

Medical Complications of Catatonia: A Case of Catatonia-Induced Deep Venous Thrombosis

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This report presents the case of a 29-year-old, medically healthy, African American woman with a history of schizoaffective disorder who was hospitalized at Sheppard Pratt Hospital for 3 weeks. The patient developed catatonia with severe psychomotor retardation, posturing, and refusal of oral intake, including medications. As her catatonic and other psychotic symptoms began to resolve with treatment, the patient was found to have developed a deep venous thrombosis. This case highlights the importance of having specific prophylactic treatment guidelines for catatonia or any other cause of sustained immobility in order to prevent deep venous thrombosis and other potential complications.

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Ms. A, a 29-year-old single African American woman, had received a diagnosis of schizoaffective disorder at age 25 years. She has required multiple hospital admissions for exacerbations of her illness. At baseline she was a responsible parent and ran her household. Her mother was a strong social support and ensured Ms. A's compliance with her medication treatment. The patient had been in a state hospital 6 months before this admission and had been discharged with a medication regimen of 2 mg/day of risperidone and 1250 mg/day of divalproex sodium.

Preceding this hospitalization, the patient had presented to a local emergency room with her mother. She stated that she was experiencing a "nervous breakdown" because of "emotional problems at home" over the last

week. She showed signs of hyperreligiosity, irritability, decreased appetite, and a decreased need for sleep. She had been intermittently compliant with divalproex sodium treatment and noncompliant with risperidone treatment for an unclear period of time.

At the initial mental status examination during this hospitalization, Ms. A, a young, healthy African American woman, was awake and alert. Her orientation could not be tested, as she was not fully cooperative. She was often observed talking to herself in a manner consistent with responses to hallucinations, and she seemed internally preoccupied. Her speech was clear and fluent but nonspontaneous. She displayed thought blocking and endorsed auditory hallucinations with accompanying delusions that God spoke to her and commanded her not to eat since "food is for dead people." The patient refused a Mini-Mental Status Examination as well as testing with the Acquired Involuntary Movement Scale.

The findings of her physical examination were unremarkable, and she denied any somatic complaints. Her vital signs were normal. The results of laboratory studies at admission included few remarkable findings. The CBC was normal except for mild thrombocytopenia of $137,000/\text{mm}^3$. The results of a basic metabolic panel showed normal electrolyte levels except for the potassium level of 3.3 meq/liter. The anion gap was 16. Urinalysis results showed a specific gravity of 1.030 and mild ketonuria and proteinuria. The result of a urine test for pregnancy was negative. The results of blood alcohol level and toxicology screens were negative. Her valproic acid level was 86.8 $\mu\text{g}/\text{ml}$.

Ms. A initially refused medications and oral intake, except for occasional fluids (<500 ml/day). She remained very paranoid and anxious and refused staff permission to check her vital signs or obtain samples for follow-up laboratory tests. She was often observed pacing the halls aimlessly. She refused to enter her room due to visual hallucinations in which she saw "many people." The patient's condition continued to deteriorate, with worsening insom-

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nia, formal thought disorder, paranoid delusions, and auditory and visual hallucinations. Ms. A continued to refuse treatment, and by hospital day 4 she started to exhibit catatonic features, such as extreme negativism, mutism, and posturing. She frequently sat or stood still for several hours. By hospital day 7, she was malodorous and displayed clinical signs of dehydration (warm, dry skin and dry oral mucosa). Several doses of intramuscular lorazepam were given emergently, and samples were obtained for laboratory studies, which showed that her blood urea nitrogen level was 29.3 mg/dl and her creatinine level was 1.2 mg/dl. She continued to refuse staff permission to measure her vital signs. Ms. A was retained in the hospital after an involuntary commitment hearing, and a clinical medication panel approved treating her with involuntary intramuscular neuroleptics and/or benzodiazepines.

