

SYNTHETIC CASE — NOT A REAL PATIENT RECORD

Anti-NMDA-Receptor Encephalitis

Treating Under Diagnostic Uncertainty

A Multidisciplinary Case Study — Psychiatry → Medicine → Neurology
Ms. A.V., 52-year-old woman

DR. DIVI CLINICAL LABS — Clinical Reasoning Case Series

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No identifying patient information is included. “Ms. A.V.” and all clinical detail in this document are fictional, constructed for demonstration purposes only.

This case was designed and directed by the author as a clinical-reasoning exercise. Drafting and cross-document synthesis were AI-assisted; all clinical content, diagnostic reasoning, and figures were reviewed and are owned by the author.

About This Exercise

This case models a common and difficult position in real practice: a multidisciplinary team converging on the same working diagnosis — anti-NMDA-receptor autoimmune encephalitis — while the one test that would confirm it is still pending. The confirmatory CSF/serum antibody panel is never resolved in this case by design. The teaching point is not “what was the diagnosis,” but how three services (psychiatry, medicine, neurology) each independently reasoned from an incomplete, partly conflicting record to the same empiric decision to treat, and how that reasoning holds up against the raw source data.

A second discipline runs alongside the clinical reasoning: exhibit-level verification. The laboratory and imaging figures quoted below are reconciled directly against the three original outside-hospital exhibits (a CSF/serum immunology report, a serum β -hCG result, and a pelvic ultrasound still) rather than against any single note's transcription of them. Earlier drafts of this case's own documentation disagreed with each other and, in places, with the source images — most often conflating CSF glucose with CSF lactate, and misstating the CSF IgG index and its reference range. Those errors are corrected in what follows and flagged where relevant, in the same spirit as the chart-reconciliation work the case itself describes.

1. Background & Objective Course

Patient: Ms. A.V., 52-year-old woman.

Past medical history: hypertension (amlodipine), diet-controlled type 2 diabetes, hypothyroidism (levothyroxine), chronic kidney disease (baseline creatinine $\sim$ 1.5 mg/dL), iron-deficiency anemia, osteoarthritis, seasonal allergies.

Allergies: structured chart field lists NKDA. Separately, a roommate reported a remote, undated “bad reaction, like hives” to an unspecified IV medication; no agent, timing, or record was ever produced, and this has not been formally reconciled with the chart allergy field.

Family history: mother with approximately one year of intermittent joint aches and vague pelvic discomfort, never formally worked up; maternal aunt with lupus.

Timeline

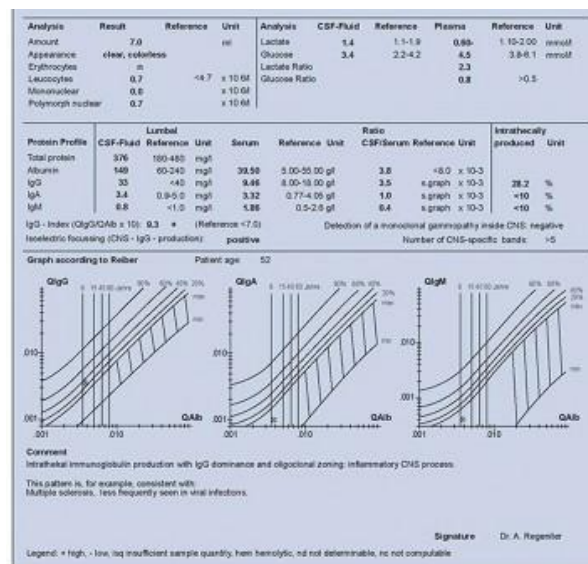
- **~3 weeks before current admission** — ED visit for fever and headache; discharged home. Three outside-ED exhibits from this visit (CSF/serum immunology report, serum β -hCG, pelvic ultrasound) were scanned into the chart but not reviewed by the treating team until much later.
- **~1 week after the fever** — new paranoia, insomnia, withdrawal, and echolalia, with orofacial lip-smacking/chewing initially read as anxiety or early extrapyramidal effect. Admitted to inpatient psychiatry; risperidone 1 mg PO BID started.
- **Hospital day 9** — routine skin check finds mild pruritus and a faint rash near an old IV site, without current infection. A roommate independently reports the remote, unverified “hives” reaction described above.
- **Hospital day 10** — roommate interview reveals diphenhydramine 100–150 mg nightly for roughly two weeks before the fever, self-treated for insomnia — two to three times the labeled OTC dose, and never present in the original psychiatry admission note.

