Resistant Depression in a Patient With Polycythemia Vera Treated With Electroconvulsive Therapy

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Polycythemia vera (PV), a blood dyscrasia, is characterized by the increased production of erythrocytes and high red blood cell mass. PV oftentimes leads to thrombosis secondary to hyperviscosity and sludging.[1] Recent studies[2] have suggested that patients with PV have an increased risk of developing depressive disorders. Treatment-resistant patients have also been described in case reports, with one responding to electroconvulsive therapy (ECT).[3] In this report, we describe a patient with longstanding PV and severe psychotic depression responding to ECT.

Case Report

A 49-year-old white married man with a 10-year history of PV, for which he was receiving phlebotomy every 3 months, and hypertension, which was currently treated with lisinopril, was admitted to the psychiatric unit for severe depressive symptoms with mood congruent psychosis. He had no prior psychiatric history. He had lost over 50 lb in the last 6 months and reported hopelessness, depressed mood, insomnia, anhedonia, and fatigue. He had persecutory delusions that he was in trouble with law enforcement as well as somatic delusions of his abdomen shaking. He also exhibited severe thought disorganization and vague suicidal ideation. Sertraline and risperidone were started for psychotic depression with poor response. His presentation continued to deteriorate rapidly. As a bizarre form of self-punishment due to his delusions of guilt, he stopped eating and laid on the ground saying, "I'm contaminated." At that time, it was decided to pursue ECT given disease severity.

His vitals were significant for intermittent tachycardia (112–127 bpm). His hemoglobin and hematocrit levels were within normal limits, as he was in regular phlebotomy therapy and had undergone a session several weeks before this admission. Other laboratory values including transaminases, alkaline phosphatase, electrolytes, and creatinine were within normal range. Abdominal imaging showed no abnormalities including normal spleen and liver size. Head computed tomography (CT) was also negative for acute intracranial pathology but did show moderate global cortical atrophy that was advanced for his age.

ECT was initiated with a Thymatron System IV (Somatics, LLC, Lake Bluff, Illinois). Methohexital and succinylcholine were used for the anesthetic and muscle paralytic, respectively. Eleven bifrontal sessions were conducted with adequate motor and electroencephalogram seizure duration. The patient was hydrated with lactated ringers (100–300 mL) prior to and during the sessions, as this is standard and may decrease sludging. Esmolol 30–60 mg was also administered after each session due to tachycardia. A baseline Mini-Mental State Examination[4] was conducted, which remained stable during and after the ECT course (29/30–30/30).

After completing the ECT course, the patient's depressive and psychotic symptoms improved significantly. His thoughts were more organized and coherent. His affect was brighter and more reactive. He was discharged home on risperidone, sertraline, and clonazepam with a referral for outpatient maintenance ECT.

Discussion

The literature on the relationship between depression and PV is scant. However, the putative etiology behind PV and depressive illnesses is thought to be precipitated by the prolonged cerebral hypoperfusion secondary to hyperviscosity and sluggish cerebral blood flow. Chronic cerebral ischemia may worsen or cause psychiatric conditions like depression that may be intrinsically more resistant to traditional therapy.[3,5,6] Chronic sludging may account for the advanced cortical atrophy in our patient that has been reported in similar cases of patients with PV and refractory depression (including one that was ECT resistant).[3,5] Another pathophysiologic mechanism that may contribute is drug-induced depression. Many patients with PV are commonly on myelosuppressive agents (ie, Busulfan); however, this was not applicable in this case, as phlebotomy was the sole treatment.[1,5]

The prevalence of depression in PV is unclear, but in one survey-based study[2] of patients with myeloproliferative neoplasms that included PV, 39.5% of respondents had a positive screening on the Patient Health Questionnaire. While limited, this study[2] does suggest a positive correlation of depressive disorders and PV. Several features of PV

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have overlapping symptoms with depression, which were present in our patient, that may cloud some diagnostic certitude, including insomnia, fatigue, and weight loss.[2] However, our patient had no signs of poorly controlled or active PV such as increased hematocrit and hemoglobin, hepatosplenomegaly, acute thrombosis, or erythromelalgia. Chronic PV likely played a role in the development of his depression as indicated by his advanced cerebral atrophy. In addition, another report[3] also described severe psychotic depression that started several years after the PV diagnosis, which might have resulted from chronic cerebral hypoxia that led to cerebral dysfunction.

Our patient was successfully treated with 11 bilateral ECT sessions. This case demonstrates the effectiveness and safety of ECT in a patient with psychotic depression with comorbid PV. The patient had no active flare secondary to his PV (ie, acute thrombotic events) that would have complicated the procedure. Minor modifications like administering fluids to prevent blood hyperviscosity and esmolol for tachycardia were implemented. More study is needed on the epidemiology of mood disorders and PV as well as effective treatments.

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