THE CARLAT REPORT HOSPITAL PSYCHIATRY

A CME Publication

Subscribe today! Call 866-348-9279

UNBIASED INFORMATION COVERING HOSPITAL PSYCHIATRY

Worth 2 CME credits!

Victoria Hendrick, MD
Editor-in-Chief

Volume 5, Issue 1&2
January/February/March 2025
www.thecarlatreport.com

IN THIS ISSUE

Focus of the Month:
Medical Mimics in Psychiatry

Learning Objectives

After reading these articles, you should be able to:

1. Distinguish common medical mimics from psychiatric disorders.
2. Implement effective treatment strategies for patients with functional neurological disorder and akathisia.
3. Diagnose and manage factitious disorder.

Medical Conditions That Mimic Psychiatric Illnesses

Manjit K. Bhandal, MD. Psychiatrist, Kaiser Permanente, North Valley, CA.

Michael Donath, MD. Psychiatrist, Venice Family Clinic, Los Angeles, CA.

Drs. Bhandal and Donath have no financial relationships with companies related to this material.

In this review, we describe six medical conditions (ordered alphabetically) that not only produce psychiatric symptoms but also mimic psychiatric disorders to the point where clinicians might fail to understand the underlying illness. In these cases, accurate diagnosis is essential to getting your patients well. Keep these conditions in mind when patients present with puzzling physical symptoms or when the time course of their illness seems unusual.

Acute intermittent porphyria

Acute intermittent porphyria (AIP) is a rare genetic disorder caused by a deficiency of the enzyme porphobilinogen deaminase, essential for heme production. This deficiency leads to the

Continued on page 2

Highlights From This Issue

Feature article
Learn how conditions like lupus, Lyme disease, and anti-NMDA receptor encephalitis can present with psychiatric symptoms and complicate your diagnoses.

Feature Q&A
Explore the latest on functional neurological disorder, previously known as conversion disorder, with insights on diagnosis and treatment.

Article on page 6
Delve into the challenges of identifying deceptive illnesses and learn how to manage them effectively through teamwork, supportive confrontation, and thorough documentation.

Q&A on page 8
Discover practical strategies for recognizing and managing akathisia, along with novel treatment tips.

Q&A With the Expert

Fundamentals of Functional Neurological Disorder
Gabriela S. Gilmour, MD, FRCPC

Clinical Assistant Professor of Neurology, University of Calgary, Alberta, Canada.

Dr. Gilmour has no financial relationships with companies related to this material.

CHPR: Please begin by giving us an overview of functional neurological disorder.

Dr. Gilmour: Functional neurological disorder, or FND, is a condition in which patients experience neurologic symptoms that result from, essentially, malfunctioning brain networks. The problem seems to lie with how signals are relayed from the brain to control movement and with how signals are integrated to interpret information coming from the body. I find the hardware/software analogy quite useful: If we think of the brain as a computer, FND represents a malfunctioning of the software. Patients with FND experience a wide variety of neurologic symptoms, ranging from weakness and movement problems to seizure-like episodes and cognitive symptoms.

Continued on page 4

Medical Conditions That Mimic Psychiatric Illnesses

Continued from page 1

accumulation of porphyrins, which are intermediates in the heme synthesis pathway, and can cause episodes or "attacks" of severe abdominal pain, nausea, and neurological issues like confusion, seizures, and even paralysis. AIP attacks can be triggered by factors that lower heme levels and trigger the production of porphyrins, like CYP450 inducers (eg, barbiturates, antiepileptics, smoking, and alcohol), low-calorie diets, stress, and hormonal changes like those seen in the menstrual cycle.

EDITORIAL INFORMATION

Publisher: **Daniel Carlat, MD**

Editor-in-Chief: **Victoria Hendrick, MD**

Deputy Editor: **Talia Puzantian, PharmD, BCPP,** professor, Keck Graduate Institute School of Pharmacy, Claremont, CA

Senior Editor: **Ilana Fogelson**

Director of Digital Content: **Laurie Martin**

Associate Editor: **Harmony Zambrano**

Editorial Contributors: **Manjit K. Bhandal, MD; Michael Donath, MD; Jonathan Heinzman, MD; Michael A. Strong, MD, MSEd**

Editorial Board:

Timothy Botello, MD, MPH, professor emeritus, Department of Psychiatry, University of Southern California, Los Angeles General Hospital, Los Angeles, CA

Jeff Cardenas, MD, psychiatric emergency room director, Olive View UCLA Medical Center; clinical professor, health sciences, Department of Psychiatry & Biobehavioral Sciences, UCLA David Geffen School of Medicine, Los Angeles, CA

Michael Gitlin, MD, distinguished professor of clinical psychiatry; director, Mood Disorders Clinic, Geffen School of Medicine at UCLA in Los Angeles, CA

Adrienne Grzenda, MD, PhD, clinical assistant professor, psychiatry & biobehavioral sciences, UCLA David Geffen School of Medicine and Olive View UCLA Medical Center, Los Angeles, CA

Brian Holoyda, MD, MPH, MBA, chief psychiatrist, Contra Costa County Detention Health Services, Martinez, CA

Sandra Hyams, PMHNP, psychiatric nurse practitioner, Acclaim Multi-Specialty Group, John Peter Smith Hospital, Fort Worth, TX

Michael A. Strong, MD, MSEd, clinical assistant professor and vice chair for clinical services; director of C-L psychiatry, Department of Psychiatry, University of Iowa, Iowa City, IA

All editorial content is peer reviewed by the editorial board. Dr. Carlat, Dr. Hendrick, Dr. Puzantian, Ms. Fogelson, Ms. Martin, Ms. Zambrano, Dr. Botello, Dr. Cardenas, Dr. Gitlin, Dr. Grzenda, Dr. Holoyda, Ms. Hyams, and Dr. Strong have no financial relationships with companies related to this material.

Carlat Publishing occasionally uses artificial intelligence (AI) tools, such as ChatGPT and Bard, in various stages of our content creation process, such as editing articles and creating preliminary drafts and outlines. In all cases, our content is extensively revised during the editorial process by human clinicians and by our board of medical experts to ensure quality and accuracy.

This CME/CE activity is intended for psychiatrists, psychiatric nurses, psychologists, and other health care professionals with an interest in the diagnosis and treatment of psychiatric disorders.

The Carlat Hospital Psychiatry Report (ISSN 2768-3877)

POSTMASTER: Send address changes to *The Carlat Psychiatry Report,* P.O. Box 626, Newburyport, MA 01950

Keys to diagnosis

We maintain a high index of suspicion for AIP in any cases of psychiatric symptoms with histories of unexplained acute abdominal pain. It's also helpful to obtain a thorough family history, as AIP is an inherited disorder. A urine sample during an acute attack showing porphobilinogen levels greater than 6 mg/L confirms the diagnosis.

