

A Complex Journey through Misdiagnosis to Functional Neurological Disorder

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BACKGROUND

- -Functional Neurological Disorder (FND) often presents with symptoms that resemble a range of neurological diseases and may co-occur with other conditions.
- -Diagnosing FND can be challenging, and misdiagnosis can trigger unnecessary, potentially harmful interventions.
- -Here is a case initially diagnosed with Myasthenia Gravis (MG), post-IVIG, on corticosteroid treatment, presenting with non-epileptic seizures, insomnia, and catatonic-like symptoms, ultimately identified as FND with mixed symptoms.

CASE

- -A 29-year-old female with prior psychiatric history of childhood trichotillomania, postpartum depression, anxiety, and prior ED visits for varied complaints including syncope, panic attacks, vertigo, palpitations, headaches, and geniculate neuralgia.
 -She was initially diagnosed with acute inflammatory demyelinating polyneuropathy (AIDP) and received IVIG in early December of last year.
 -During a subsequent admission in mid-December, she was diagnosed with seronegative MG, received IVIG and started on prednisone and pyridostigmine. Poor sleep pattern started since then.
- -She had another hospitalization in less than a month which was approached as myasthenia flare up and treated again with IVIG.
- -Was readmitted in February for new onset of seizure-like activity. Neurology investigations, including head CT, MRI, vEEG, yielded negative results, suggesting a non-epileptic seizure disorder. Additionally, negative MG antibody testing, no thymoma on chest CT scan and atypical symptom progression per further history taking and records, was less indicative of MG.

- -Psychiatry was consulted for suspected mania vs catatonia:

 Mania was deemed unlikely given the absence of other manic symptoms.
- -Catatonia was considered due to brief episodes of unresponsiveness but ruled out due to their short duration, intermittent frequency, and low Bush-Francis score.
- -Diagnosis of FND with mixed features was made.

| WORKUP | FINDINGS |
|-------------------|---|
| Seizure | -CBC/diff: mild anemia; CMP: normal, CK: 66, LA initial 2.3, repeat 1.9 |
| | -U/A: normal, UDS: positive for benzos (iatrogenic), Urine pregnancy test: |
| | Negative |
| | -Head CT scan, MRI brain w/o contrast, 1-hour video EEG (vEEG): All |
| | Normal |
| Syncope | -EKG & Transthoracic Echocardiogram (TTE): Normal |
| | -Chest X-ray and Chest CT scan: Normal (no Thymoma) |
| Neuropathy | -Syphilis screen, HIV: Negative |
| | -Vitamin B12, TFT, SPEP, Immunofixation, Hgb A1C: Normal |
| Protein Gap | -Protein - Albumin > 4 |
| | -HIV, hepatitis, and multiple myeloma can elevate globulin gap: All |
| | negative |
| Myasthenia Gravis | -AChE antibodies and MuSK antibodies: Negative |
| | -AChR antibody recheck: 0.1 (Negative) |
| | -Reported abnormal EMG (NM transmembrane abnormality) at another |
| | center; outpatient SFEMG after discharge: Pending |
| Neurological Exam | -Neuro exam including Cranial Nerves, reflexes, sensation and motor |
| | function all normal except absent Achilles tendon reflex bilaterally; while |
| | having normal vibration, strength, and no numbness. |
| | -Clinical interpretation: Possible asymptomatic chronic polyneuropathy; |
| | possible normal variant |

TREATMENT

- -Treatment involved addressing sleep disturbances and mood symptoms, resulting in significant improvement in 2 days with Mirtazapine 15 mg (for sleep/appetite/depression) and Trazodone 100 mg for sleep.
- -To confirm the absence of MG and stop steroids, an outpatient single-fiber EMG was scheduled.
- -The patient was informed of the diagnosis and provided with further information about FND, and scheduled CBT.
- -At the one-month follow-up, the patient reported that despite initial relief with Mirtazapine and Trazodone, she began waking up frequently at night and experiencing increased anxiety after two weeks. -Trazodone was switched to Quetiapine XR 25 mg at bedtime. In a follow-up call three days later, the patient reported improvement in her sleep, although she experienced some daytime sedation. She was encouraged to continue her therapy sessions and follow up with her FND expert therapist.

DISCUSSION

-Clinicians should avoid relying on a single sign to diagnose FND and rather base the diagnosis upon multiple features that constitute the syndrome. A delicate balance is essential, ensuring neither overlooking neurologic/general medical disorders nor failing to accurately diagnose FND(Stone, 2020). -Undiagnosed FND is more common than misdiagnosed cases of other neurological conditions as FND and many patients have both FND and a recognized neurologic condition (Stone, 2009). Over 50% of patients show no improvement over time, with better outcomes in younger population -This case highlights the importance of refraining from premature diagnoses, such as mania, catatonia, and narcolepsy, based on single encounters. A comprehensive, longitudinal assessment not only aids in identifying FND features earlier but also helps avoid misdiagnoses of other conditions and unnecessary medication, ensuring appropriate management.

CONCLUSION

FND diagnosis is challenging but key for proper treatment. Misdiagnoses may lead to unnecessary, harmful interventions. Sleep disturbances could originate from mood disorders, or medication.

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