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Author: Shaun Gerschwitz

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Author: Prashant Tibrewal

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Author: Tarun Bastiampillai

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Author: Rohan Dhillon

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Author: Aparna Laddipeerla

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# Electroconvulsive Therapy and Type 1 Chiari Malformation

Shaun Gerschwitz[a], Dr Prashant Tibrewal[b], Professor Tarun Bastiampillai[c], Associate Professor Rohan Dhillon[d], Dr Aparna Laddipeerla[e]

[a] **Corresponding author**
Adelaide University School of Medicine
Adelaide Health and Medical Sciences Building
Corner of North Terrace and George Street
University of Adelaide, SA 5000
Australia
Tel: (+61) 0447 820 892
Email: a1670769@student.adelaide.edu.au

[b] MBBS MD FRANZCP
Consultant Psychiatrist
The Queen Elizabeth Hospital
28 Woodville Road,
Woodville, SA 5011
Australia
E-mail: Prashant.Tibrewal@sa.gov.au

[c] MBBS FRANZCP
Department of Psychiatry
Flinders University
Sturt Road
Bedford Park, SA 5042
Australia
E-mail: Tarun.Bastiampillai@sa.gov.au

[d] MBBS FRANZCP
Department of Psychiatry
Flinders Medical Centre
Flinders University
Bedford Park, SA 5042
Australia
E-mail: Rohan.Dhillon@sa.gov.au

[e] MBBS FRANZCP
Consultant Psychiatrist
Country Mental Health SA
Glenside
226 Fullarton Road
Fullarton, SA 5063
Australia
E-mail: aparna.laddipeerla@sa.gov.au

**Electroconvulsive therapy and Type 1 Chiari Malformation.**

Type 1 Chiari Malformation (T1CM) refers to a congenital herniation of the cerebellar tonsils though the foramen magnum of ≥5mm (Pickard et al., 2012). Total prevalence in normal adults is almost 1%, yet only 0.01-0.04% of the population are symptomatic (Tubbs, 2013). The ectopic tonsils may cause obstruction of cerebrospinal fluid (CSF) flow through the subarachnoid space at the craniocervial junction (Pickard et al., 2012). Patients may be asymptomatic, or display varied symptoms due to brainstem compression, cerebellar displacement, traction on cranial nerves or interference with CSF flow. Transiently elevated intracranial pressure (ICP) due to trapped cerebrospinal fluid causes the most common symptom, headaches, in 81% of patients (Fischbein et al., 2015). 10% of patients have associated hydrocephalus (Tubbs, 2013), and an unknown percentage have idiopathic intracranial hypertension.

Depression is the most commonly diagnosed psychiatric disorder in T1CM patients, with a prevalence of approximately 32% (Fischbein et al., 2015) and is sometimes severe enough to require electroconvulsive therapy (ECT) (Electronic Therapeutic Guidelines, 2013). Traditionally, elevated ICP was considered an absolute contraindication for ECT due to perceived risk of adverse outcomes, by further increasing ICP. However there are emerging perspectives to suggest it should only be a relative contraindication (Adam and Crowe, 2003) based on a careful risk-benefit analysis.

We describe a patient with T1CM and treatment-resistant depression who received ECT.

Ms X, a 46-year-old female, had suffered from depressive illness for 26 years with limited response to multiple trials of antidepressants from different classes and augmentation with lithium and quetaipine. Psychotherapeutic interventions produced limited response. Her symptoms had recently worsened with increasing thoughts of suicide and formulation of a plan, in the context of deterioration of her previously supportive relationship with her mother. Her Montgomery–Åsberg Depression Rating Scale (MADRAS) score was 30. She requested ECT wanting “to feel better, [hoping] that ECT may improve things”. Given the emergence of suicidality, poor quality of life and the refractory nature of treatment with no symptomatic remission, ECT was considered as a treatment option.

Ms X was known to have asymptomatic T1CM, leading to the possibility of further increasing ICP, with administration of ECT. In this context concern arose that T1CM may be a contraindication to commencing ECT.

A risk-benefit analysis was conducted to determine Ms X’s suitability. No clinical evidence of elevated ICP, such as papilloedema or neurologic deficits, were noted. A neurosurgeon reviewed her case and performed T1 and T2 saggital cranial, cervical and thoracic spine MRI sequences and 3D Constructive Interference in Steady State (CISS) MRI imaging, which confirmed the diagnosis of T1CM with 5mm of tonsillar ectopia and no cord syringomyelia. As the degree of tonsillar ectopia and the presence and severity of clinical symptoms is not correlated with CSF flow (Siddiqi and Narla, 2015), MRI cerebrospinal fluid flow studies were undertaken, with no abnormalities detected. After careful deliberation and consultation, patient consent was obtained and ECT was commenced based on the asymptomatic clinical presentation and normal CSF flow studies.

Ms X received 8 bifrontal ECT treatments, three times a week over 2.5 weeks with careful monitoring of blood pressure. She experienced only minor side effects attributable to the ECT itself, including minor headaches and memory impairment on the ECT treatment day, with no clinical signs of raised ICP. She had a partial response to ECT with a reduction in MADRAS score to 16 and remission of her suicidal thoughts.

Most research into performing ECT in patients with raised ICP is based on space occupying lesions (Adam and Crowe, 2003; Moreno et al., 2011). To our knowledge, only two cases of ECT have been documented in patients with T1CM. R Katz et al. (2017) describe a patient with severe treatment-refractory depression treated successfully with ECT on a background of asymptomatic T1CM. There were no side effects, however the patient had undergone neurosurgical decompression prior to ECT (Katz et al., 2017). Therefore, this case does little to document the safety of ECT in patients with a native Chiari malformation. H Montgomery and D Vasu (2007) mention successfully treating a patient suffering somatic delusions with ECT who had a possible T1CM identified on MRI, but the suggested T1CM did not exclude her from treatment (Montgomery and Vasu, 2007). In this case of an uncertain T1CM diagnosis the results therefore must be considered with caution.

Our case suggests that a T1CM should not rule out ECT as a treatment modality in patients that require it. A careful risk-benefit analysis should be conducted looking specifically for raised ICP and obstruction, using specific MRI investigations and CSF flow studies. Given the prevalence of depression in T1CM, the situation described above may not be that uncommon in which ECT may be required.

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