Treatment

Treatment involves IV heme and carbohydrate loading (either as IV glucose or as a high-carbohydrate diet). The production of heme is tightly connected with the availability of energy in the body. When the body is under stress, or when it doesn't have enough energy (eg, when fasting or on a low-calorie diet), it tries to make more heme as a way to boost the energy-producing processes. However, in AIP, this leads to an accumulation of porphyrins and triggers an attack. Administering glucose (or other carbohydrates) provides the body with energy so it doesn't need to boost heme production, and this helps to alleviate the symptoms.

Treatment of acute attacks usually relieves the psychiatric symptoms. However, patients' depression and anxiety sometimes continue even after the acute attacks have ended, and we provide psychiatric treatment in these cases (Millward LM et al, *J Inherit Metab Dis* 2005;28(6):1099–10107).

Other considerations

Many medications can trigger acute AIP attacks, including benzodiazepines (eg, chlordiazepoxide, clonazepam), antidepressants (eg, phenelzine, venlafaxine), sleeping medications (eg, ramelteon, trazodone), antiepileptic drugs (eg, carbamazepine, topiramate), and antipsychotic medications (eg, quetiapine, ziprasidone). Always check the American Porphyria Foundation's Drug Safety Database before prescribing medications to patients with AIP—you'll be surprised to see the wide range of psychiatric medications that carry warnings. We do have some safe options, though, including amitriptyline, chlorpromazine, diphenhydramine, fluoxetine, fluphenazine, gabapentin, haloperidol, lithium, and lorazepam. For a full list, see: www.tinyurl.com/2x32uy9d.

Anti-NMDA receptor encephalitis

Anti-NMDA receptor encephalitis is an autoimmune encephalitis characterized by immunoglobulin G (IgG) antibodies against NMDA receptors in the central nervous system. This syndrome affects both sexes but is more common in young females. In many cases, an ovarian teratoma is the source of the antibodies.

Symptoms

Patients typically present with a prodromal viral illness followed by a quick progression to prominent psychiatric symptoms (within weeks), including agitation, catatonia, delusions, hallucinations, insomnia, and mania. Symptoms can quickly progress to life-threatening blood pressure and heart rate fluctuations, dyskinesias (uncontrolled, involuntary writhing), and seizures. Patients typically require intensive care admission.

Keys to diagnosis

Misdiagnosis is common in the early phases, and patients are often mistakenly diagnosed with schizoaffective disorder or other psychotic illnesses. You'll need to obtain cerebrospinal fluid (CSF), which can be tricky as patients may need significant sedation to remain still for a lumbar puncture. Be cautious with serum NMDA receptor antibody testing, because it has lower sensitivity than CSF testing and can be falsely low if patients are started on IV immunoglobulins or plasmapheresis before a sample is obtained. The presence of IgG antibodies in the CSF is diagnostic for anti-NMDA receptor encephalitis.

Treatment

Prompt administration of immunosuppressive therapies (IV immunoglobulins, corticosteroids, and plasmapheresis) is critical. If a teratoma is present, it must be removed for treatment to be successful.

The usefulness of antipsychotics for agitation in these cases is inconclusive and may worsen dyskinesias (Chapman MR et al, *Am J Psychiatry* 2011;168:3245–3251). We use benzodiazepines when catatonic features are present, but they can exacerbate confusion and delirium. Treatment should be tailored to each patient as guidelines for psychiatric management are still lacking (Warren N et al, *Psychological*

Continued on page 3

Continued from page 2

Medicine 2021;51(3):435–440). Most patients recover, especially with early intervention, but full recovery can take months or years.

Lupus

Systemic lupus erythematosus (SLE) is an inflammatory autoimmune disorder that affects multiple organ systems and follows a relapsing and remitting course. Primarily diagnosed in women of childbearing age, SLE affects an estimated 1.5 million patients in the US. Patients with family histories of autoimmune disorders are at elevated risk.

Symptoms

Typical symptoms include a characteristic butterfly-shaped rash over the face and nose and discoid skin lesions (round scaly red plaques, typically on the face, scalp, or ears), as well as fatigue, fever, joint pain, and swelling. Some patients also experience mood symptoms, delusions, and hallucinations that can be indistinguishable from schizophrenia, bipolar disorder, or delirium.

Keys to diagnosis

Consider SLE in patients with psychiatric symptoms who also report the characteristic symptoms of SLE listed above, especially if there's a family history of autoimmune illnesses. We've occasionally encountered patients whose main symptom was psychosis with no other physical symptoms, but on further exploration uncovered a recent history of classic SLE symptoms like rashes or joint pain. If you suspect SLE, obtain a rheumatology consult. A positive antinuclear antibody titer of at least 1:80 indicates autoimmune disorder involvement but is nonspecific for SLE. The presence of anti-double-stranded DNA antibodies confirms the diagnosis.

Treatment

Treatment consists of immunosuppressive therapies (eg, high-dose glucocorticoids, cyclophosphamide, rituximab) and plasmapheresis for more refractory cases. These help reduce mood or psychotic symptoms, but we also prescribe antidepressants, antipsychotics, or mood stabilizers as needed to speed up recovery (Yoon S et al, *J Rheum Dis* 2019;26(2):93–103). Most patients show improvement in SLE symptoms, including neuropsychiatric manifestations, within eight weeks of treatment; however, some cases improve slowly over six to 24 months.

Lyme disease

Lyme disease, or Lyme borreliosis, is a tick-borne illness with 30,000 new cases reported each year. Ticks thrive in wooded, shrubby, and grassy habitats and are endemic to northeast and mid-Atlantic states (Virginia to Maine), northern central states (Wisconsin and Minnesota), and northern California.

Symptoms

Early-stage symptoms such as cognitive clouding, fatigue, and poor appetite may be mistaken for depressive or adjustment disorders.

Later-stage symptoms like dizziness, irregular heartbeat, neuropathic pain, shortness of breath, and tremor may be misdiagnosed as an anxiety disorder.

Keys to diagnosis

Consider Lyme disease in outdoorsy patients who report fever, joint pain, or malaise, especially if they develop a gradually expanding "bullseye" rash, which occurs around the tick bite in most cases. Though less common, facial paralysis—like Bell's palsy—sometimes occurs one to three weeks after the tick bite.

Treatment

Collaborate with the patient's PCP to screen using serological testing that looks for antibodies against the bacterium. The CDC recommends a two-step process: After positive antibodies are found on screening, confirmation is made using a Western blot to detect *B. burgdorferi* proteins. A short course of antibiotics, like amoxicillin or doxycycline, cures most cases, although severe presentations may need IV antibiotics. For unknown reasons, about 10% of patients continue to experience fatigue, pain, and poor concentration after completing antibiotic treatment (□ cieszka J et al, *Reumatologia* 2015;53(1):46–48). This condition, termed post-treatment Lyme disease syndrome (PTLDS), can last for months and, unsurprisingly, can lead to anxiety and depression. Consider treating with anxiolytics or antidepressants in these cases.

