier, the patient had undergone adnexectomy for torsion of a mature ovarian teratoma. From the initial evaluation, limbic encephalitis, likely mediated by antibodies against N-methyl-D-aspartate receptor (NMDA-R), was suspected.

She was admitted to the neurology ward for further investigation. Brain MRI revealed increased T2 and FLAIR signal in the head and body of the right hippocampus, without restricted diffusion (Fig. 1). Lumbar puncture demonstrated cerebrospinal fluid (CSF) with 10 cells/mm3, predominantly mononuclear, normal glycorrhachia and protein concentration, and negative Gram stain. These findings, together with the clinical picture and history, supported the suspicion of autoimmune limbic encephalitis. Corticosteroid therapy was started 24 h after admission, consisting of intravenous methylprednisolone pulses of 1 gr/d during five days, followed by oral prednisone taper at 1 mg/Kg/d.

During the first hours of admission, the patient's agitation increased, showing no response to benzodiazepines or neuroleptics. On day 4 of steroid therapy, she required transfer to the intensive care unit (ICU) for deep sedation and mechanical ventilation to control agitation. Due to clinical deterioration, intravenous immunoglobulin (IVIg) therapy (0,4 g/Kg/d for five days) was initiated, along with an additional five pulses of 1 gr intravenous methylprednisolone. Despite these interventions, her condition remained unchanged, with fluctuating consciousness, intense agitation, continuous orofacial dyskinesias and signs suggestive of dysautonomic crisis. At this stage, immunological testing of serum and CSF samples performed at a reference center following previously described methodology [6], confirmed the presence of

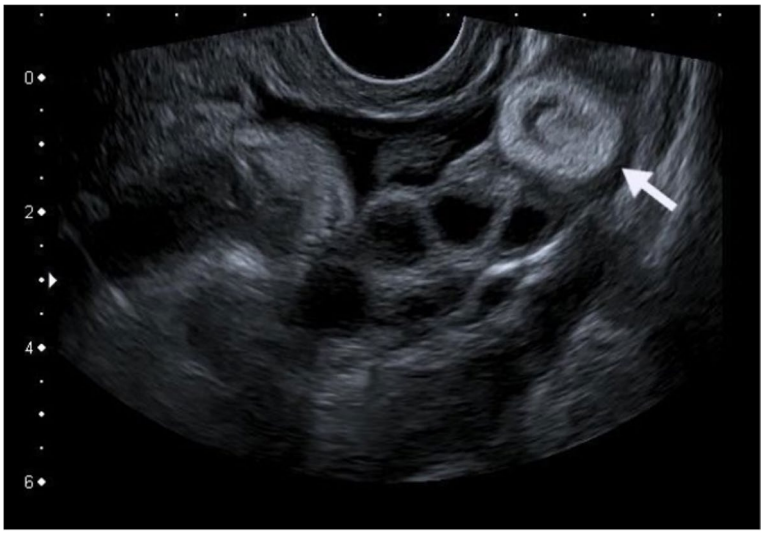

Fig. 2 Transvaginal Ultrasound Identifying Suspected Tubal Lesion. Legend: Cross sectional transvaginal ultrasound image that shows a normal ovary of a young woman with correct follicular reserve and an oval hyperechogenic image in the ampullary area of the fallopian tube (arrow)

