CLINICAL VIGNETTE

A Unique Presentation of Wernicke’s Aphasia

Catherine Lindsay, MD and Simon Wu, MD

Summary of Case

A 60-year-old veteran with history of heart failure, and substance use disorder was brought in by police on a 5150 hold for danger to self after he jumped out of his apartment window while trying to run from imagined assailants in his house. Upon presentation in the ED, he had an elevated affect, pressured speech, inattention, and a tangential thought process, all initially felt to be a presentation of mania with psychotic features. He was admitted to the psychiatric unit and was started on olanzapine for acute psychosis, but continued to have word salad and a tangential thought process. Given his lack of response to antipsychotics and a late age of onset for psychosis, his presentation was felt to be atypical.

On hospital day 7, medicine was consulted after his significant other provided new information that he was recently diagnosed with stroke and an unspecified cancer. On interview, he continued to be unable to understand simple commands or questions. He spoke fluently but at times with paraphasias. Medicine recommended a brain MRI to evaluate for possible stroke, which showed a subacute infarct in the left posterior-superior temporal lobe extending into the left parietal region and also a large chronic right temporal-parietal infarct (see Figure 1 for representative image from patient’s MRI brain).

Neurology was consulted and felt this subacute lesion in Wernicke’s area was consistent with his clinical presentation of fluent aphasia. Transthoracic echocardiogram showed an echodensity in the left ventricle (LV), concerning for thrombus. Thus, his strokes were likely cardioembolic in etiology and he was started on a heparin drip. CT angiogram of the neck showed no vascular abnormalities and cardiac monitoring showed normal sinus rhythm. CT imaging of his chest, abdomen, and pelvis did not show any evidence of malignancy.

He continued to have disinhibited behavior on olanzapine and divalproex. Prior to discharge, he was transitioned to warfarin. His significant other reported the abnormal behavior began five weeks prior to his hospitalization and that he had been previously diagnosed with an LV thrombus and was non-adherent to anticoagulation.

Discussion

Psychosis - Differential Diagnosis

Many medical illnesses can manifest with psychosis. Broadly, these conditions include: infectious etiologies (e.g., HIV, syphilis, viral encephalitis), endocrine disorders (e.g., hyper- or hypothyroidism, hyperparathyroidism, or hypercortisolism), metabolic disorders (e.g., hepatic/uremic encephalopathy, porphyrias), autoimmune disorders (e.g., multiple sclerosis, systemic lupus erythematosus, Sjogren’s syndrome), neurologic conditions (e.g., strokes, temporal lobe epilepsy, traumatic brain injury, dementia, Parkinson’s disease, Huntington’s disease), oncologic complications (e.g., ovarian teratoma, paraneoplastic limbic encephalitis), and nutritional deficiencies (vitamin B12, niacin, thiamine). Delirium, a state of confusion that has a characteristic acute onset, waxing-and-waning nature, and inattention with cognitive tasks, can often be misdiagnosed for psychosis.

Medication and substance use can also manifest with psychosis. These include stimulant intoxication including cocaine, meth-amphetamines, MDMA/ecstasy; depressant withdrawal with alcohol, barbiturates, benzodiazepines; hallucinogenic use with LSD, PCP, ketamine, inhalants (e.g., toluene), anabolic steroids or corticosteroids, anticholinergics, antidepressants (in setting of bipolar disorder), antimalarials, cannabinoids, and over-the-counter medications (e.g., dextromethorphan, diphenhydramine). Given this lengthy list of potential culprits, a detailed medication list and substances used prior to one’s first presentation of psychosis is needed.