Other considerations

Even among individuals who don't experience PTLDS, a history of Lyme disease appears to significantly increase their risk of mood disorders in the months following infection. One study reported a 75% higher rate of suicide among patients treated for Lyme disease compared with controls for three years following diagnosis (Fallon BA et al, *Am J Psychiatry* 2021;178(10):921–931). Reasons for the higher rate of mood instability following infection remain unclear but may be related to effects of inflammation on the central nervous system. Regardless, we develop safety plans with our patients and provide them and their families with psychoeducation about warning signs.

Neuroendocrine tumors

Neuroendocrine tumors (NETs) are rare solid malignant cancers that originate from hormone-secreting tissues, such as those found in the adrenal and thyroid glands and pancreas. NETs secrete metabolically active substances into the systemic circulation, including hormones and neurotransmitters. Adrenocortical and thyroid carcinomas overproduce cortisol and thyroid hormones, respectively, and pheochromocytomas overproduce catecholamines. Carcinoid syndrome (CS) is an NET that typically originates in the gastrointestinal system or, less often, in the lungs. CS diverts tryptophan, the precursor to serotonin and niacin, to overproduce serotonin, so patients become niacin deficient.

Symptoms

For adrenocortical and thyroid carcinomas and pheochromocytomas, symptoms include:

- Anxiety
- Insomnia
- Rapid heartbeat
- Restlessness
- Shortness of breath
- Sweating
- Tremor

For CS, symptoms include:

- Diarrhea
- Dizziness
- Flushing
- Respiratory difficulties

Watch for niacin deficiency, which can lead to pellagra—a severe nutritional deficiency that produces fatigue, abdominal pain, loss of appetite, tremor, mouth sores, and cognitive impairment.

Continued on page 10

Expert Interview – Fundamentals of Functional Neurological Disorder

Continued from page 1

CHPR: And these symptoms are often quite disabling, right?

Dr. Gilmour: Absolutely. And it's important to emphasize that this diagnosis is separate from factitious disorder and malingering. In FND, the symptoms are real, they are involuntary, and they are disabling.

CHPR: For readers who are familiar with the term "conversion disorder," it's worth pointing out that when the DSM-5-TR came out in 2022, the terminology was changed to "functional neurological symptom disorder."

Dr. Gilmour: Many people still use older terminology, like "conversion disorder," "psychogenic nonepileptic seizures," or "nonepileptic attacks," so the nomenclature is a work in progress. I would recommend using the term "functional," which can be combined into "functional neurological disorder," "functional seizures," and "functional movement disorder," for example.

CHPR: What are the most common presentations of FND?

Dr. Gilmour: FND can present with essentially any neurological symptom, but the most common are functional seizures and functional movement disorder. In functional seizures, patients experience episodes that resemble epileptic seizures or syncope with altered responsiveness. With functional movement disorder, patients present with abnormal movements, like tremor, dystonia, jerky or myoclonic-like movements, gait disorders, or weakness. Around half of patients have multiple motor phenomenologies (Gilmour GS et al, *CNS Spectr* 2023;28(6):747–755). Another common manifestation is persistent postural-perceptual dizziness, or PPPD, where patients experience chronic dizziness and unsteadiness. There are also functional cognitive disorder, functional somatosensory or visual symptoms, and functional speech or swallowing disorders.

"Patients with FND face so much iatrogenic harm—not only is it important to take away medications that are not indicated, but it's also important to communicate to other providers involved in this patient's care about the diagnosis and treatment modalities so we can prevent ongoing risks of harm."

Gabriela S. Gilmour, MD, FRCPC

CHPR: It's interesting to hear that patients often present with multiple and varied symptoms.

Dr. Gilmour: Right. Many patients with FND have more than one of these subtypes, and the subtypes often shift over time. In my functional movement disorder clinic, I focus my assessment on the most bothersome symptoms at the present time, knowing they might shift the next time I see the patient. And our patients often report other symptoms like pain, fatigue, cognitive fog, and sleep disturbances, which can be even more disabling than their primary neurological symptoms. So, we think of FND as a multisystem syndrome.

CHPR: Some years ago, I encountered a patient with functional blindness. Have you encountered such patients?

Dr. Gilmour: Absolutely, yes. I say "never say never" in FND—there is such a wide spectrum of possible presentations. With functional blindness, patients will display an optokinetic nystagmus response, indicating that light perception is happening, despite the patient's experience of blindness. There isn't much literature on this condition or on treatment strategies, but occipital transcranial magnetic stimulation has been successful in some cases (Ramsay N et al, *Eye (Lond)* 2024;38:2257–2266).

CHPR: What risk factors have been identified for FND?

Dr. Gilmour: Given that FND is a complex neuropsychiatric condition, there isn't a single unifying etiology. It's best conceptualized using a biopsychosocial framework. One important risk factor is stressful life events and maltreatment, and in particular emotional neglect. Patients with FND are 3.5 times more likely to have experienced adverse life events compared to control subjects (Ludwig L et al, *Lancet Psychiatry* 2018;5(4):307–320). Female gender is sometimes considered to be a risk factor, but this may also partially be attributable to higher rates of childhood adverse events in women (Kletenik I et al, *Mov Disord Clin Pract* 2019;7(2):177–181). And psychiatric comorbidities like posttraumatic stress disorder, depression, and anxiety appear to be risk factors. Also, patients with certain neurological disorders like migraine, multiple sclerosis, and Parkinsonism have higher rates of FND (Walzl D et al, *J Neurol* 2022;269(2):654–663).

CHPR: Please describe the workup of FND.

Dr. Gilmour: I'll start by saying that FND is no longer considered a diagnosis of exclusion or medically unexplained. We assess for positive signs, both on history and physical examination, that allow us to rule in this diagnosis. Patients often describe a sudden or explosive onset of symptoms followed by a rapid accumulation of individual symptoms that lead to disability. It's different from other neurological illnesses, which often have an insidious onset with a progressive worsening. Inconsistency in symptoms is common; patients describe good and bad days, waxing and waning of symptoms, and even spontaneous remissions. On exam, we look for evidence of symptomatic inconsistency, like variability of neurological signs, distractibility, and worsening of signs with attention. Variability manifests as changing patterns of neurological deficits throughout the assessment, such as a functional tremor changing frequency, direction, or body location. We observe the effects of distractibility when a patient's abnormal signs cease as they stop paying attention to the affected body region. Conversely, signs will worsen in the context of attention, for example when we formally examine an affected limb. By following these steps, we can rule in the diagnosis, in many cases without requiring further investigation.

CHPR: And for functional seizures, you'd also want to get an EEG, right?

Dr. Gilmour: Right. There are some FND presentations where we typically need to do more investigations, including functional seizures. There, a video EEG is the gold standard, ideally capturing one of the events in question.

Continued on page 5

Continued from page 4

CHPR: Once you've made your diagnosis, what do you do next?