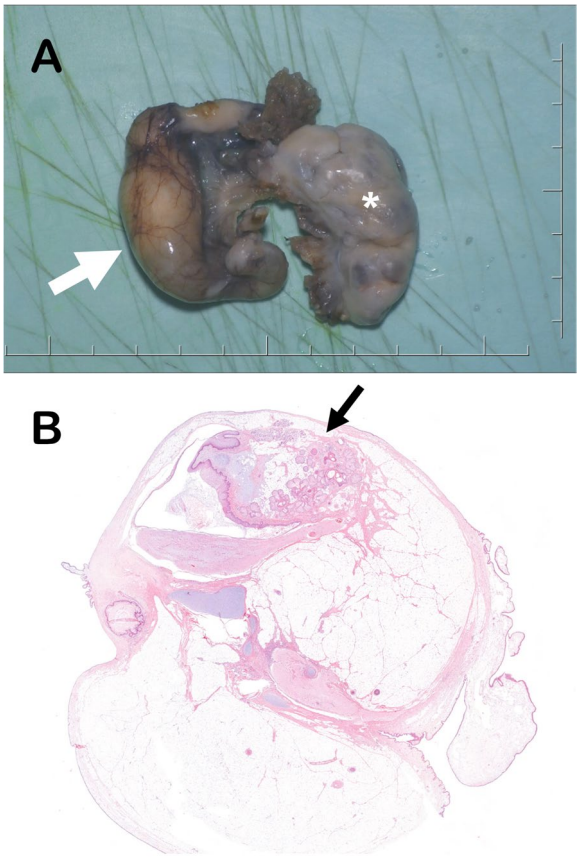

Fig. 3 Macro- and micro- anatomopathological sample of the fallopian tube and ovary. Legend: **A** Surgical specimen of the teratoma included in the fallopian tube (arrow), where it can be visualized expanded tube and the normal ovary (*) which was removed. **B** Benign intraluminal proliferation with keratin-containing and ectodermal-derived epithelial tissue (arrow) constituting the teratoma

anti-NMDA-R antibodies in both CSF and serum. Coexisting anti-GFAP antibodies were also detected in CSF, while onconeuronal antibodies were negative. Whole-body CT scan ruled out recurrence of teratoma or other neoplasms, and 18-FDG PET-CT showed no uptake suggestive of malignancy.

A repeat brain MRI performed 20 days after admission was normal, but the patient's clinical symptoms showed no improvement. Intravenous rituximab was initiated at 375 mg/m2 weekly for four weeks, and after the first week, a new 5-day course of IVIg was administered. Despite four doses of rituximab and the additional IVIg course, there was no clinical improvement. Given the lack of response, plasmapheresis was started one week after the last rituximab dose. Clinical improvement became evident after the third session of plasmapheresis. After 67 days in the ICU, the patient was transferred back to the neurology ward. However, this improvement was not sustained, and eight days later, she experienced clinical deterioration, requiring high dose sedative therapy and readmission to the ICU. Clinical impairment could not be explained by any other cause, suggesting it was due to the underlying autoimmune encephalitis. Despite previously normal body imaging, the recurrence of symptoms and resistance to treatment prompted further evaluation for an occult neoplasm. Pelvic ultrasound revealed a small lesion in the contralateral adnexa (Fig. 2), which was difficult to characterize due to its size. Exploratory laparoscopy identified a small tumor in the fallopian tube. A left adnexectomy, including the ipsilateral fallopian tube, was performed (Fig. 3A), and histopathology confirmed a mature tubal teratoma (Fig. 3B).

Following surgery, the patient demonstrated progressive clinical improvement. Neuropsychological

assessment three weeks postoperatively revealed deficits in verbal and visual memory, learning, and delayed recall. Impairments were also observed in naming by visual confrontation, mental calculation, and executive functions, including attention, working memory, and phonemic fluency. Improvement continued over the subsequent days, and she was discharged home after 118 days of hospitalization (Fig. 4 shows a timeline of disease course and treatments). At one month follow-up, the patient and her relatives reported good recovery, she was performing daily life activities at home (modified Rankin Scale [mRS] 1/6, compared to 5/6 during the acute phase). Six months later, follow-up neuropsychological testing showed only mild residual deficits in naming and executive function (for detailed scores, see Table S1 in Additional File 1).

Discussion and conclusions

Our case highlights the importance of searching for occult neoplasm in patients with coexisting anti-NMDA-R and anti-GFAP encephalitis who fail to respond to immunotherapy or relapse after a period of improvement. Diagnostic difficulty in this patient was compounded by infrequency of tubal teratomas, which are far less common than ovarian teratomas and often challenging to detect on imaging. To date, only two cases of immature tubal teratoma associated with anti-NMDA-R encephalitis have been reported in the literature [7, 8], and, to our knowledge, there are no previous reports of overlapping anti-GFAP and anti-NMDA associated with teratomas at this location.

