CLINICAL VIGNETTE

Electroconvulsive Therapy and Intracranial Aneurysms

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Case Report

An 83-year-old male with longstanding depression, refractory to medications, and cerebral arteriovenous malformation (AVM) was admitted to the neuropsychiatric unit for consideration of electroconvulsive therapy. The patient was found to have an AVM in the left sylvian fissure twenty years ago as part of his workup for obsessive-compulsive disorder. He eventually underwent gamma knife radiation in 2002 without further issues. Based on prior imaging, AVM has been stable in size without any evidence of new hemorrhage. He has failed extensive treatment pharmacologically and various augmentation strategies.

MRI of the brain showed hemosiderin deposition, likely related to prior bleeding, without evidence of recent bleeding. He underwent extensive consultations from geriatric medicine, neurology, and neurosurgery. Ultimately, the patient decided to defer ECT given personal concern for risk of AVM bleed. A decision was made to proceed with the use of an MAOI medication to treat his depression.

This case prompted investigation into the safety profile and potential concerns for ECT in cases of AV malformations, intracranial aneurysms, and brain lesions.

Discussion

Electroconvulsive therapy (ECT) is one of the therapeutic options for treatment-refractory depression. There are no absolute contraindications for ECT; however, the literature is very limited on ECT in patients with intracranial vascular malformations. Intracranial vascular malformations include capillary malformations, cavernous angiomas, venous angiomas, and arteriovenous malformations (AVMs). There have been selected case reports but lack of randomized, prospective, or controlled clinical studies that discuss the successful course of ECT performed on patients with intracranial malformations.

The major concern is that ECT increases vascular permeability and alters the pressure gradient across already vulnerable vascular walls, which increases risk of rupture. Additional risk involving AVMs and ECT may increase risk of seizures. AVMs themselves are epileptogenic and with ECT may decrease threshold for seizures. However, naturally occurring seizures do not appear to have a dangerous effect on intracranial aneurysms.[1]

ECT have cardiovascular effects including increases in blood pressure, heart rate, oxygen consumption, as well as changes in intracerebral pressure and blood flow. According to the American Psychiatric Association (APA) Task Force on Electroconvulsive Therapy, conditions considered at increased cardiovascular risk include: space-occupying lesions (especially those causing increased intracranial pressure), recent MI, intracerebral bleeding, or unstable vascular malformation or aneurysms.[2] Such changes may pose theoretical concern for patients with arteriovenous malformations or intracranial malformations, whether there is increased risk of rupture when undergoing ECT treatment.

The APA Task Force on ECT cited no "absolute" contraindications to ECT but "substantial risk" to be associated with ECT for patients with space occupying or other cerebral lesions with increased intracranial pressure with bleeding or unstable vascular aneurysm or malformation.[1] Patients with intracranial vascular lesions may be at risk for rupture, mass effect and increased morbidity and mortality.

ECT is associated with a hyperdynamic state characterized by arterial hypertension, tachycardia, and considerably increased cerebral blood flow rate and velocity. The cardiovascular side effects can alter the pressure gradient across an already more vulnerable vascular wall. These responses lead to concerns for increased risk for subarachnoid hemorrhage when an intracranial aneurysm coexists.[3] However, there has been a paucity of reports that show ECT-related complications or rupture of intracranial aneurysms, despite approximately 4% prevalence in the adult population.[4] Rather, ECT has been successfully performed in patients with intracranial vascular masses without adverse results.[4,5] In the Journal of ECT, there was discussion that incidentally found intracerebral aneurysms in patients undergoing ECT were not of clinical significance or concern.[6]

Beta blockade control with various agents has been utilized to control ECT-induced hypertension.[3-5] Salaris et al[3] presents additional cases of ECT performed with positive results and no adverse events. Cases included a patient with a large cerebellar venous angioma and blood pressure maintained at or below 220/120 mm Hg by beta blockade without complications. Another case discussed a patient with a 1cm aneurysm in the internal carotid artery with blood pressure maintained below 210/100 by beta blockade, also without adverse events. Additionally, ECT performed with multiple aneurysms by controlling blood pressure with nitroprusside and timolol were reported without complications.[5] Another report showed successful ECT on a patient with a left parietal angioma without complications, when systolic blood pressure

was maintained below 200 mmHg.[7] A summary of published cases on the use of ECT in patients with intracranial aneurysms is summarized in an article by Malek-Ahmadi et al.[1]

Another study recorded the cerebral artery flow velocity during ECT in a patient with an intracranial aneurysm. The combination of beta blockade with atenolol and intravenous infusion of sodium nitroprusside prevented tachycardia and hypertension, and greatly attenuated the expected increase in flow velocity in the middle cerebral artery.[8] Direct intra-arterial pressure monitoring and transcranial doppler ultrasound was used to monitor blood pressure, which remained within 20 mmHg from control. The flow velocity was only 20% above the baseline, and the systolic arterial blood pressure was maintained below 140 mm Hg.