Dr. Gilmour: The most important next step, which is often overlooked, is to give patients a clear diagnostic explanation. It's hard for people with FND to get better if they don't understand why they have their symptoms. I start by saying, "You have functional neurological disorder," then I explain how the symptoms arise, showing patients the positive signs on their examination. I also emphasize that the condition is common and disabling, and I provide patients with the website www.neurosymptoms.org so that they can continue to educate themselves about FND after our consultation. A fantastic resource to help patients understand their diagnosis is Jon Stone's article in *Practical Neurology,* "Functional Neurological Disorders: The Neurological Assessment as Treatment" (Stone J, *Pract Neurol* 2016;16(1):7–17).

CHPR: It must be very reassuring for patients to hear that their symptoms are real.

Dr. Gilmour: Absolutely. When I tell a patient with FND, "I don't think you're crazy," I see their shoulders relax. It's very important for them to hear that their symptoms and experiences are real and valid.

CHPR: And what does treatment entail?

Dr. Gilmour: Rehabilitation for FND varies based on the most disabling symptoms. So, for functional movement disorder, we can help patients improve their motor symptoms with FND-specific physiotherapy techniques that aim to minimize self-focused attention, instead using distraction (such as with a cognitive task, like counting backwards by sevens) or tapping into automatically generated movements (like tossing a ball between the hands). It's important for rehabilitation to focus on function, using specific activities as goals. It's helpful to have patients use visualization to imagine normal movement prior to performing the movement, and I also like to use mirrors and video as they provide both distraction and feedback about patients' movements. For functional seizures, various forms of psychotherapy are helpful, with cognitive behavioral therapy having the most evidence (Goldstein LH et al, *Lancet Psychiatry* 2020;7(6):491–505). Mindfulness-based psychotherapy, psychodynamic psychotherapy, and prolonged exposure therapy also appear effective at reducing symptoms.

CHPR: Do you have any other tips for us to keep in mind when we treat these patients?

Dr. Gilmour: Before initiating a rehabilitation program, I ensure that patients understand and agree with their FND diagnosis. This is necessary for the therapeutic techniques we offer, which can be quite intensive and challenging. Are they ready and motivated to improve their symptoms? Are there barriers that first need to be addressed, like severe pain or fatigue? Have we identified their most bothersome symptoms so that we can select the most appropriate rehabilitation modality? So, we take an opt-in approach to rehabilitation.

CHPR: And what is the prognosis for patients with FND?

Dr. Gilmour: Without treatment, the prognosis is quite poor. A large systematic review of patients with functional movement disorder found that after about a mean duration of seven years, 40% of patients were unchanged or worse, and only about 20% had gone into remission (Gelauff J et al, *J Neurol Neurosurg Psychiatry* 2014;85(2):220–226). For functional seizures, the remission rate is a little bit higher, around 40%–50%. The mortality rate of patients with functional seizures is higher than the general population, so this isn't a benign condition (Nightscales R et al, *Neurology* 2020;95(6):e643–e652). But with treatment, about two-thirds of FND patients get better and experience improvement in their quality of life. It's important to think of FND as a chronic condition that will involve relapses over time, and the goal with rehabilitation is to support a patient's self-management strategies so that they can recognize and manage their exacerbations when they occur.

CHPR: What medications, if any, are used to treat FND?

Dr. Gilmour: Generally speaking, there is minimal evidence for the use of medications in the treatment of FND symptoms. If there is a psychiatric comorbidity, like depression, anxiety, or posttraumatic stress disorder, I often use psychiatric medications like antidepressants as appropriate for these symptoms. And for PPPD, there is some evidence for the use of selective serotonin reuptake inhibitors or serotonin/norepinephrine reuptake inhibitors in reducing dizziness (Staab JP, *Semin Neurol* 2020;40(1):130–137).

CHPR: Do you find yourself discontinuing medications that patients might not actually need, like antiepileptic drugs for patients with functional seizures?

Dr. Gilmour: Yes. Patients with FND face so much iatrogenic harm, so not only is it important to take away medications that are not indicated, but it's also important to communicate the diagnosis and treatment modalities to other providers involved in the patient's care so we can prevent ongoing risks of harm. But about 20% of patients with functional seizures also have comorbid epileptic seizures, so we must be very careful about not discontinuing antiepileptic medications until we are certain there haven't been any epileptic events in the past (Kutlubaev MA et al, *Epilepsy Behav* 2018;89:70–78).

CHPR: In my experience, colleagues in neurology have often been reluctant to taper and discontinue antiepileptic drugs in patients with functional seizures even when the evidence of prior epileptic events is very slim. However, once we get a multidisciplinary dialogue going and have sufficient evidence for everyone to feel confident that it's the right thing to do, then we have been able to successfully discontinue these medications, though this process can take some time.

Dr. Gilmour: Right. It's not a risk-free decision, so it must be done with caution. Collaboration among team members is vital to achieve the best possible outcomes for our patients.

CHPR: What advice do you have for psychiatrists who treat patients with FND?

Dr. Gilmour: Start by helping patients understand their illness, and consider using bedside techniques that

Continued on page 6

Continued from page 5

can be helpful even in a short visit, like sensory grounding techniques, mindfulness, and relaxation and breathing techniques. We often see autonomic hyperarousal in patients, and these techniques are helpful not only for addressing those symptoms, but also for reducing FND symptoms. Other helpful interventions include screening for and managing comorbid psychiatric conditions, assessing for contributing psychosocial factors, and providing or facilitating psychotherapy.

CHPR: Please elaborate on those sensory grounding techniques.

Dr. Gilmour: Sensory grounding techniques can help for episodic symptoms or in moments of symptom escalation. The technique I often use is "5, 4, 3, 2, 1": having the patient identify five things in the environment that they can see, four they can hear, three they can feel, two they can smell, and one they can taste, for example. Many of my patients carry something in their pocket, like a smooth stone, to help them ground in moments when symptoms escalate. They might also carry sour candies. I'm from Canada, where it gets really cold in the winter, so my patients will use their cold hands to touch their face as a seizure is coming on, and this helps prevent the seizure from occurring.

CHPR: Thank you for talking with us about FND. It's one of the most fascinating conditions we encounter.

Dr. Gilmour: Maybe I'm biased, but I totally agree.

CHPR: Thank you for your time, Dr. Gilmour.

❖ ❖ ❖

Factitious Disorder: Navigating the Challenges of Deceptive Illnesses

Jonathan Heinzman, MD. Department of Psychiatry, University of Iowa, Iowa City, IA.

Michael A. Strong, MD, MSEd. Clinical Assistant Professor and Vice Chair for Clinical Services; Director of C-L Psychiatry, Department of Psychiatry, University of Iowa, Iowa City, IA.

Drs. Heinzman and Strong have no financial relationships with companies related to this material.

Emily, a 34-year-old nurse, is admitted with severe abdominal pain and recurrent episodes of hypoglycemia. Despite reporting intense pain, she appears unusually comfortable. She has a history of multiple hospitalizations for similar symptoms, but extensive workups have never identified a clear cause. Emily claims numerous drug allergies and refuses to share her past medical records.