Previous case reports and series have described the coexistence of anti-NMDA-R and anti-GFAP antibodies (See Table 1). Consistent with the clinical presentation in our patient, typical of anti-NMDA-R encephalitis and supported by characteristic MRI findings, many previous case reports and a large series of anti-NMDA-R encephalitis patients have shown that in cases of overlap between these two antibodies, the clinical presentation and imaging findings are often predominantly driven by anti-NMDA-R pathology [6, 9, 10], although, in some cases, the typical perivascular radial enhancement of GFAP astrocytopathy can also be observed [5, 11]. Some studies suggest that GFAP autoimmunity may represent a secondary phenomenon triggered by anti-NMDA-R antibodies, which may induce an immune response and secondary astrocytic injury [11, 12]. This mechanism could explain why the clinical picture is primarily dominated by anti-NMDA-R autoimmunity.

The coexistence of anti-NMDA-R and GFAP antibodies has been associated with both an increased risk of teratoma and a higher likelihood of treatment resistance in overlapping encephalitis cases compared to either antibody alone [5, 16]. When a tumor is present, surgical removal has been associated with better clinical outcomes. There are few published cases of patients with treatment-resistant anti-NMDA-R encephalitis who improved after empiric ovariotomy, despite no suspicious lesions being detected on initial CT or pelvic ultrasound; subsequent histopathology confirmed the presence of an ovarian teratoma [17–20]. The interval between symptom onset and surgery in these cases ranges from 1.5 to 11 months, with clinical improvement becoming evident within days after surgery. However, when surgery was delayed, long-term sequelae appeared to be more pronounced [17, 21]. In our patient, early surgery, within the first three months, likely contributed to her significant long-term cognitive recovery.

In conclusion, the coexistence of anti-GFAP and anti-NMDA-R encephalitis, especially in young women, should raise suspicion of underlying teratoma. In the

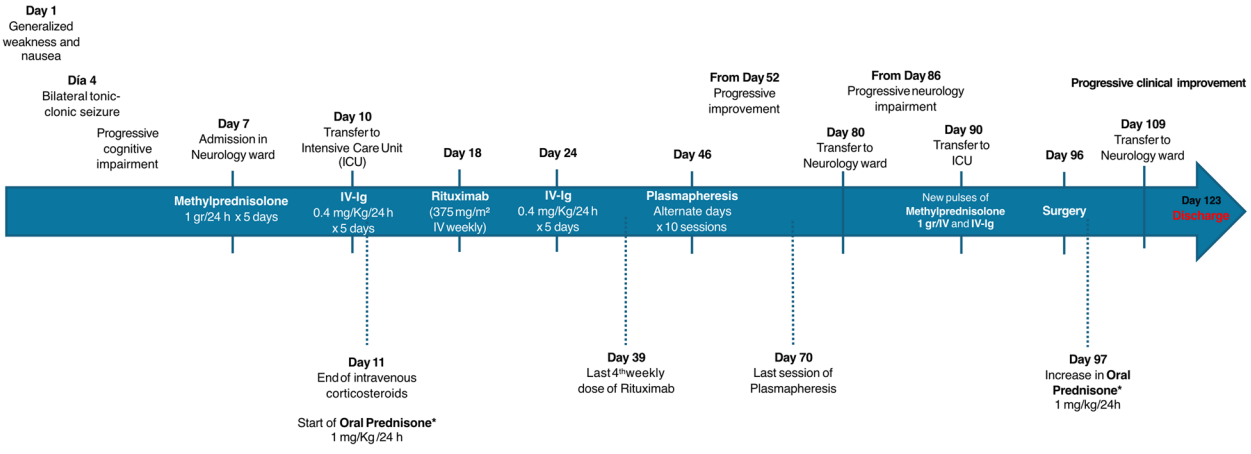

Fig. 4 Timeline illustrating disease progression and treatments administered from admission to discharge. Legend: *Oral prednisone was first introduced on Day 11, following the completion of intravenous corticosteroid pulses, at an initial dose of 1 mg/kg/day. The dose was gradually tapered, reaching 20 mg/day by Day 80. After surgery, the dose was increased again to 1 mg/kg/day and then progressively tapered once more, with the treatment concluding three months after discharge

Table 1 Comparison of our case with previously reported cases of autoimmune encephalitis with overlapping anti-NMDA-R and anti-GFAP antibodies

