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GERIATRICS FOR THE PRACTICING PHYSICIAN

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Electroconvulsive Therapy in Older Adults

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CASE

Your patient is a 78 year old widowed woman with a history of hypertension, hyperlipidemia, osteoporosis, and major depressive disorder. She was doing well on verapamil, hydrochlorothiazide, aspirin, atorvastatin, calcium with vitamin D, nortriptyline, and trazodone until the death of her brother. During a family squabble over the allocation of the brother's assets, your patient develops chest pain, diaphoresis, and left arm heaviness & numbness. She is admitted to a hospital where workup including cardiac isoenzymes and dobutamine stress test is negative. She is discharged with a diagnosis of Atypical Chest Pain and referred for psychotherapy. Over the next few months, your patient develops poor sleep and low appetite. She can't concentrate to pay her bills, and stops going to the senior center for aerobics classes. She believes she is dying, fears being alone, and starts relentlessly telephoning you, her therapist and her son. You switch her from nortriptyline to mirtazepine, but she only gets worse. When you see the patient with her son in your office, she is contemplating suicide. Her son asks, "Isn't there anything you can do to help my mother?" Since your patient has failed trials of medication & psychotherapy and is suicidal, you refer her to a geriatric psychiatry inpatient unit for electroconvulsive therapy.

BACKGROUND

Electroconvulsive therapy (ECT) was invented in 1938 when two Italian physicians, Cerletti and Bini, used electricity to induce a seizure in their successful treatment of a schizophrenic patient suffering from catatonia.[1] In the early days of ECT, practitioners were reluctant to use ECT in older adults because of the multiple medical problems frequently found in this age group. But with procedural innovations such as oxygenation, general anesthesia, muscle relaxants, and physiological monitoring, ECT is now a safe and effective treatment for a number of medical conditions including: major depressive disorder, bipolar disorder, schizophrenia, schizoaffective disorder, neuroleptic malignant syndrome, and catatonia.[2] Despite its efficacy, the mechanism of action of ECT is unknown. One theory postulates that ECT works by targeting the hypothalamic-pituitary axis.[3] Another theory holds that ECT increases the amount of neurotransmitters in the central nervous system.[4] A third theory maintains that ECT produces changes in neurotransmitter receptor activity and density.[5]

"Is this the right treatment for my mother?" the son asks.

While several clinical considerations must be weighed in deciding whether ECT is appropriate for any given elderly patient, if a rapid response is needed because of severe psychiatric or medical morbidity, then ECT is a first line treatment.[6] ECT has the highest rate of response and remission of any form of antidepressant treatment, with up to 90% of patients showing improvement.[7] ECT should be considered for patients with major depressive disorder who have the following.

- Not responded to psychotherapeutic and/or pharmacologic interventions
- Psychotic features
- Catatonia
- Suicidal risk
- Food refusal leading to nutritional compromise
- Previous positive response to ECT
- Preference for ECT[8]

"What is the treatment like?"

A patient under consideration for ECT is seen in consultation by a psychiatrist, internist or family practitioner, and an anesthesiologist. If the patient is deemed to be suitable for ECT, the risks, benefits, and alternatives to the procedure are discussed. A consenting patient is not permitted to eat or drink after midnight on the evening before treatment. Some medications such as antihypertensives are permitted with a sip of water, while other medications such as anticonvulsants may be withheld. The patient lies on a stretcher and is attended by an anesthesiologist, a nurse, and a psychiatrist. Blood pressure, pulse oximetry, heart rate, and respiration are monitored. Electrocardiogram and electroencephalogram leads are attached. An intravenous line is started and a short acting anesthetic like methohexital is given to sedate the patient. Succinylcholine is given to induce paralysis, and a bite block is inserted to prevent dental injury. Electrodes are positioned in either bitemporal or right unilateral configuration, an electrical stimulus is applied, and the patient is carefully monitored. Just after the electrical stimulus, there is a brief parasympathetic outflow, which can cause bradycardia, hypotension, and asystole. [9] This vagal response can be attenuated by pre-treatment with an anticholinergic like glycopyrolate, which is the standard practice. The transient parasympathetic phase is followed by a sympathetic discharge, which can cause tachycardia and hypertension lasting up to twenty minutes. Excessive tachycardia can be alleviated with a beta blocker like esmolol, and excessive hypertension with nitroglycerine or other intravenous anti-hypertensive. Approximately 10% of patients will be given intravenous medications such as beta blockers to control these vital sign changes.

"Are there any side effects?"