Factitious disorder (FD), previously known as Munchausen syndrome, describes a condition where individuals fabricate or induce medical or psychiatric symptoms without clear external incentives, such as financial gain or evading legal trouble. Unlike malingering, which is driven by tangible external benefits, FD stems from a psychological need to assume the sick role. This complicates its diagnosis and management, as many patients lack awareness of their underlying motivations (Lawlor A and Kirakowski J, *Psychiatry Res 2014*;218(1–2):209–218).

FD affects approximately 1% of patients on medical floors, 0.5% in inpatient psychiatry, and 0.3% in inpatient neurology. About 70% of cases occur in women, many of whom are in fields related to health care (Bauer M and Boegner F, *J Nerv Ment Dis* 1996;184(5):281–288; Yates GP and Feldman MD, *Gen Hosp Psychiatry* 2016;41:20–28). But the true incidence of FD may be higher, as clinicians often hesitate to record such a diagnosis due to the potential of damaging patient rapport through false accusations. Another factor contributing to underdiagnosis is the complexity of the disorder—since it can mimic real medical conditions, it's often difficult to rule out other diagnoses before concluding that the symptoms are factitious.

Recognizing FD

In Emily's case, you note several red flags that suggest FD. The inconsistencies between her reported symptoms and objective findings, her extensive medical history with no clear diagnoses, and her reluctance to share medical records are all indicative. In addition, you observe that her symptoms seem to worsen in the presence of medical staff and improve rapidly once medical attention is provided.

FD is diagnosed based on the following DSM-5-TR criteria: 1) falsification of symptoms, 2) deceptive behavior, 3) absence of external incentives, and 4) exclusion of other mental disorders. Key indicators of FD include:

- Rapid symptom recovery after receiving medical attention
- Symptoms only appearing when medical staff are present
- Extensive hospital visits with vague or inconsistent medical histories
- Reluctance to share medical records
- Symptoms that do not align with typical disease presentations
- Numerous reported drug allergies
- Development of new symptoms as previous ones resolve

Diagnostic workup

Your workup will vary based on the patient's presentation. For psychiatric symptoms, the diagnosis hinges on comparing the patient's symptoms against known psychiatric disorders. In cases of alleged psychosis, for example, you might see symptoms that do not match with typical patterns in schizophrenia, like visual or tactile hallucinations but no auditory hallucinations or delusions. Don't rely on a single atypical characteristic to diagnose FD—but when several unusual symptoms are present, an FD diagnosis becomes increasingly likely.

In medical presentations, access to objective test results is invaluable. Other typical presentations include chest pain, recurrent hypoglycemia, severe renal pain, and generalized skin lesions. Familiarity with these presentations can guide the choice of diagnostic tests. For a table of common medical conditions in FD, visit: www.thecarlatreport.com/diagnosingfd.

Continued on page 7

Factitious Disorder: Navigating the Challenges of Deceptive Illnesses

Continued from page 6

The medical team's suspicions strengthen when Emily's lab results show high insulin levels but low C-peptide levels, a combination that suggests she may be injecting herself with insulin. Normally, when the body produces insulin, it also produces C-peptide in equal amounts. So, when insulin levels are high but C-peptide is low, it's a strong indication of exogenous insulin use.

Management strategies

Managing FD in patients like Emily requires a balance of empathy and firmness. Here are the steps for addressing FD:

1. Supportive confrontation

A "supportive confrontation" approach, where you discuss evidence of fabricated illness openly but nonjudgmentally with the patient, can be effective (Bass C and Halligan P, *Lancet* 2014;383(9926):1422–1432). To facilitate this, arrange a multidisciplinary team meeting with the patient that includes psychiatry, medical or surgical consultants, nursing staff, and security. Present the findings, such as an implausible history or inexplicable lab results, in a nonjudgmental manner. Invite the patient to share their perspective and thoughts on the information presented. This step will help you understand their viewpoint and can reveal underlying motivations that might be driving their behavior.

2. Explaining the illness

Discuss with the patient why feigning of symptoms is the most likely explanation, and explain that further inpatient care is not indicated and could even be harmful by leading to unnecessary tests, procedures, or medications. Offer outpatient follow-up with counseling to address underlying psychological issues.

3. Documentation

We recommend completing notes and documentation prior to the confrontation in case of a discharge involving threats, strong emotions, and security personnel. This ensures that all relevant information is accurately recorded and provides an objective basis for defending the health care team's actions and decisions in the event of a contentious discharge. Consider involving the hospital's legal team, particularly if the patient is a health care worker, as a diagnosis of FD may impact their future employment and require additional reporting to licensing boards. Following the meeting, be sure to update your documentation to reflect the details of the confrontation and any subsequent discussions.

You and the care team meet with Emily to discuss her diagnosis. You tell her, "Emily, we've taken a close look at your medical history and the results from your tests. We've noticed some inconsistencies that don't quite match with any known medical conditions. It's possible that stress or other psychological factors might be contributing to your symptoms. Our aim is to support you and help you feel better. We'd like to involve our psychiatric team to explore these possibilities together and find the best approach for your care. This way, we can make sure you're getting the right kind of help and treatment."

Differential diagnosis

The differential diagnosis for FD includes several psychiatric disorders.

Malingering

This disorder occurs when a patient fabricates symptoms for clear external incentives, like financial compensation or evasion of criminal charges.

Factitious disorder imposed on another

Previously known as Munchausen syndrome by proxy, this disorder involves a caregiver fabricating or inducing symptoms in someone else, typically a child or dependent adult, driven by a need for attention or sympathy. The caregiver might give misleading medical histories, tamper with tests, or induce symptoms. In these cases, the treatment team must protect the victim by reporting to legal authorities or protective services.

Somatic symptom disorder

In this disorder, patients experience physical symptoms along with significant, disproportionate anxiety about their health, even when medical evaluations show no underlying disease or cause that matches the intensity of their distress. The symptoms and distress are authentic and not intentionally produced.

Functional neurological symptom disorder (conversion disorder)

This disorder involves neurological symptoms (like paralysis, blindness, or seizures) that cannot be explained by a medical evaluation. The symptoms are a response to psychological stressors, and as with somatic symptom disorder, they are not consciously or intentionally produced.

Antisocial personality disorder

This disorder is characterized by a disregard for the rights of others and a pattern of deceit and manipulation. While individuals with antisocial personality disorder might feign illness, it is typically for external gains, such as financial benefits or avoiding legal consequences, rather than a need to be seen as ill.

Borderline personality disorder

Individuals with this disorder typically exhibit impulsivity, unstable relationships, and self-damaging behaviors, which can sometimes include inducing or faking illness as a cry for help or to manipulate others. However, the intent behind these actions is not solely to assume a sick role but often to manage emotional pain or to seek care in dysfunctional ways. Borderline personality disorder is often comorbid with FD (Yates and Feldman, 2016).

Prognosis

The prognosis for FD is generally poor due to low treatment adherence. Less than 20% of patients acknowledge their behavior and engage in psychiatric treatment (Bass and Halligan, 2014). However, a study of individuals in online support groups for FD found that about one-fifth were successful at reducing their symptoms with various strategies, like participating in therapy and learning to communicate their needs (Lawlor and Kirakowski, 2014).