Publication	Age at onset	Sex	Main clinical features	EEG	Brain MRI findings	Ab detection NMDA-R Ab/ GFAP Ab	Associated tumor	Immunotherapy | Surgery	Follow up, mRS
Present case*	32	F	Generalized asthenia, progressive cognitive decline and behavioral changes, psychiatric symptoms, seizures, consciousness fluctuation, dysautonomic crisis, orofacial dyskinesias	Epileptiform discharges in right temporal lobe	FLAIR hyperintensity in right mesial temporal lobe	Serum ± CSF +/+	Tubal teratoma	Second line (Rituximab) Salpingo oophoforectomy	1 (1 mo after discharge)
E. Martínez-Hernández et al. Neurology, 2020 [6]** (10 cases)	24 (13–60)	9/10 F	All 10 pts with anti-NMDA-R "typical symptoms" 2 pts: brainstem-cerebellar symptoms 1 pt: meningeal signs	-	1/10 had meningeal gadolinium enhancement	6 pts: GFAP only in CSF 1 pt concomitant GlyR-Ab	3/10: ovarian teratoma	6 pts: first line 2 pts: second line 2 pts: teratoma removal	5 pts: 0 (0–1) (45 [16–53] mo)
X. Tu et al. Frontiers in Immunology, 2025 [13]	24	F	Fever, headache, cognitive impairment and psychiatric symptoms Seizures during follow up	Diffuse generalized slowing and dysrhythmia Temporal epileptiform discharges (during follow-up)	1st: Diffuse leptomeningeal thickening and enhancement 10 d. after: diffuse linear enhancement along cerebral sulci and cortical surfaces	Serum +/+ CSF +/+	Bilateral ovarian teratomas	Right salpingooforectomy and left ovarian cystectomy Second line (Ofatumumab, Efgartigimod) after the surgery	1 (1 mo after discharge)
R Cui et al. Medicine, 2024 [14]	50	M	Generalized weakness, fever, consciousness impairment, seizures	NA	Normal	Serum NA/NA CSF +/+ NMDA-R Ab = 1:10 GFAP Ab = 1:32 Anti-sulfatide Ab in serum	No tumor Abdominal aortic aneurysm with mural Thrombosis at admission	First line	6† (18 days after admission)
P. Bai et al. BMC Neurology, 2022 [11]	35	M	Fever, headache, meningeal signs, cognitive impairment, psychiatric symptoms, sleep disturbances, urinary incontinence	Diffuse generalized slowing	Paraventricular, corona radiata, semioval center and right subcortex FLAIR hyperintensities, with patchy and linear perivascular radial gadolinium enhancement	Serum -/- CSF +/+ NMDA-R Ab = 1:32 GFAP Ab = 1:32	No tumor	First line Second line as maintenance (Mycophenolate mofetil)	0 (1.5 mo after disease start)
AL. Martin et al. J Pediatr Adolesc Gynecol, 2018 [10]	13	F	Fever, headache, psychiatric symptoms, pyramidal sings Brainstem and cerebellar signs	Diffuse generalized slowing	Normal	Serum –/- CSF +/+	Ovarian teratoma	First line Ovarian cystectomy	1 (2 mo after discharge)

Table 1 (continued)

Publication	Age at onset	Sex	Main clinical features	EEG	Brain MRI findings	Ab detection NMDA-R Ab/ GFAP Ab	Associated tumor	Immunotherapy | Surgery	Follow up, mRS
EP. Flanagan et al. Ann Neurol., 2017 [5] (22 cases)	-	-	19 pts: encephalitis 1 pt: meningo-encephalitis 1 pt. meningo-encephalomy-elitis 1 pt: ataxia	NA	4 available MRI: 3 pts: radial gadolinium enhancement 1 pt: leptomeningeal serpentine gadolinium enhancement	Serum NA/NA CSF +/+ 7 pts with AQP4 Ab coexistence	13 pts: ovarian teratoma (5/13 with coexistent AQP4-Ab)	Data available for 3 pts: 2 pt long term second line (Mycophenolate mofetil, Azathioprine)	Data available for 3 pts: 1 pt relapse 2 mo after symptoms onset
E. Zengin et al. BMJ Case Rep 2023 [9]	30 (16 weeks' gestation)	F	Headache, sleep disturbances, psychiatric symptoms, seizures (faciobrachial), consciousness impairment, dysautonomia	Diffuse generalized slowing (initially) Delta brush pattern (during progression)	Normal	Serum NA/NA CSF +/+	Ovarian teratoma	Second line (Rituximab) Bilateral oophorectomy + Cesarean Sect. (34 w)	1 (12 mo after discharge)
FA. Ayala et al. Neurology: Clinical Practice 2021 [15]	70	M	Fever, consciousness impairment, dysautonomia	Diffuse generalized slowing	No encephalitis related findings	Serum NA/NA CSF +/+	Known nonsmall- cell lung carcinoma, in treatment with Nivolumab	First line Discontinuation of Nivolumab	0 (follow up visit, time not specified)