- **Hospital day 17** — acute overnight decompensation: sinus tachycardia 130s–140s, blood pressure swinging $\approx 160/105$ to $92/58$ within the same shift, temperature to 38.9°C , markedly worsened orofacial movements, and new choreoathetoid arm movements. Transferred to medicine/neurology. Repeat LP: 24 WBC/ μL , lymphocyte-predominant, glucose 62 mg/dL, mildly elevated protein; head CT unremarkable. Empiric IV acyclovir started while infectious PCR's were pending, dosed at 10 mg/kg every 8 hours on an admission weight of 131 kg (≈ 1.31 g per dose, ≈ 3.93 g/day) — see Attention Points, below, on reconciling this against renal function; a CSF autoimmune-encephalitis antibody panel was sent (laboratory-quoted turnaround 2–3 weeks).
- **Hospital day 18** — neurology starts empiric high-dose IV methylprednisolone without waiting for the antibody result.
- **Hospital day 24 (present)** — vitals stable, afebrile for 48 hours; autonomic instability has settled, but the movement disorder and intermittent encephalopathy persist. The antibody panel remains pending.

2. Outside-ED Exhibits — Reconciled Against Source Images

The three images below were obtained at the original fever/headache ED visit, roughly three weeks before the current admission, and were not reviewed by the treating team until the medicine consult. Values are quoted here exactly as they appear on the source images.

Exhibit A — CSF/Serum Quotient (Reiber-type) Report



Source image — CSF/serum protein profile and Reiber quotient graph

- Sample: clear/colorless CSF, essentially acellular — erythrocytes 0; leukocytes $0.7 \times 10^6/\text{L}$ (0.0 mononuclear, 0.7 polymorphonuclear; reference $<4.7 \times 10^6/\text{L}$).
- CSF glucose 3.4 mmol/L; plasma glucose 4.5 mmol/L; CSF/plasma glucose ratio 0.8.
- CSF lactate 1.4 mmol/L (reference 1.1–1.9 mmol/L).
- CSF total protein 376 mg/L; albumin 149 mg/L.
- CSF IgG 33 mg/L, IgA 3.4 mg/L, IgM 0.8 mg/L; paired serum IgG 9.46 g/L, IgA 3.12 g/L, IgM 1.08 g/L.

- **CSF IgG Index = 9.3, reference <7.0.** Several drafts of this case mis-transcribed both the value and the reference range as 0.9–0.93 / <0.7 — 9.3/<7.0 is the value printed on the source image and is used throughout this document.
- Intrathecal IgG production 28.2%; intrathecal IgA and IgM each <10%.
- Isoelectric focusing positive, >5 CSF-specific oligoclonal bands; CSF monoclonal gammopathy: negative.

Laboratory comment: *“Intrathecal immunoglobulin production with IgG dominance and oligoclonal zoning: inflammatory CNS process,” with multiple sclerosis given as an example pattern and viral infection noted as a less frequent association. The report's own printed patient-age field reads 52, consistent with this patient.*

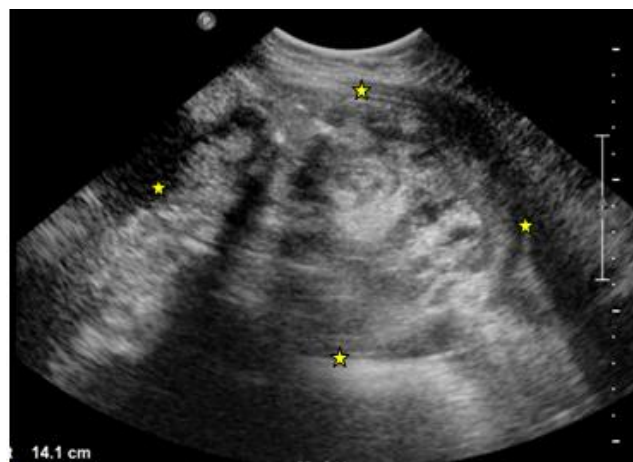
Exhibit B — Serum β-hCG

<u>BETA HCG SERUM</u>	
<u>TEST NAME</u>	<u>RESULT</u>
Serum HCG (Human Chorionic Gonadotropin) : Method: Electro Chemi Luminescence Immuno Assay (ECLIA)	2

Source image — serum β-hCG result

- Serum human chorionic gonadotropin (hCG) = 2, by electrochemiluminescence immunoassay (ECLIA).
- No reference interval is shown on the source image; the value is reported exactly as printed. In this postmenopausal-age patient, it does not support pregnancy and is not evidence against a pelvic mass being present.

