A Psychiatric Patient With Catatonia and Pulmonary Thromboemboli

To the Editor: Our patient, a 34-yearold woman with a previous diagnosis of bipolar mood disorder from childhood and a previous episode of treated catatonia, was brought to the emergency ward because of refusing to eat and talk for the last week. The patient had first become depressed and restless, stopped her psychiatric medication, and then stopped eating. She had gradually become ill and weak and lay down in her bed, awake, not paying any attention to her surroundings.

At her first visit, she was lying down on the bed, with closed eyelids, refusing to open them. She showed mutism and negativism, but no rigidity or waxy flexibility. She did not have a fever or any other positive finding on her physical examination. She had a normal sinus rhythm and normal heart rate without any abnormal findings on her electrocardiogram. Her routine lab tests were also normal. The initial diagnosis was catatonia, and she was treated with 2 mg of oral lorazepam, every 8 hours.

On the second, third, and fourth days, her mental status did not change, other than eating and drinking a little bit. The internists recommended intravenous infusion of 2 L of normal saline every 12 hours. On the fifth day, the patient became febrile and had a creatinine phosphokinase (CPK) rise of 3,235 and a lactate dehydrogenase (LDH) of 911. Because of poor response to benzodiazepines, electroconvulsive shock (ECT) was administered. On the sixth day, she received a session of ECT. In her lab tests, she had a rise in her liver enzymes; SGPT was 623, and SGOT was 909; CPK was reduced to 1,400. In the afternoon of that day, she started to have respiratory distress and became restless. Her axillary temperature was 38.1°C, and her respiratory rate was 30/min. She had a rise in BUN, creatinine, and BS, and a decline in calcium, hemoglobin, and platelet count. She also had a high white blood cell count (WBC). In her abdominal ultrasonography, she had an enlarged liver, with a normal echo signal, and an enlarged bladder, with 700 ml of urine. The internists ordered subcutaneous enoxaparine and a Foley catheter. After 3 hours, and a relative relief of symptoms, she started to have respiratory distress again, this time with a decline in the level of consciousness, low blood pressure, weak distal pulses, tachycardia, and cold extremities. She was transferred to the CCU, and treated with oxygen, heparin, and dopamine. In her electrocardiogram, she had sinus tachycardia, S1Q3T3 pattern with a prolonging in the Q-T interval, and a strain pattern in V1 to V3. On echocardiography, she had right atrial dilatation, mild pulmonar and tricuspid insufficiency, and a mobile mass in the right atrium. Because of unstable vital signs, brain and thorax computed tomography (CT) scans and CT angiography were not done.

On the morning of the seventh day, the patient showed delirium. She had tachycardia and a variable blood pressure. She still had a high WBC count, hyperglycemia, hypocalcemia, and a rise in BUN and creatinine. All of the psychiatric medications were discontinued, and the heparin was continued. On the afternoon of that day, she had a decline in consciousness and cardiovascular collapse. Cardiopulmonary resuscitation was not successful, and, unfortunately, our patient expired.

Comment

For many reasons, catatonia might result in medical complications, or medical conditions might accompany a catatonic state. Some treatable medical conditions are dehydration, malnutrition, urinary incontinence, dry eyes, bed sores, or joint contracture. Other complications include pulmonary aspiration, acute renal failure, adult pulmonary distress syndrome, gastrointestinal bleeding, hepatocellular damage, hypoglycemia, pneumonia, pulmonary thromboemboli (PTE), rhabdomyolysis, seizure, thrombophlebitis, and urinary retention.^{1,2}

In the literature, it is said that the risk of vein thrombosis and PTE rise in three psychiatric conditions, which is when a patient is fixed, with catatonia, and NMS.^{3–12}

Several patient case reports and autopsy case reports have shown PTE in severe catatonia with reduced psychomotor activity.^{3,4,7,9,10} Immobility and refusing to drink fluids are risk factors for this condition. One study showed that PTE led to death after 2 weeks without any warning signs,⁷ but one case report showed the occurrence of PTE in the first week of the condition.⁹

PTE is commonly mistaken for sudden cardiac arrest. In one study, 10 out of 27 lethal PTE cases were only diagnosed in autopsy;¹³ therefore, some cases might be missed and accounted for by other mortal states.