This table summarizes the main clinical characteristics, test findings, treatments, and outcomes of our case and previously reported cases and series of overlapping anti-NMDA-R and anti-GFAP encephalitis. References for the publications included in this table are provided in the main manuscript. In the "Antibody Detection" column, we report, when available, the positivity in CSF and serum for the two antibodies (anti-NMDA-R/anti-GFAP), including titer information if provided. In the column related to the treatment, for immunotherapy, we indicate the maximal line of treatment required for each patient

Abbreviations: *Ab* Antibody, *AQP-4* Aquaporin-4, *EEG* Electroencephalography, *F* Female, *GlyR-Ab* antibodies against the Glycine Receptor, *M* Male, *mo* Month, *MRI* Magnetic Resonance Imaging, *Mrs* modified Rankin Scale, *NA* Not Available (or not specifid in the paper); pt(s) = patient(s); w = week

[*] Present case refers to the patient described in this report

[**] E. Martínez et al., Neurology, 2020 [6]. Information regarding the case reports in this review is based on the data presented in their manuscript and associated table

absence of response to immunotherapy, additional imaging studies are recommended, and, even when imaging is unremarkable, exploratory abdominopelvic surgery should be considered to search for occult teratomas, including those in atypical locations such as the fallopian tubes. Oophorectomy may also be warranted in cases with equivocal findings, given the high probability of ovarian, or even tubal, teratomas.

Abbreviations

CSF	Cerebrospinal Fluid
CT	Computed Tomography
FLAIR	Fluid-Attenuated Inversion Recovery
GFAP	Glial Fibrillary Acidic Protein
ICU	Intensive Care Unit
IVIg	Intravenous Immunoglobulin
MRI	Magnetic Resonance Imaging
NMDA-R	N-methyl-D-aspartate receptor
PET-CT	Positron Emission Tomography–Computed Tomography
18-FDG	: Fludeoxyglucose F18

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12883-025-04488-8.

Supplementary Material 1.

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Authors' contributions

- M.R.S. and C.C.G.L. contributed equally to this article and share first authorship. - M.R.S. was one of the main neurologist caring for the patient during her admission and played a primary role in writing the manuscript alongside C.C.G.L. - C.C.G.L. was one of the lead surgeons and also played a primary role in writing the manuscript with M.R.S. - J.M.A., M.F.P., C.F.R., V.G.Q., and F.O.V. were part of the neurology team responsible for the patient's care during admission. - S.V.C., A.C.M., R.M.R.M., and A.D.C. were members of the gynecology team responsible for the gynecological tests, their interpretation, the surgery, and the follow-up. - T.O.G. was the main intensivist responsible for the care of the patient during her stay in the intensive care unit. - E.M.H. was in charge of antibody analysis and assisted with the interpretation of the results. - S.H.R. was the lead pathologist who conducted the anatomic pathology

analysis of the fallopian tube tumor. - J.F.T. was the main neurophysiologist responsible for performing and interpreting the electroencephalograms. - E.R.P. was the main radiologist responsible for interpreting the MRI scans. - E.R.R. was one of the main neurologists responsible for the patient's care during admission and follow-up after discharge. He was also the primary supervisor of this manuscript's preparation. - All authors reviewed the manuscript and approved its publication.

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Data availability
Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Declarations

Ethics approval and consent to participate
This case report was conducted in accordance with ethical standards and followed the CARE (CAse REport) guidelines for case reports.
Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.
We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this work is consistent with those guidelines.

Consent for publication
Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

Competing interests
The authors declare no competing interests.

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