CARLAT VERDICT

FD is a complex, often underdiagnosed condition that requires careful, empathetic handling. Patients often go to great lengths to induce or fabricate medical symptoms, putting themselves at risk of genuine harm. Engage patients in frank but nonjudgmental dialogue to best manage this condition.

Q&A With the Expert

Akathisia: Manifestations and Treatments

Michael Poyurovsky, MD

Associate professor, Rappaport Faculty of Medicine, Technion Israel Institute of Technology, Haifa, Israel.

Dr. Poyurovsky has no financial relationships with companies related to this material.

CHPR: Please begin by reviewing the main manifestations of akathisia.

Dr. Poyurovsky: Akathisia is a subjective sense of restlessness, like an overwhelming urge to move or a constant inner agitation, with or without restless motor movements such as pacing, shifting positions, or fidgeting. The term comes from the Greek for "inability to sit," reflecting its primary impact on the legs. It is rare that a patient will tell you about restlessness in the hands or upper body.

CHPR: Can akathisia present in different forms?

Dr. Poyurovsky: Yes, there are several subtypes. **Acute akathisia** develops shortly after starting or increasing an antipsychotic drug treatment. When akathisia lasts for more than three months, it's considered **chronic akathisia. Tardive akathisia** shows up after long-term use of antipsychotics, often following months or years of treatment, similar to tardive dyskinesia. It's unclear why some patients develop this delayed form, but it may be linked to long-term dopamine receptor changes. Tardive akathisia can be more persistent and challenging to treat, and it often overlaps with other tardive movement disorders. **Withdrawal akathisia** arises from reducing or discontinuing medication and may occur despite long-term use of the drug without prior symptoms. **Pseudo-akathisia** involves motor movements without the subjective component of true akathisia. Understanding these subtypes is essential for effective treatment strategies.

"Akathisia can be an emergency because it can cause severe distress potentially leading to suicidal and aggressive behaviors. Don't arrange a follow-up visit without first initiating an intervention."

Michael Poyurovsky, MD

CHPR: Interesting. I hadn't realized there were so many subtypes.

Dr. Poyurovsky: There's a paper by Theodore Van Putten, "The Many Faces of Akathisia," that I always recommend to my residents. It was published 50 years ago and is only three pages long, but if you want to know what akathisia is, read this paper (Van Putten T, *Compr Psychiatry* 1975;16(1):43–47). It describes the many manifestations, including, for example, rare cases of panic attacks or sexual disinhibition as expressions of akathisia. It's very important to be aware of these various manifestations when treating patients with schizophrenia and other psychiatric disorders. I read Van Putten's work early in my career, and it prompted me to focus on researching this phenomenon. I went to work with Professor Thomas Barnes, author of the Barnes Akathisia Rating Scale (BARS), to explore treatment options for akathisia. After years of researching this clinical phenomenon, I want to highlight how challenging it can be to identify and treat akathisia.

CHPR: Please say more about this scale.

Dr. Poyurovsky: The BARS is a reliable and valid rating scale and has clinical utility. It is quick and easy to use. It measures the severity of drug-induced akathisia by assessing observable restlessness, the patient's subjective awareness of restlessness, the distress caused by the condition, and the clinician's overall judgment of severity *(Editor's note: See www.thecarlatreport.com/BARS to view the scale).*

CHPR: Are there any other symptoms that might represent akathisia?

Dr. Poyurovsky: Actually, I have noticed that obsessive-compulsive-like phenomena can sometimes be manifestations of akathisia. For example, you might see an abrupt change in behavior where a patient keeps coming to you with repetitive questions and inquiries, seeking reassurance. This compulsive, repetitive, intrusive behavior can sometimes be a sign of akathisia. This phenomenon is quite frequent, so it is important to be on the lookout for it. Unfortunately, it is not included in the BARS.

CHPR: Often I meet patients who are clearly experiencing akathisia, pacing from one foot to the other, yet when asked if they'd like treatment for that symptom, they say, "No, I'm fine." So, would that be considered pseudo-akathisia., like you said?

Dr. Poyurovsky: A patient's ability to describe what they are feeling and to attribute uneasy feelings to the effect of medication may vary. You have to ask precise questions to figure out if it's akathisia or other extrapyramidal side effects or psychotic agitation, anxiety, or anxious depression.

CHPR: What types of questions do you recommend asking?

Dr. Poyurovsky: I ask about an inner sense of restlessness specifically attributed to the lower extremities; a physical need to move and change body position; and inability to sit still or remain in one place for extended periods. I also inquire about specific times of day when the restlessness is more pronounced and the proximity of symptoms to taking the medication.

CHPR: With our patients, I worry that akathisia can sometimes manifest as agitation or can exacerbate agitation, and then they can end up getting more antipsychotic medication, which in turn worsens the akathisia.

Dr. Poyurovsky: Absolutely. It's of primary importance to distinguish akathisia from the psychomotor

Continued on page 9

Continued from page 8

agitation that frequently occurs in psychotic patients. It's one of the reasons I use the BARS in my daily practice, not just in research, as it helps accurately differentiate between agitation and akathisia.

CHPR: What's known about the pathophysiology of akathisia?

Dr. Poyurovsky: The exact cause is unknown, but it's believed to be related to dopamine receptor blockade in the brain, particularly in areas that regulate movement, like the nigrostriatal pathway.

CHPR: Going back to withdrawal akathisia, how soon after stopping the medications does the akathisia begin, and how long does it take for symptoms to resolve?

Dr. Poyurovsky: Withdrawal akathisia typically begins **within a few days to a few weeks** after stopping or reducing the dose of an antipsychotic medication. The timeline for resolution of symptoms can vary, but in many cases, symptoms resolve **within days or weeks.** However, in some patients, withdrawal akathisia can persist for a longer period, particularly if the medication was used for a long time or at a high dose. In these cases, reintroducing the medication at a lower dose may be necessary to manage the condition.

CHPR: How does the incidence of akathisia vary depending on underlying factors like the choice of medications or in different patient populations?

Dr. Poyurovsky: Akathisia is a common phenomenon. With first-generation antipsychotics, the rate is as high as 25%–30% and probably higher, especially if you use high-potency antipsychotics such as haloperidol. We initially hoped the use of second-generation antipsychotics would resolve the issue, but while the incidence of akathisia is lower, it is still a problem. A recent meta-analysis reported that depending on the type of medication, up to 17% of patients treated with second-generation antipsychotics develop akathisia (Demyttenaere K et al, *CNS Drugs* 2019;33(6):549–556). Special attention is needed when prescribing partial dopamine agonists such as aripiprazole and cariprazine, which have notable rates of akathisia. Increasingly, I see akathisia in patients with affective disorders who are given aripiprazole or cariprazine as augmentation therapy.

CHPR: Are patients with affective disorders more susceptible to developing akathisia?