Exhibit C — Pelvic Ultrasound



Source image — transabdominal pelvic ultrasound still, 14.1 cm scale marker visible

- Large, markedly heterogeneous pelvic/adnexal mass with mixed solid/cystic echotexture and posterior acoustic shadowing; 14.1 cm scale marker visible on the image itself.
- Appearance is compatible with — not diagnostic of — a mature cystic (dermoid) teratoma. No formal radiology report accompanies the image; laterality, histology, and benignity are not established from this still alone.

Reconciliation note: earlier drafts of this case variously reported the CSF glucose as “1.4 mmol/L” or “34 mg/dL,” transposing it with the lactate value, and reported the IgG index as anywhere from 0.9 to 0.93 against a <0.7 reference. The figures in

Exhibits A–C above are taken directly from the source images and take precedence over any conflicting figure elsewhere in this case's documentation history.

3. Attention Points — Citation-Backed Flags

The points below are included only where their clinical significance is backed by a checkable, dated source — the same discipline the case's own background documentation applies to itself. They are listed in the order a reader should weight them, most safety-relevant first.

1. Acyclovir dose should be reconciled with current renal function

The documented regimen is **10 mg/kg IV every 8 hours**, calculated on an admission weight of **131 kg** (≈ 1.31 g per dose, ≈ 3.93 g/day). The FDA ZOVIRAX label identifies renal excretion as acyclovir's major elimination route, requires dose adjustment by estimated creatinine clearance, and separately lists confusion, hallucinations, agitation, and seizures among the drug's own encephalopathic adverse effects. That matters directly here: this patient has CKD (baseline creatinine ~ 1.5 mg/dL), and encephalopathy is already part of the presenting syndrome the team is trying to explain. This dose should be reconciled against current renal function rather than carried forward unreviewed.

2. Risperidone exposure is a relevant confounder to the hospital-day-17 episode

The FDA RISPERDAL prescribing information carries warnings for neuroleptic malignant syndrome and tardive dyskinesia/extrapyramidal symptoms. The orofacial movements reportedly predated risperidone, while the later febrile/autonomic episode and marked movement escalation occurred after exposure — the chronology is relevant to interpreting the syndrome but does not by itself establish causality.

3. Unreported diphenhydramine exposure is clinically relevant background

100–150 mg nightly is two to three times the 50 mg dose specified on current OTC nighttime-sleep-aid labeling (DailyMed). Published toxicology literature (Gilbert et al., *Pharmacotherapy* 2025) links diphenhydramine's anticholinergic effects to delirium, hallucinations, tachycardia, and other CNS/autonomic manifestations, particularly at excessive doses. This does not by itself establish that diphenhydramine caused the syndrome, but it is a previously unrecognized exposure that may affect interpretation of the encephalopathy and autonomic findings.

4. Diagnosis can proceed before antibody confirmation

The combination of subacute psychiatric change, altered mental status, abnormal movements, and autonomic instability, together with CSF pleocytosis on the day-17 LP, sits within the published clinical framework for recognizing probable autoimmune encephalitis before antibody results return. The 2016 Graus criteria explicitly allow diagnosis to proceed from syndrome plus supportive findings rather than waiting for antibody testing; the 2021 best-practice recommendations (Abboud et al.) likewise frame CSF studies, neuronal antibodies, EEG/MRI, and exclusion of infectious causes as the diagnostic workup itself, not a precondition for treating.

5. Pelvic imaging is relevant if an anti-NMDAR phenotype remains under consideration

The 2021 best-practice recommendations note that pelvic ultrasound functions as targeted tumor screening when the clinical picture suggests anti-NMDAR encephalitis. Because the supplied image has no

accompanying formal report, and the patient has a history of vague pelvic discomfort, the actual radiology report would be materially more informative than the still alone. This point does not imply that a teratoma is confirmed.