There is a post-ictal confusion that lasts at least one hour after the seizure. Any post-ictal agitation can be alleviated with midazolam. Headache and muscle ache are common complaints that can be ameliorated with ibuprofen. Nausea is another common side effect that can be remedied by ondansetron. A less rare complication is dental injury and even rarer, is skin singing. These are carefully watched for, and a pre-ECT dental evaluation is always completed by the anesthesiologist and consulting physicians who do screening workup.

ECT is associated with a memory impairment that nearly always subsides. Anterograde amnesia typically resolves within one week to one month after the last ECT treatment, whereas retrograde amnesia may continue for up to six months. Rarely, mild residual memory complaints may persist in some patients but formal testing of patients has not documented persistent memory loss. Since older adults tend to have greater and more prolonged cognitive impairment with ECT,[10] it is sometimes necessary in elderly patients, particularly in those with pre-existing cognitive impairment, to employ unilateral electrode placement, lower electrical stimulus, and less frequent treatments.[11]

"What about her high blood pressure?"

Older adults referred for ECT frequently have pre-existing medical illnesses. While some illnesses increase the risk of ECT, none should be considered an absolute contraindication. The decision of whether to pursue a course of ECT should involve a careful weighing of the risks and benefits of treatment. The overall mortality of ECT is roughly one death per 80,000 treatments, a rate comparable to the use of general anesthesia in minor surgery.[12]

The majority of serious complications associated with ECT are cardiovascular in nature. In general, most adverse events can be prevented or limited by providing adequate oxygenation and strict control of heart rate and blood pressure. While individuals with congestive heart failure, active cardiac ischemia, severe valvular disease, uncontrolled hypertension, high grade atrio-ventricular block, and arrhythmias are at increased risk of cardiac complications following ECT; ECT has nonetheless successfully been carried out in many of them.[13] Other conditions that increase the risk of ECT include:

- Space occupying cerebral lesions or other conditions with increased intracranial pressure;
- Recent cerebral hemorrhage or infarction;
- Bleeding or otherwise unstable vascular aneurysm or malformation;
- Retinal detachment;
- Pheochromocytoma;
- Anesthetic risk rated at ASA level 4 or 5;
- Severe pulmonary condition[12]

In older patients, dosages of anticholinergic, anesthetic, and relaxant agents may need modification because of the physiologic changes associated with aging. Stimulus intensity should be selected with an awareness that the seizure threshold increases with age. Because patients with diabetes are prone to have hypoglycemia with fasting before ECT, insulin doses may need to be adjusted. In patients with asthma or COPD, the risk for post-treatment bronchospasm can be mitigated by pre-treatment with bronchodilators. Patients with osteoporosis and unstable fractures can safely be treated with ECT by using an increased dose of succinylcholine to ensure adequate relaxation.

"How long is the treatment?"

Evaluation of the patient's symptoms and any adverse reactions to treatment is needed to determine the efficacy of a course of ECT.[14] In an index episode of illness, ECT is ordinarily administered three times per week on nonconsecutive days, and is continued until the patient reaches a plateau in improvement over two treatments. Most patients reach this plateau within six to twelve treatments.

Without further treatment, half of all patients will relapse within six months, so continuation/maintenance therapy, typically consisting of psychotropic medication or ECT, is indicated for virtually all patients.[12] Continuation ECT refers to the practice of giving additional treatments at a reduced frequency for six months after discontinuation of the index course of treatment. Maintenance ECT refers to treatments administered beyond the continuation phase.

In a study of the efficacy of continuation ECT and antidepressant drugs compared to long-term treatment with antidepressants alone, the findings provided strong support for the efficacy of continuation ECT plus long-term antidepressant treatment in preventing relapse and recurrence in chronically depressed patients who have responded to acute treatment with ECT.[15] In a study of maintenance medication comparing placebo, nortriptyline alone, or nortriptyline combined with lithium in depressed patients after index phase ECT, only the combination of nortriptyline and lithium significantly reduced relapse rates.[16] Further research focusing on augmentation strategies to protect against relapse following ECT in depressed older adults is needed.

Conclusion

Your patient elects to pursue ECT. After six treatments, her depression improves and she is no longer suicidal; but she develops confusion, which abates when the frequency of her treatments is decreased from three times weekly to twice weekly. After the ninth treatment, her depression resolves. Your patient is started on venlafaxine and discharged home. When you see her in your office, your patient is tolerating the venlafaxine and doing well on an outpatient course of continuation ECT. Her goal is a trial of venlafaxine alone after she completes continuation ECT, with an option of switching to maintenance ECT if the venlafaxine trial is unsuccessful.

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