The discussion also indicated that potential risk factors include smoking, chronic hypertension, and use of oral contraceptives. Aneurysm size greater than 10mm was a risk factor for bleeding and aneurysm enlargement was a strong predictor for rupture. The mechanisms behind rupture were more elusive, yet it is understood that wall stress in an aneurysm is increased by elevated blood pressure. Structural fatigue may result from increased flow velocity and flow velocity may be a determinant of stress at the site of the aneurysm.[8]

Additionally, coil embolization can be considered in the treatment of cerebral aneurysms as a safe and effective alternative to surgical clipping. Detachable coil embolization is performed to alleviate much of the danger presented by aneurysms. Local or general anesthesia can be used for this procedure. A catheter is inserted into an artery and then maneuvered to the aneurysm's position. Once in position, small coils go through the catheter into the aneurysm. The body responds by forming a blood clot around the coil blocking off the aneurysm. Overall, ECT has been shown to be safe after coil embolization for a central nervous system aneurysm.[9]

The authors of case and small series studies have reported on the safe use of ECT in patients with intracranial aneurysms or various AV malformations.[9-11] Patients with cerebral aneurysms and vascular malformations can be safely treated with ECT if general precautions are reviewed and monitored. Generally, neuroimaging and neurosurgery consultation should be part of the pre-ECT evaluations. In patients with hypertension, blood pressure control should be considered. For patients with intracranial aneurysms, case reports have indicated goal to keep systolic blood pressure at least <220 with preferred goal for blood pressure below 180/100.[4] Although pre-treatment with anti-hypertensive agents may reduce blood pressure peaks outside recommended range, this may not always prevent acute elevation of blood pressure induced by ECT.[6] Therefore, the decision for ECT should involve cautious review of indications with risk/benefits considered to any higher risk patient.

Further study of intracranial and systemic hemodynamics under hyperdynamic stress with various attenuating agents is necessary to evaluate the safety of ECT with aneurysms. The literature has been sparse discussing the safety profile of ECT and patients with AVM. Aneurysms in patients who anticipate ECT are still of great significance and concern, and the various reports add to the growing literature on the safety and efficacy of ECT for such patients. In general, pharmacologic treatment to decrease the arterial pressure, heart rate, and blood flow velocity, and appropriate monitoring should be considered. While such numbers do not establish unequivocal safety in this population, the ECT practitioner should continue to make a risk/benefit analysis on a case-by-case basis.

Conclusion

Electroconvulsive therapy is used to treat severe and refractory depression and mood disorders. There have been limited cases and reports on the risk of intracranial aneurysms and ECT. This case of an elderly gentleman with longstanding depression and history of AVM reviews the effects of ECT and potential risks and benefits on patients with underlying brain lesions or aneurysms.

At this time, there is limited consensus on a recommended ECT protocol for patients with intracranial aneurysms. However, literature review reports numerous cases of ECT performed in individuals with cerebral aneurysms without complications, if particular cardiovascular parameters are monitored. Surgical evaluation for correction would be advised when indicated, and pharmacological attenuation of the hemodynamic responses during ECT should be considered. Further study and evaluation of these areas are needed.

REFERENCES

1. **Malek-Ahmadi P, Hanretta AT**. Successful ECT in a patient with intracranial venous angioma. *J ECT*. 2002 Jun;18(2):99-102. PubMed PMID: 12195139.
2. **American Psychiatric Association. Task Force on Electroconvulsive Therapy**. The Practice of ECT: Recommendations for Treatment, Training and Privileging. *Convuls Ther*. 1990 Jun;6(2):85-120. PubMed PMID: 11659302.
3. **Salaris S, Szuba MP, Traber K**. ECT and intracranial vascular masses. *J ECT*. 2000 Jun;16(2):198-203. PubMed PMID: 10868330.
4. **Bader GM, Silk KR, Dequardo JR, Tandon R**. Electroconvulsive therapy and intracranial aneurysm. *Convuls Ther*. 1995 Jun;11(2):139-43. PubMed PMID: 7552055.
5. **Drop LJ, Bouckoms AJ, Welch CA**. Arterial hypertension and multiple cerebral aneurysms in a patient treated with electroconvulsive therapy. *J Clin Psychiatry*. 1988 Jul;49(7):280-2. PubMed PMID: 3391981.
6, **Najjar F, Guttmacher LB**. ECT in the presence of intracranial aneurysm. *J ECT*. 1998 Dec;14(4):266-71. PubMed PMID: 9871849.
7. **Greenberg LB, Anand A, Roque CT, Grinberg Y**. Electroconvulsive Therapy and Cerebral Venous Angioma. *Convuls Ther*. 1986;2(3):197-202. PubMed PMID: 11940866.
8. **Viguera A, Rordorf G, Schouten R, Welch C, Drop LJ**. Intracranial haemodynamics during attenuated

responses to electroconvulsive therapy in the presence of an intracerebral aneurysm. *J Neurol Neurosurg Psychiatry*. 1998 Jun;64(6):802-5. PubMed PMID: 9647316; PubMed Central PMCID: PMC2170127.

9. **Okamura T, Kudo K, Sata N, Sameshima T, Doi N, Kato N**. Electroconvulsive therapy after coil embolization of cerebral aneurysm: a case report and literature review. *J ECT*. 2006 Jun;22(2):148-9. Review. PubMed PMID: 16801833.
10. **Rasmussen KG, Flemming KD**. Electroconvulsive therapy in patients with cavernous hemangiomas. *J ECT*. 2006 Dec;22(4):272-3. PubMed PMID: 17143161.
11. **Zahedi S, Yang C, O'Hanlon D, Tanev K, Shea WP**. Electroconvulsive therapy and venous angiomas: two case reports and review of the literature. *J ECT*. 2006 Sep;22(3):228-30. PubMed PMID: 16957543.

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