4. Known vs. Pending — Hospital Day 24

Documented to date

- Psychiatric syndrome preceded the marked neurologic/autonomic deterioration.
- Marked orofacial dyskinesia later joined by choreoathetoid arm movements and intermittent encephalopathy.
- Day-17 CSF: 24 WBC/ μ L, lymphocyte-predominant; glucose 62 mg/dL; mildly elevated protein. Day-17 head CT unremarkable.
- Fever and autonomic instability resolved over the days after transfer; the movement disorder and encephalopathy have not.
- Exposure/collateral gaps: risperidone began after the psychiatric symptoms; substantial pre-fever diphenhydramine exposure; an unverified remote IV-medication “hives” history.

Pending / not yet reconciled

- CSF autoimmune-encephalitis antibody panel sent hospital day 17 — laboratory-estimated turnaround 2–3 weeks.
- Formal pelvic ultrasound / gynecologic report — teratoma is not yet confirmed, only compatible with the image.
- Complete infectious CSF PCR results from the hospital-day-17 lumbar puncture.
- Allergy history remains internally discordant — NKDA in the structured field versus an unverified collateral “hives” history.

5. Overnight Medicine Consult

Overnight, hospital day 3 of the psychiatric admission — before the hospital-day-17 decompensation, at the point psychiatry first asked whether risperidone could safely be increased.

Reason for consult

Medical clearance and assessment of the safety of increasing the risperidone dose.

Records reviewed

- Psychiatry admission note and current inpatient course.
- Three outside-ED images, scanned into the chart from the earlier fever visit and not previously reviewed by this team.

Outside records reviewed

1. CSF/serum immunoglobulin and oligoclonal-band study (outside, earlier ED visit): clear/colorless CSF, essentially acellular (leukocytes $\sim 0.7 \times 10^6/L$; no significant RBCs); CSF glucose 3.4 mmol/L (paired plasma glucose 4.5 mmol/L, ratio 0.8); CSF lactate 1.4 mmol/L; CSF total protein 376 mg/L. IgG index elevated at 9.3 (reference <7.0), with positive isoelectric focusing, intrathecal IgG production of 28.2%, and >5 CSF-specific oligoclonal bands. Report comment describes intrathecal immunoglobulin production with IgG dominance and oligoclonal zoning — an inflammatory CNS pattern, not diagnostic of multiple sclerosis, and best read in the current clinical context.

2. Serum β -hCG: 2 (ECLIA). In this 52-year-old, postmenopausal-age patient, this is not suggestive of pregnancy.

3. Transabdominal pelvic ultrasound still: large, markedly heterogeneous pelvic/adnexal mass, mixed solid/cystic echotexture, posterior acoustic shadowing; 14.1 cm scale marker on the image. Compatible with, not diagnostic of, a mature cystic teratoma. No formal radiology report accompanies the image.

Assessment

- **1. New-onset psychosis with catatonia-spectrum features and abnormal involuntary orofacial movements, following a febrile prodrome** — concerning for an organic neuropsychiatric process rather than an uncomplicated primary psychotic disorder.
- **2. High concern for autoimmune encephalitis, particularly anti-NMDA-receptor encephalitis** — given the clinical phenotype plus outside CSF evidence of intrathecal IgG production and CSF-specific oligoclonal bands, together with a pelvic-mass appearance compatible with an ovarian teratoma. Not yet confirmed: no anti-NMDAR antibody result is available, and the pelvic finding still requires formal radiologic and gynecologic characterization.
- **3. Risperidone dose escalation — not recommended tonight.** Dopamine blockade can complicate assessment of movement abnormalities and, in anti-NMDAR encephalitis, may worsen dyskinesias or contribute to a neuroleptic-malignant-syndrome-like picture. Continue 1 mg PO BID only if clinically necessary; avoid escalation.

- **4. Medical clearance** — routine admission CBC/BMP/LFT/TSH/urine toxicology are reportedly unremarkable, but the outside findings materially change the clearance assessment. This should not be treated as an uncomplicated primary psychiatric admission until autoimmune and infectious neurologic causes have been evaluated.
- **5. Chronic kidney disease, baseline creatinine ~1.5 mg/dL** — no current electrolyte or hepatic abnormality reported; obtain and review current BMP and eGFR/CrCl, and avoid nephrotoxins.