Dr. Poyurovsky: Patients with affective disorders, especially depression, seem to be more vulnerable to acute antipsychotic-induced movement disorders and akathisia than those with schizophrenia. However, the mechanism for this is yet to be clarified (Gao K et al, *J Clin Psychopharmacol* 2008;28(2):203–209).

CHPR: What other factors are associated with a higher incidence of akathisia?

Dr. Poyurovsky: Patients with schizo-obsessive disorder—schizophrenia and OCD—are particularly at risk. While there don't appear to be significant gender differences in the incidence of akathisia, age is an important factor: Children and adolescents are especially susceptible and may present with more agitation, hyperactivity, and disruptive behaviors compared to adults. Ethnicity also plays a role, with Black and Asian patients appearing to be more vulnerable, and White patients less so. However, the estimates are less robust than for tardive dyskinesia.

CHPR: Can you tell us about treatment approaches?

Dr. Poyurovsky: First, I want to say that you should deal with akathisia as soon as you identify it. Akathisia can be an emergency because it can cause severe distress potentially leading to suicidal and aggressive behaviors. Don't arrange a follow-up visit without first initiating an intervention. To address it, the first approach is to modify the treatment regimen by either reducing the dose of the offending antipsychotic by 25%–50% or switching to an antipsychotic with a lower propensity to induce akathisia. Both interventions run the risk of producing withdrawal akathisia or psychotic exacerbation, so you have to be vigilant for that. The second approach is to add an anti-akathisia medication.

CHPR: When switching to a different medication, what would be your go-to antipsychotic?

Dr. Poyurovsky: Haloperidol, aripiprazole, risperidone, and cariprazine are associated with the highest risk, whereas clozapine, quetiapine, and iloperidone have the lowest *(Editor's note: For a table comparison, visit www.thecarlatreport.com/akathisiaantipsychs)*. But we cannot predict individual responses to medication. For clinicians, predicting who will develop akathisia with a particular antipsychotic and who will respond best to an anti-akathisia medication is a major challenge. We certainly would like to have better predictors of response.

CHPR: Speaking of anti-akathisia medications, which are most effective?

Dr. Poyurovsky: Two recent meta-analyses of medications with anti-akathisia effects identified propranolol and low-dose mirtazapine as the best evidence-based choices (Gambolo L et al, *CNS Spectr* 2024:1–9; Gerolymos C et al, *JAMA Netw Open* 2024;7(3):e241527). Benzodiazepines also offer some therapeutic benefit for akathisia, likely due to their nonspecific antianxiety and sedative effects. While anticholinergic agents like biperiden are effective in treating antipsychotic-induced Parkinsonism and dystonia, their clinical utility in akathisia is less clear. They may be better suited for patients with akathisia who also exhibit parkinsonian symptoms.

CHPR: Are there any drawbacks to using propranolol for akathisia?

Dr. Poyurovsky: Yes. Beta-blockers, especially propranolol, carry the risk of side effects that are often underestimated, such as bradycardia and orthostasis. In our study comparing propranolol, mirtazapine, and placebo, about one in six patients on propranolol withdrew due to these side effects (Poyurovsky M et al, *Biol Psychiatry* 2006;59(11):1071–1077). This is a significant rate, particularly when propranolol is combined with antipsychotic medications that have alpha-adrenergic effects and can also cause orthostasis. Akathisia can be an emergency, so we want to use a medication with immediate and robust effects. But propranolol needs to be titrated gradually to achieve the desired effect. Some patients respond to 20 mg, while others need up to 120 mg a day.

Continued on page 10

Expert Interview – Akathisia: Manifestations and Treatments

Continued from page 9

We typically use immediate-release propranolol for quicker symptom relief, but extended release can be an option for patients who prefer once-daily dosing or require more consistent control. We generally avoid going beyond 40–50 mg with either formulation because of the risk of side effects such as orthostasis, especially when combined with medications such as antipsychotics.

CHPR: So, what's an alternative option?

Dr. Poyurovsky: Mirtazapine, a 5-HT2A antagonist, can effectively treat akathisia at low doses (15 mg). My colleague Professor Avi Weizman and I developed this approach. At this dose, the 5-HT2A serotonin antagonism of postsynaptic receptors is prominent and may account for mirtazapine's anti-akathisia effect. At higher doses, the noradrenergic effects will become significant, potentially inducing akathisia instead. Our studies showed that even 7.5 mg of mirtazapine could alleviate aripiprazole-induced akathisia (Poyurovsky M and Weizman A, *J Clin Psychopharmacol* 2018;38(6):609–611). But do not increase to 30 or 45 mg due to the risk of opposite effects. Importantly, mirtazapine achieved an anti-akathisia effect with more convenient dosing and better tolerability than propranolol, with mild transient sedation as the only observed side effect and no significant changes in vital signs. Low-dose mirtazapine did not interfere with the antipsychotic effect of medications, but long-term use can be associated with weight gain and, rarely, agranulocytosis (Maidwell-Smith A and Kirk C, *J Med Case Rep* 2023;17(1):163).

CHPR: What about vitamin B6?

Dr. Poyurovsky: Vitamin B6 at 600 mg/day has been efficacious for antipsychotic-induced akathisia in two small randomized controlled trials conducted by the same research group (Lerner V et al, *J Clin Psychiatry* 2004;65(11):1550–1554; Midownik C et al, *Clin Neuropharmacol* 2006;29(2):68–72). Its major advantage lies in high tolerability and acceptability. However, we still need independent replication of the study results and a demonstration of its clinical utility.

CHPR: And do you treat pseudo-akathisia, or does it not need addressing, given that the patients don't report subjective distress?

Dr. Poyurovsky: This side effect should be addressed as true akathisia and adequately treated. The same holds true for akathisia with a predominance of subjective over objective fidgety movements.

CHPR: Are there any nonpharmacologic approaches that are helpful?

Dr. Poyurovsky: Psychoeducation is essential before initiating an antipsychotic. I always describe the typical symptoms of akathisia and ask patients to immediately contact me if they start feeling fidgety or restless leg movements. We tend to focus on educating patients about weight gain and metabolic syndrome, but we need to remember to talk about akathisia as well.

CHPR: Thank you for your time, Dr. Poyurovsky.

❖ ❖ ❖

Medical Conditions That Mimic Psychiatric Illnesses

Continued from page 3

Keys to diagnosis

We ask about weight loss, respiratory difficulties, flushing, diarrhea, tachycardia, and dizziness, and maintain a low index of suspicion for further workup and referral to medical services.

Treatment

Symptom improvement and time course will depend on the location of the tumor, the type of tumor, the patient's stage of disease, and the treatment modality. Successful surgical resection will have a shorter recovery period, and psychiatric symptoms typically resolve within eight weeks. For patients needing chemotherapy, treatment for the tumor may take many months to produce visible improvement. In some advanced cases, oncology interventions may only manage the psychiatric symptoms or dampen the progression of illness; unfortunately, resolution may not be achieved for these patients.

