

## Introduction

Paraneoplastic syndromes are neurological syndromes associated with underlying tumors. In contrast, autoimmune channelopathies arise either in association with or independent from malignancy. Cystic ovarian teratomas are often associated with Anti-N-methyl-D-aspartate receptor (NMDAR) encephalitis. We discuss a patient with initial presentation of dermoid cyst and subsequent development of neuropsychiatric symptoms suspected to be secondary to voltage-gated K<sup>+</sup> channelopathy and ovarian teratoma.

## Case Presentation

A 30-year-old female with a medical history of anxiety presented with two days of sharp left-lower quadrant pain radiating to back and worsened when bending forward. Initial abdomen/pelvis CT showed right ovarian dermoid cyst measuring 3.5 x 3.4 x 3.2 cm, and she was recommended outpatient follow-up. Two months later, she complained of visual hallucinations of geometric shapes, B&W lines, difficulty with balance, and an impending sense of doom. She developed episodes involving seizure-like activity, bizarre thoughts, and agitation followed by flat affect. The patient was vitally stable with unremarkable CT abdomen/pelvis, MRI brain, and lumbar spine. EEG revealed excess beta activity. Tumor markers (AFP, CA-125, and hCg) were normal. Given the acuity of the patient's neuropsychiatric symptoms, right oophorectomy was planned; however, postponed per patient preference.

Further workup with lumbar puncture was normal (Table 1), and a positive CSF antibody for voltage-gated potassium channel increases from 0.06 to 0.16 nmol/L. She presented a month later for episodes of upper extremity muscle clenching, agitation, visual changes, and sleep-wake cycle disturbances. Initially, she received Dilantin and prednisone, but was later placed on a five-day course of IVIG for suspected paraneoplastic syndrome and Keppra for ASD. Repeat brain imaging was normal. Following salpingo-oophorectomy, she was discharged with prednisone, Keppra, and hydroxyzine. The patient had improved neuropsychiatric symptoms and continued urogynecological follow-up for recurrent vulvodynia.

**Table 1.1** CSF Findings

Components	Value	Reference Range
Glucose, CSF	61 mg/dL	40-70 mg/dL
Protein, CSF	20 mg/dL	15-45 mg/dL
Color, CSF	Colorless	Colorless
Neutrophils, CSF	4%	0-6%
Lymphocytes, CSF	83%	40-80%
Nucleated Cells, CSF	1	<=5
Meningitis/Encephalitis Panel	Negative	Negative
Fungal Culture	Negative	Negative
Anti-NMDA receptor antibody	Not detected (<1:1)	Not detected (<1:1)
Paraneoplastic P1 CSF (Anti-Hu, Anti-Yo, Anti-Ri Abs)	<1:10	<1:10
Oligoclonal banding, CSF	No oligoclonal banding	No oligoclonal banding
Cytology, CSF	Negative for malignancy and inflammation	Negative for malignancy and inflammation
CASPR2-IgG CBA	Negative	Negative
LGI1-IgG CBA	Negative	Negative

## Discussion

Our objective was to review a unique presentation of autoimmune encephalitis and identify the need for clinical management guidelines.

Autoimmune encephalitis presents due to antibody-mediated CNS damage. Voltage-gated potassium channel (VGKC) encephalitis can progress to intractable epilepsy with suggested mechanisms involving high potassium channel concentration and epileptogenic potential of circulating antibodies. To our knowledge, this is the first reported case of an initial presentation of teratoma leading to encephalitis despite normal NMDA panel and positive CSF VGKCAb titers. Clinical management requires coordination between primary care, gynecology, neurology, and psychiatry. While standard medical management is not established, teratoma resection and first-line immunotherapy with steroids, IVIG, and plasmapheresis have been reported to help achieve a neurological response.

## References

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