Recommendations tonight

- Urgent neurology consultation; consider transfer to a medical/neurology service if symptoms progress or the needed workup cannot be safely completed on the psychiatric unit.
- Obtain EEG promptly given the brief altered-awareness spells and possible seizure activity.
- Obtain MRI brain with and without contrast if feasible.
- Send paired serum and CSF autoimmune-encephalitis testing, specifically anti-NMDA receptor (GluN1) IgG, with a broader autoimmune/paraneoplastic panel per neurology; CSF is the more sensitive specimen.
- Confirm the infectious-encephalitis workup (HSV/VZV PCR and other studies) with neurology/ID; oligoclonal bands alone do not establish autoimmune encephalitis.
- Obtain the formal outside pelvic ultrasound report and/or repeat formal pelvic imaging; consider gynecology consultation given the mass appearance.
- Obtain ECG/QTc before any further antipsychotic escalation; monitor for extrapyramidal symptoms, rigidity, fever, autonomic instability, and worsening abnormal movements.
- If agitation or catatonia becomes difficult to manage, favor a benzodiazepine-based strategy over escalating dopamine blockade, per psychiatry/neurology.
- Document the reported remote “bad reaction” to an IV medication clearly in the chart before any future immunotherapy, even though it has not yet been formally reconciled with the NKDA field.

Bottom line for psychiatry

The outside records materially change the medical assessment. This should be treated as possible autoimmune encephalitis — most notably anti-NMDA-receptor encephalitis, with a pelvic mass appearance compatible with an ovarian teratoma — until evaluated otherwise. Hold risperidone at 1 mg PO BID tonight rather than increase it, and arrange urgent neurologic evaluation.

6. Neurology / Medicine Co-Management — Overnight Recommendation

Hospital day 24, after the hospital-day-17 decompensation and transfer to medicine/neurology.

Clinical impression

The overall syndrome is highly concerning for probable anti-NMDA receptor autoimmune encephalitis (NMDARE), likely paraneoplastic in the setting of a large, complex ovarian/adnexal mass. The phenotype is unusually characteristic: a psychiatric prodrome with insomnia, paranoia, withdrawal, and echolalia, followed by encephalopathy, prominent orofacial dyskinesias and choreoathetoid limb movements, fever, tachycardia, and marked blood-pressure lability. The hospital-day-17 CSF shows lymphocytic pleocytosis with mildly elevated protein, and the outside CSF study independently shows an inflammatory intrathecal IgG pattern. The outside pelvic ultrasound image shows a large (~14.1 cm) complex adnexal/ovarian mass whose morphology may be compatible with a teratoma, but the formal radiology report and definitive gynecologic characterization are still needed. Serum β -hCG is 2 and does not support pregnancy.

The combination of syndrome, inflammatory CSF, and a probable ovarian teratoma makes NMDARE substantially more likely than primary psychiatric disease, isolated drug-induced dyskinesia, or diphenhydramine toxicity. Risperidone exposure can worsen movement disorders and confound the picture — including raising concern for neuroleptic malignant syndrome — but it does not adequately explain the preceding psychiatric/encephalitic course or the CSF inflammation.

Recommendation tonight

Do not hold therapy solely to await the pending autoimmune-encephalitis antibody panel. The clinical probability is high enough to treat empirically once CNS infection has been reasonably excluded.

- Proceed with additional first-line immunotherapy now: IVIG or plasma exchange. IVIG should be regarded as an addition to first-line therapy, not as “second-line” treatment; in severe NMDARE presentations, steroids plus IVIG/PLEX is a standard acute approach.
- Given baseline CKD (creatinine ~1.5) and diabetes, if IVIG is selected, use a non-sucrose-containing preparation and the lowest practical infusion rate/concentration, and monitor creatinine/eGFR, urine output, volume status, hemolysis, and thrombotic risk closely. If renal risk or volume constraints make IVIG unattractive, discuss plasma exchange promptly with apheresis.
- Do not escalate tonight directly to rituximab or cyclophosphamide solely because the movements have not improved after the steroid pulse; these are second-line agents. Complete or augment acute first-line therapy first and reassess objectively over the following one to two weeks.
- Urgently involve gynecology/gynecologic oncology for definitive evaluation and removal of the suspected adnexal tumor if confirmed. Tumor-directed treatment should not be deferred until the CSF antibody returns when the clinical/imaging picture is this compelling; coordinate operative timing with neurology, anesthesia, and medicine, since NMDARE can produce perioperative autonomic instability and hypoventilation.
- Obtain or review the formal pelvic ultrasound report; pursue pelvic MRI or other staging imaging only if needed for surgical planning and if it will not delay definitive evaluation.