Syphilis

Syphilis is a sexually transmitted disease caused by the bacterium *Treponema pallidum*. Often called the "great imitator," its presentation can mimic many other diseases.

Symptoms

Primary stage

- 10–90 days after exposure, sores or "chancres" develop in the genitals or mouth. These are usually firm, round, and painless.

Secondary stage

- After the sores go away, patients develop a pink and brown rash all over their body. In 50% of cases, it is present on the palms and soles.
- Mucous patches resembling "snail trails" can be seen in the mouth.
- Ocular neurosyphilis can affect any part of the eye but most commonly presents as uveitis.
- Syphilitic meningitis can occur and produces stiff neck, headache, and vertigo; it can also lead to stroke and seizures.
- Other symptoms include fatigue, fever, and swollen lymph glands.

Latent and tertiary stages

- Without treatment, the infection moves into a latent stage that can last for years, during which patients are free of any outward symptoms. Many people stay in this latent stage indefinitely, but if left untreated, about 30% go on to the tertiary stage.
- The tertiary stage involves invasion of the spirochete into the central nervous system and can present with general paresis (eg, memory loss, mood changes, psychosis, seizures, dementia); it has a very poor prognosis. Irritability, depression, anxiety, apathy, and agitation are common.

Continued on page 11

CME Post-Test

To earn CME or CE credit, log on to www.TheCarlatReport.com with your username and password to take the post-test. You must answer 75% of the questions correctly to earn credit. You will be given two attempts to pass the test. Tests must be completed within a year from each issue's publication date. The Carlat CME Institute is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians. The Carlat CME Institute maintains responsibility for this program and its content. The Carlat CME Institute designates this enduring material educational activity for a maximum of two (2) *AMA PRA Category 1 Credits*™. Physicians or psychologists should claim credit commensurate only with the extent of their participation in the activity.

For those seeking ABPN Self-Assessment (MOC) credit, a pre- and post-test must be taken online at http://thecarlatcmeinstitute.com/self-assessment/

This page is intended as a study guide. Please complete the test online at www.TheCarlatReport.com. Learning Objectives (LO) are listed on page 1.

1. True or False: Patients diagnosed with Lyme disease do not have additional psychiatric symptoms once antibiotic treatment has been completed (LO #1).
   [ ] a. True [ ] b. False

2. Which of the following nonpharmacologic approaches has the most evidence for managing functional seizures (LO #2)?
   [ ] a. Visualization techniques
   [ ] b. Exercise and sleep hygiene
   [ ] c. Motivational interviewing
   [ ] d. Cognitive behavioral therapy

3. Which of the following conditions is suspected when an individual fabricates or induces medical or psychiatric symptoms without a clear external incentive, such as food, shelter, or financial gain (LO #3)?
   [ ] a. Malingering
   [ ] b. Functional neurological disorder
   [ ] c. Factitious disorder
   [ ] d. Somatic symptom disorder

4. Which of the following conditions can mimic schizoaffective disorder with a prodromal viral illness, heart rate fluctuations, dyskinesias, or seizures (LO #1)?
   [ ] a. Anti-NMDA receptor encephalitis
   [ ] b. Hashimoto's encephalopathy
   [ ] c. Systemic lupus erythematosus
   [ ] d. Syphilis

5. Which of the following scales is used to assess for severity of akathisia (LO #2)?
   [ ] a. ABS [ ] b. BARS [ ] c. CIWA [ ] d. GAD-7

6. Which of the following statements is true regarding the incidence of factitious disorder (LO #3)?
   [ ] a. A majority of cases occur in women in the health care field
   [ ] b. A majority of cases occur in men in the health care field
   [ ] c. There is a relation to gender but not occupation
   [ ] d. There is no relation to either occupation or gender

7. For which of the following patients should clinicians suspect carcinoid syndrome (LO #1)?
   [ ] a. A young adult with recurrent rashes and joint pain
   [ ] b. A patient with hypertension and episodes of profuse sweating and palpitations
   [ ] c. A patient with niacin deficiency and chronic diarrhea
   [ ] d. A female patient of childbearing age presenting with abdominal bloating

8. True or False: Functional neurological disorder is regarded as a diagnosis of exclusion (LO #2).
   [ ] a. True [ ] b. False

Medical Conditions That Mimic Psychiatric Illnesses

Continued from page 10

Keys to diagnosis

Early detection of syphilis is crucial, so we screen all new patients admitted to the psychiatric unit. Populations at highest risk include men who have sex with men, individuals engaging in commercialized sex, and those with substance use disorders. Initial testing consists of a nontreponemal test such as rapid plasma reagin or venereal disease research laboratory test (VDRL). If the results are reactive with titers >1:4, proceed with a confirmatory treponemal test like fluorescent treponemal antibody absorption or *T. pallidum* particle agglutination. New-onset mood and psychotic symptoms, especially in elderly patients, raise our suspicion for neurosyphilis. A combination of clinical symptoms, CSF abnormalities (pleocytosis, elevated protein, positive VDRL), and brain imaging showing cerebral atrophy help distinguish neurosyphilis from primary psychiatric illness. Keep in mind that syphilitic infection of the central nervous system, or neurosyphilis, can occur at any stage of the illness.

Treatment

For primary, secondary, and early latent stages, we use a single dose of long-acting intramuscular penicillin G. In late latent stages, three doses of intramuscular

Continued on page 12

THE CARLAT REPORT
HOSPITAL PSYCHIATRY
P.O. Box 626
Newburyport, MA 01950

This Issue:
Medical Mimics in Psychiatry
January/February/March 2025

Next Issue:
Psychiatry in Corrections
April/May/June 2025

Your subscription expires:

Renew or extend online at
www.thecarlatreport.com
or by check using the order form below.

Medical Conditions That Mimic Psychiatric Illnesses

Continued from page 11

penicillin G are given at weekly intervals, and in neurosyphilis, patients require intravenous penicillin G for 10–14 days. If a patient has a penicillin allergy, it's best to get an infectious disease consultation. In general, for primary, secondary and latent stages, we can use oral doxycycline. If a patient is pregnant, doxycycline should not be used and desensitization and treatment with penicillin G is needed. For neurosyphilis, IV ceftriaxone can be used.

Keep in mind that patients can develop fever and flu-like symptoms within the first 24 hours of any syphilis treatment, called the Jarisch-Herxheimer reaction. This should not be confused with a true penicillin allergy and is self-limiting, needing only supportive treatment.

Second-generation antipsychotic medications like aripiprazole, olanzapine, and quetiapine help reduce mood and psychotic symptoms in the tertiary stage. However, once patients progress to general paresis, their response to treatment is poor.

CARLAT VERDICT

Many medical diagnoses present with prominent psychiatric symptoms. Consider medical causes for psychiatric illnesses when your patients report co-occurring physical symptoms, like rashes, joint pain, abdominal distress, autonomic instability, or weakness. A thorough review of systems and medical and family history are important for broadening your differentials and alerting you to the appropriate screening tests.

Write for us! www.thecarlatreport.com/join