Neuropsychiatric Manifestations of Tumefactive Demyelinating Lesion

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INTRODUCTION

Tumefactive demyelinating lesion (TDL) is a rare variant of multiple sclerosis, occurring in 1-3/1000 cases of multiple sclerosis[1], that can present with a wide variety of neuropsychiatric symptoms.[2] Unlike typical presentations of multiple sclerosis characterized by focal deficits which correspond with the anatomic location of the demyelinating lesions, TDL typically causes diffuse neurological symptoms due to mass effect including changes in cognition and affect which may initially appear to be psychiatric in nature.[2] The following case report describes a patient who initially presented with depressive and psychotic symptoms attributed to primary psychiatric conditions.

CASE

History of Present Illness

- 26-year-old female brought to ED by family for **altered mental status for 1 week**. Family reports patient "seeming off", **bizarre behavior, responding inappropriately to questions**. She was sent home from work for the change in behavior and had been unable to take care of her children.
- Remote history of depression took antidepressants previously but has been stable for several years without the use of medications
- Smokes marijuana 2 times per week. Denies other drug use but family is concerned about other substance abuse by patient causing her current presentation.

Physical Exam

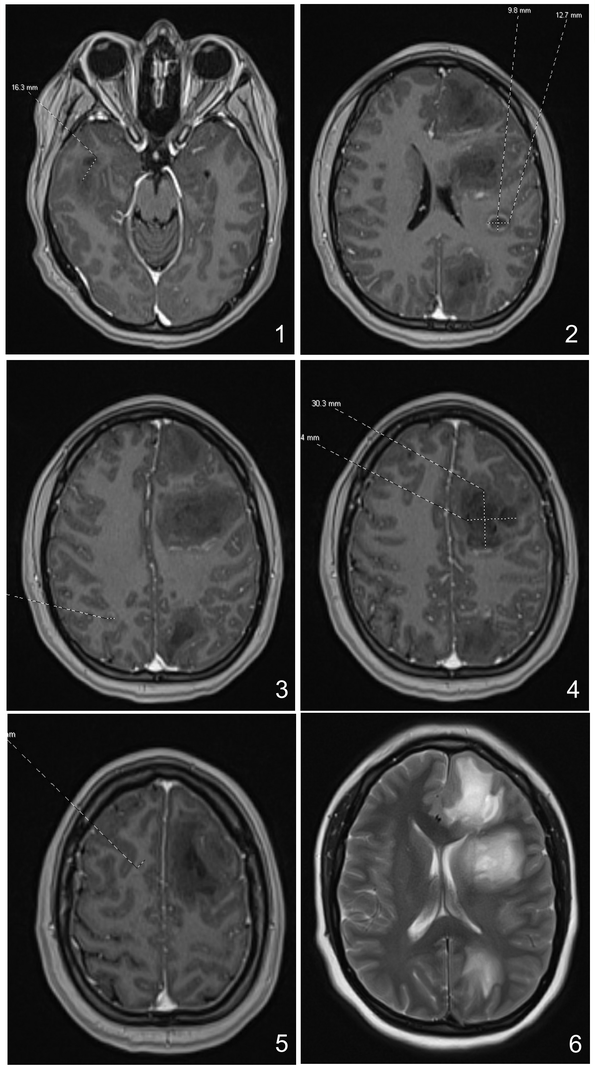
- Initial Vitals: BP 128/75, Pulse 54, Temp 98 ° F, RR 17, SpO2 98%
- No pertinent findings on physical exam. Neurological exam normal.

Mental Status Exam

- **Oriented to place and person only**. Depressed with flat affect and significant psychomotor retardation. Confused with **impaired cognition, memory, and attention. When asked if she would hurt herself, she stated "probably".**

Labs

- CMP and CMP unremarkable
- TSH within normal limits
- Pregnancy test negative
- UDS positive for cannabinoids
- UA contaminated sample otherwise unremarkable



Figures 1-5: MRI T1 enhancing and non-enhancing lesions of right temporal, bilateral frontal, and bilateral temporal lobes.
Figure 6: MRI T2 hyperintense lesions of the left frontal and left parietal lobes.

COURSE OF HOSPITALIZATION

Admitted to Psychiatric Inpatient Unit

- Diagnosed with **major depressive disorder with psychotic features**. Started on Sertraline 25mg and Olanzapine 5mg nightly which were tapered to 50mg and 10mg, respectively.
- Hospital Day 8: Failure to respond to treatment, as well as new-onset nausea and emesis, prompted ordering a **head CT which revealed vasogenic edema within the left frontal, parietal lobes and bilateral temporal lobes, mild mass effect on the left lateral ventricle with midline shift**.

Transferred to Medicine Service

- Repeat Labs: leukocytosis with a neutrophilic predominance (WBC 13.01, ANC 11.34), inflammatory markers elevated (CRP 1.10, ESR 35).
- **MRI: T1 enhancing and non-enhancing lesions, T2 hyperintense lesions** (Figures 1-6).
- Started dexamethasone for edema and inflammation, oxcarbazepine for seizure prophylaxis, empiric broad-spectrum IV antibiotics. TTE unremarkable. Blood cultures negative.
- **Progressive neuropsychiatric symptoms**; episodes of inconsolable screaming and weeping, global aphasia, moderate right upper and lower extremity weakness. Stroke workup unremarkable.
- Brain biopsy obtained. Intraoperative pathology report: **reactive astrocytes with macrophages, suggestive of demyelinating disorder. Presumptive diagnosis of multiple sclerosis.**
- Started **5-day course of IV methylprednisolone**. Antibiotics were discontinued. Olanzapine was discontinued and Sertraline was continued.
- Gradual improvement of extremity weakness and aphasia however, **suffered residual neurologic deficits**. Discharged to home with family, was able to follow commands and communicate verbally.
- Final pathology report: **active demyelinating lesion**. Patient followed in outpatient neurology clinic, repeat imaging showed new T2-enhancing lesions confirming the diagnosis of **multiple sclerosis given dissemination of lesions in time and space**.[3] Patient began disease-modifying therapy, monoclonal antibodies – ofatumumab (Kesimpta).

DISCUSSION

Tumefactive demyelinating lesions pose significant diagnostic challenges as presenting symptoms can be non-specific and mimic other psychiatric and neurological conditions. Even with imaging, it can be difficult to narrow down the differential diagnosis.[4] It is important to maintain a broad differential diagnosis for patients with new-onset psychosis. All cases of new-onset psychosis require medical workup to exclude medical and toxic causes. Baseline head imaging should be strongly considered as part of the workup.

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