Less than 24 hours later, Ms. A spoke and appealed the decision of the medication panel. At this time she began to accept some oral fluids and occasional medications. She was given 2 mg of oral lorazepam three times a day to start, and she promptly slept 7.5 hours for the first time since admission. On hospital day 8, she refused oral lorazepam, but she did accept divalproex sodium and risperidone, as well as more oral fluids. By hospital day 10, her catatonic features started to improve, and she became more verbal. She now allowed vital signs to be checked. Her blood pressure was 92/50 mmHg, and her pulse was 112 bpm. She continued to accept more fluids, and she ate some fruit. On hospital day 11, laboratory findings for the basic metabolic panel were normal, and Ms. A's blood urea nitrogen/creatinine ratio was normal (<20). On hospital day 14, the patient was awake, alert, and oriented to person, place, and time, and she was complying with the prescribed medication treatment. Her lorazepam dose was decreased to 2 mg b.i.d. to address her complaint of sedation. Her speech was clear and fluent and showed evidence of logical thought processes. Her paranoia improved despite continued auditory hallucinations, although the patient began to display some insight into the unreal nature of her hallucinations and paranoid beliefs. She showered and groomed for the first time that day. Over the next few days, her risperidone dose was titrated to 4 mg/day and her divalproex sodium dose was titrated to 1250 mg/day.

On hospital day 16, Ms. A was no longer responding to internal stimuli and her catatonia had resolved. On this day, the patient began to complain of right lower extremity pain involving her calf area. A medicine consultation was arranged, and the consultant confirmed right calf tenderness and edema. She was sent to a nearby emergency room,

where findings of a Doppler study showed that she had right-dorsalis pedis and popliteal deep venous thromboses. She received 100 mg/day of enoxaparin subcutaneously for 7 days, a dose of 5 mg/day of warfarin was initiated, and anti-embolism stockings were placed. Her prothrombin time and international normalized ratio were monitored, and her dose of warfarin was titrated to 7.5 mg/day. On hospital day 21, the patient developed new-onset akathisia. Her risperidone dose was tapered to 3 mg/day, and treatment with 60 mg of long-acting propranolol twice a day was initiated. The patient's symptoms improved, and by hospital day 22, she had experienced a full resolution of psychotic symptoms. Her right calf pain had also resolved. The patient was discharged, and at discharge her medications were 1 mg of risperidone three times a day, 1250 mg/day of divalproex sodium, 60 mg of propranolol twice a day, and 7.5 mg/day of warfarin. Appropriate psychiatric and medical follow-up were arranged.

Discussion

Catatonia is a syndrome characterized by an array of motor and behavioral abnormalities that require medical and psychiatric evaluation and treatment. Various medical complications that have been associated with catatonia include malnutrition, dehydration, urinary retention and obstructive nephropathy, urinary tract infection, deep venous thrombosis, pulmonary embolism, aspiration pneumonia, flexion contractures, and rhabdomyolysis.^{1,2}

This case depicts a somatically healthy young woman with a history of schizoaffective disorder who developed a deep venous thrombosis during a catatonic state. Contributing factors included immobilization, dehydration, and perhaps local trauma. Clinically, the patient's catatonia was manifested as posturing for prolonged periods of time as well as profound negativism consisting of refusal of oral intake (including medications) and physically restricting passive movement. Efforts to practice range-of-motion exercises with her proved futile, and adequate hydration was similarly difficult to maintain.

The case of Ms. A demonstrates the need for aggressive prophylaxis to prevent the development of deep venous thrombosis in patients with catatonia for whom sustained immobility is a core feature.³⁻⁵ We therefore recommend 1) maintaining an awareness of deep venous thrombosis as a complication of catatonia, with a high level of concern for stuporous patients; 2) minimizing other risk

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factors such as dehydration, infection, and trauma; 3) conducting frequent physical examinations focusing on edema and tenderness of the lower extremities; and 4) instituting range-of-motion exercises whenever clinically feasible. If instituting the last precaution is not possible due to the

patient's negativism or concerns for the patient's safety, then other deep venous thrombosis prophylactic measures (i.e., administration of subcutaneous heparin, placement of elastic stockings, use of pneumatic compression, etc.) should be proactively instituted.

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