Primary psychiatric diagnoses should be considered only after ruling out the above-mentioned organic causes. These primary psychiatric conditions include schizophrenia, schizophreniform disorder, and brief psychotic disorder. Diagnosis depends on the duration of psychotic symptoms. Psychosis can also coexist with mood symptoms as seen in the following disorders: schizoaffective disorder (psychosis that occurs with or without episodes of mood disturbances), bipolar disorder with psychotic features (psychosis that always accompanies manic episodes), and major depressive disorder with psychotic features (psychosis that always accompanies depressive episodes). We recommend psychiatric consultation to assist with the diagnosis and treatment of a patient’s psychosis once organic causes have been definitively excluded. Figure 2 shows a summarized schema listing possible causes of psychosis as well as a suggested initial diagnostic approach.

Stroke – Etiologies and Initial Workup

Strokes can be classified as being either ischemic or hemorrhagic, reflecting the mechanism of brain cell death.
Ischemic stroke can be further divided based on etiologies into the subtypes of thrombotic, embolic, or systemic hypoperfusion. Thrombotic strokes involve the occlusion of blood vessels that are already diseased from existing atherosclerotic plaques that then rupture, leading to thrombus formulation. These thrombi can either form extracranially (e.g., common carotid artery) or intracranially; once formed, they lead to critically decreased blood flow distal to the occlusion. Neurologic symptoms from thrombi occlusion typically fluctuate over time in a stuttering fashion. Risk factors include hypertension, diabetes, dyslipidemia, smoking, obesity, age, and family history. Embolic strokes are caused by debris typically either plaque or blood clots that has formed proximally elsewhere, such as the heart, aorta or other large vessels and travels distally to block arterial blood supply to the brain. This subtype of stroke is often characterized by sudden onset of neurologic symptoms. Another sign of embolic stroke is the presence of multiple infarcts across different vascular territories, such as what happened in our patient’s case. Individuals at risk for embolic strokes include patients with atrial fibrillation or valvular heart disease. Additionally, the presence of a patent foramen ovale (PFO) can lend to the possibility of a venous thromboembolism causing intracranial arterial occlusion and subsequent stroke i.e., “paradoxical” emboli. Strokes from systemic hypoperfusion typically affect the “watershed areas” or regions where two major cerebral arterial territories meet as these areas are most vulnerable to hypoxia. Hemorrhagic stroke can be further classified into intracerebral versus subarachnoid hemorrhage.

Initial workup of a new stroke can thus utilize these stroke subtypes to help guide testing including ambulatory ECG monitoring to look for atrial tachyarrhythmias, echocardiogram to check for a cardioembolic source and PFO, carotid imaging to evaluate for critical stenosis, and risk stratification labs including lipid panel, hemoglobin A1c.

Wernicke’s aphasia – A Brief Overview
Wernicke’s aphasia is a fluent aphasia with impaired comprehension that is associated with lesions in the posterior superior temporal gyrus. The resulting speech impairment has been commonly described as “word salad”, with many paraphasic errors either of whole word substitutions (e.g., “gate” for “door”) and/or syllable substitutions (e.g., “window” for “window”). The most common etiology is ischemic stroke, which accounts for 170,000 new cases of aphasia annually. Other causes include hemorrhagic stroke, neoplasm, CNS infections, and traumatic brain injury. Speech therapy is the main treatment for patients with Wernicke’s aphasia. The likelihood a patient will recover their language function post-stroke largely depends on the severity of the aphasia at initial presentation as well as the size of the associated infarct.

Conclusion
This case demonstrated a unique clinical presentation of Wernicke’s aphasia in the setting of LV thrombus causing cardioembolic stroke. Our patient’s mania and psychosis were initially felt to represent a presentation of primary psychiatric illness, though it would have been very atypical given his older age of onset and lack of response to antipsychotics. Further evaluation was able to determine an alternative, non-psychiatric cause for his disorganized speech and behaviors. Thus, our case highlights the importance of first ruling out stroke and other organic etiologies in individuals presenting with new onset of psychiatric symptoms, especially in older adults and regardless of patients’ prior psychiatric histories.

Figure 1. Image from MRI brain showing large subacute infarct in the left posterior-superior temporal lobe extending into the left parietal region. There is also a large old right temporal-parietal infarct.
REFERENCES