- Hold risperidone for now and avoid additional dopamine-receptor antagonists if clinically feasible, given the severe dyskinesias. If behavioral control is required, favor a regimen agreed with psychiatry that minimizes dopamine blockade.
- Do not transfer back to inpatient psychiatry tonight. She remains intermittently encephalopathic, inconsistently follows commands, and has persistent severe abnormal movements while active encephalitis treatment is being escalated, and she requires a setting able to monitor for recurrent dysautonomia, aspiration, seizures, and hypoventilation.

Immediate diagnostic and monitoring items

- Verify the complete infectious-encephalitis evaluation from the hospital-day-17 LP, especially CSF HSV/VZV PCR and bacterial studies; maintain appropriate empiric coverage while completing it.
- Obtain MRI brain with and without contrast if not already completed — a normal MRI would not exclude NMDARE.
- Obtain EEG to evaluate for nonconvulsive seizures and establish a baseline; continue seizure surveillance.
- Send or confirm paired CSF and serum neuronal antibody testing, specifically ensuring CSF anti-NMDAR IgG is included — CSF is the more sensitive specimen.
- Trend CBC/CMP, creatinine/eGFR, glucose, magnesium/phosphate, CK, and liver tests during acute therapy; monitor ECG/QTc and respiratory status given dysautonomia and medication exposure.
- Clarify the historical “bad reaction to an IV medication” from prior records before IVIG if possible — an undocumented, nonspecific prior reaction should not by itself be treated as a contraindication, but any prior immunoglobulin reaction must be specifically excluded.
- Consider rheumatologic screening (ANA, dsDNA, complements, urinalysis) given the arthralgias and family history of lupus, run in parallel and not delaying treatment of the more compelling NMDARE phenotype.

How response would be assessed

Use objective neurologic markers rather than expecting rapid resolution of dyskinesias: level of arousal and command-following, speech output, sleep, dysautonomia, abnormal-movement burden, seizures/EEG, respiratory function, and a standardized severity measure if available. Improvement in autonomic instability ahead of the movement disorder can still represent early response. Persistent severe encephalopathy after combined first-line therapy should prompt formal discussion of second-line immunotherapy, typically rituximab for a strongly suspected antibody-mediated disorder.

Bottom line for morning rounds

Treat this as probable NMDA-receptor autoimmune encephalitis with a likely ovarian teratoma until proven otherwise: add IVIG or PLEX now rather than waiting for the send-out antibody, urgently pursue gynecologic-oncology evaluation and tumor removal, hold risperidone, continue close medical-neurologic monitoring, and defer psychiatric transfer. If she fails to improve after combined first-line therapy, or worsens, escalate to second-line therapy — generally rituximab — in conjunction with neuroimmunology.

7. Reflection

What makes this case worth building is not the diagnosis — the phenotype is textbook enough that most readers will land on anti-NMDA-receptor encephalitis within the first page. It is that three services arrived at the same working diagnosis independently, from three different vantage points in the chart, while the one test that would have made it easy stayed pending throughout. Psychiatry saw a first-episode psychosis with odd movements and asked a narrow medication-safety question. Medicine, reviewing three scanned outside images nobody had looked at yet, reframed the entire admission. Neurology, arriving after an acute decompensation, had to decide whether to add immunotherapy on a probability judgment rather than a confirmed result — and explained, in writing, exactly what would have to be true for that judgment to be wrong.

The second layer is quieter: none of this holds up if the numbers are wrong. Reconciling the CSF and imaging exhibits against the actual source images — rather than against whichever note repeated them last — caught a cluster of transcription errors (glucose swapped for lactate, an IgG index off by an order of magnitude) that would have changed how a reader interprets the CSF picture if left uncorrected. That reconciliation discipline is as much the point of this exercise as the clinical reasoning itself.

Sources

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