Preventive measures range from paying attention to symptoms, routine physical examinations, and

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encouraging mobility, to prescribing anticoagulants.^{8,10,14} Also, adequate hydration is important. The second step is to pay attention to complaints such as limb pain and edema and size change, chest pain, or dyspnea. In the third step, elastic compression stockings and intermittent pneumatic compression or subcutaneous heparin in high-risk patients should be considered. Patients with several risk factors receiving antipsychotics, long term fixation, immobility caused by severe catatonia or NMS, are at high risk for vein thrombosis.¹⁵

Various therapeutic agents containing heparin are used to treat and prevent vein thrombosis. Low-molecular-weight heparin (LMWH), for instance, enoxaparine, has fewer adverse reactions. Platelet count has to be done every 7–10 days, to reduce the risk of thrombocytopenia, but there is no need to check PTT. The contraindications of using heparin are active bleeding, hepatic impairment with rise in PTT, malignant hypertension, and suspicion of heparininduced thrombocytopenia.¹⁶

The important point is to assess the cost and benefit of these preventive measures, because they can be potentially harmful to psychiatric patients. One of these dangers can be the decreased coagulable state, which can lead to uncontrolled bleeding in cases of aggression. Also, the pipes connected to the intermittent pneumatic compression devices, and also the elastic compression stockings, are dangerous for patients who might commit self- injury or suicide.⁴

In general, prophylaxis, using 40 mg of daily subcutaneous enoxaparin in catatonic patients who do not respond to therapy in the first few days, especially with the presence of other risk factors for PTE, is recommended.^{14,17} Finally, we would like to emphasize that the treatment of catatonia is a complex process that needs the cooperation of the nursing staff, physical medicine specialists, diet specialists, and others who work on the prevention of PTE. Despite the preventive measures, medical complications usually occur, and the therapeutic team has to be ready for the assessment, diagnosis, and suitable therapy for these conditions.

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References

- 1. Daniels J: Catatonia: clinical aspects and neurobiological correlates. J Neuropsychiatry Clin Neurosci 2009; 21:371–380
- Philbrick KL, Rummans TA: Malignant catatonia. J Neuropsychiatry Clin Neurosci 1994; 6:1–13
- Levenson LJ: Medical aspects of catatonia. Updates in Psychosomatic Medicine and Consultation–Liaison Psychiatry. 2009; 16:23–26
- Michael I, Santokh S, Marie R: Pulmonary embolism as a complication of major depressive disorder with catatonic features. Psychiatry (Edgmont) 2007; 4:51–56
- 5. Hem E, Steen O, Opjordsmoen S: Thrombosis associated with physical restraints.

Acta Psychiatr Scand 2001; 103:73–75; discussion 75–76

- Lazarus A: Physical restraints, thromboembolism, and death in two patients. J Clin Psychiatry 2001; 62:207–208
- McCall WV, Mann SC, Shelp FE, et al: Fatal pulmonary embolism in the catatonic syndrome: two case reports and a literature review. J Clin Psychiatry 1995; 56:21–25
- 8. Arnone D, Hansen L, Davies G: Pulmonary embolism and severe depression. Am J Psychiatry 2002; 159:873–874
- 9. Woo BK: Basal ganglia calcification and pulmonary embolism in catatonia. J Neuropsychiatry Clin Neurosci 2007; 19:472–473
- Lachner C, Sandson NB: Medical complications of catatonia: a case of catatonia-induced deep venous thrombosis. Psychosomatics 2003; 44:512–514
- Van Harten PN, Van Agtmael MA: Complete anticoagulation for treatment of neuroleptic malignant syndrome? Am J Psychiatry 1995; 152:1103–1104
- Laursen SB, Jensen TN, Bolwig T, et al: Deep venous thrombosis and pulmonary embolism following physical restraint. Acta Psychiatr Scand 2005; 111:324–327, discussion 327
- Vandenbroucke JP, Bertina RM, Holmes ZR, et al: Factor V Leiden and fatal pulmonary embolism. Thromb Haemost 1998; 79:511–516
- Malý R, Masopust J, Hosák L, et al: Assessment of risk of venous thromboembolism and its possible prevention in psychiatric patients. Psychiatry Clin Neurosci 2008; 62:3–8
- Els GN, Ward V, Mark L: Deep venous thrombosis and pulmonary embolism in psychiatric settings. Eur J Psychiatry 2009; 23:19–30
- Dipiro JT, Talbert RL, et al: Pharmacotherapy: A Pathophysiological Approach. Columbus, OH, Mc Graw-Hill Companies, 2005
- Geerts WH, Pineo GF, Heit JA, et al: Prevention of venous thromboembolism: the Seventh ACCP Conference on Antithrombotic and Thrombolytic Therapy. Chest 2004; 126(Suppl):338S–400S