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## **Subdural hematoma and electroconvulsive therapy: a case report and review of the literature**

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## **Subdural hematoma and electroconvulsive therapy: a case report and review of the literature**

### **Abstract**

Introduction: Electroconvulsive therapy (ECT) is a common therapeutic procedure in psychiatry associated with a low rate of complications. We report a rare case of subdural hematoma (SDH) associated with ECT. Clinical presentation: A 64 year old woman, with a medical history of persistent depression which required ECT six years previously, underwent ECT following a new acute episode. After four ECT sessions, a left hemiparesis occurred. Brain CT scan revealed a right SDH. The patient underwent surgery and fully recovered three months after the drainage of the hematoma. We conducted a review of all cases in which SDH was associated to ECT. Conclusion: Early stage brain imaging is indispensable prior to starting ECT. Moreover, a previous medical history of SDH may not be a contraindication to ECT. In these situations, a clinical and radiological follow-up by both the psychiatrist and the neurosurgeon during all the ECT sessions is highly recommended.

### **Introduction**

Electroconvulsive therapy (ECT) is a common therapeutic procedure in psychiatry associated with a low rate of complications. Subdural hematoma (SDH) is a frequent pathology in neurosurgery, and often followed by cranial trauma which requires hemostatic drugs. We report a rare case of SDH associated with ECT and propose to codify the management of these patients in order to avoid the occurrence of this type of complication.

### **Clinical Presentation**

A 64-year-old woman was admitted to the psychiatric department for a drug-resistant depression treated by valpromide (300mg x 2/d) and venlafaxine (75mg x 1/d).

The patient had a previous medical history of controlled hypertension and hypothyroidism and was not undergoing anticoagulant or antiplatelet drug treatment.

Six years previously, she was diagnosed as having persistent depression (PD). Initial psychiatric examination confirmed anhedonia, insomnia, anorexia, apathy, feelings of hopelessness, decreased interest in usual activities, and some suicidal tendencies. Escitalopram and clomipramine had been successively tried previously but with no significant effect. The patient received ECT which led to a substantial psychiatric improvement.

Six months prior to the present history, following an episodic memory deficit and attention disorders, the patient underwent extensive neurological investigation (including brain MRI which revealed a cortico-subcortical atrophy with no subdural collection) which confirmed the absence of any inflammatory, infectious, or neurodegenerative disorders. This episode spontaneously resolved, after adaptation of medication (the patient had been over-treated by levothyroxine, valpromide and venlafaxine), and her psychiatric evaluation was similar to the post-ECT status six years earlier. Unfortunately, three months later, the patient was admitted to a private psychiatric hospital in Lyon, France, for symptoms similar to those observed before receiving the previous ECT.

A diagnosis of intermittent major depressive episode considered as drug-resistant was reached and the patient underwent new ECT sessions. No brain imagery was repeated before ECT as a brain CT-scan had been performed three months earlier (Figure 1a). A neurological examination did not establish any motor deficit.

ECT was performed under general anesthesia (0.5 mg Atropine, 50 mg propofol and 25 mg suxamethonium) using standard brief pulse ECT machine with bifrontotemporal electrode placement and delivery of brief pulse waveform electrical stimulus of 176 mC (for the first session), 252 mC (for the second), 302 mC (for the third), and 327 mC (for the fourth), at 0.5 MHz frequency, resulting in an adequate motor seizure duration between 20 and 30 seconds.

Aside from a high systolic blood pressure (190 mmHg), which normalized spontaneously, during the second session, no clinical abnormality was found until the fourth session. Two days after this last session, a left hemiparesis was observed and the Glasgow Coma Scale (GCS) score was fluctuant (lowest GCS was E1V2M4). An emergency brain CT scan was performed (Figures 1b and 1c) and showed a right chronic SDH with acute bleeding.

Within the three days following the SDH evacuation by single burr hole and drainage, left lower limb motor function fully recovered and left upper limb paresis improved dramatically. Disorders of consciousness totally disappeared. Three months after surgery, neurological examination and brain CT scan were normal. A psychomotor retardation associated with major anxious symptomatology and sad mood were still observed. The ECTs were discontinued by the psychiatrist who did not modify the antidepressant treatment and initiated psychotherapy. Retrospectively, the repetition of ECT sessions ten days before the appearance of symptoms, probably explaining the chronic component of the SDH (based on the same principle as “shaken babies”).

## **Discussion**

SDH is a rare complication of ECT and only five cases (three men and two women, aged from 38 to 76 years) have been reported in the literature (Table 1). Two patients suffered from bipolar affective disorder (BAD) [1,2] and the three others presented PD [3,4]. With the exception of patient 4 [3], all cases had more than one session of ECT (4 to 12 sessions, mean 8) before SDH was diagnosed [3]. SDH diagnosis is therefore usually delayed with respect to the beginning of ECT. Consequently it could be either related to new-bleeding involving a pre-existent SDH or a bleeding occurring during one of the ECT sessions. In two cases [1,2], imaging before ECT was not available, and may thus have an undiagnosed SDH, and one had a known SDH [4]. Patient 1, who was medically treated, died [4]; all other cases were successfully treated by neurosurgical drainage.

ECT is the reference treatment for drug-resistant *major depression and its indications are clearly defined* [5]. According to a recent study [6], the death rate is 2.4 per 100.000 treatments for one day of ECT. This death rate increases to 18 per 100,000 within 14 days of ECT treatment but it includes all causes, regardless of the causal role of ECT. Thus, even if ECT is considered a safe treatment, many medical conditions can increase the complication rate and the benefit/risk ratio has thus to be discussed individually for each case [7]. ECT increases intracranial pressure as well as the cerebral blood volume [8] and can thus lead to cerebral herniation in cases of pre-existing mass effect [9] or to intracranial bleeding in cases of pre-existing hematoma [7]. The present report tends to confirm such a risk and underlines the importance of an immediate pre-ECT brain imaging, especially in patients with cerebral atrophy, taking hemostatic medication or with a past history of SDH. SDH is a frequent neurosurgical pathology associated with a high rate of recurrence after surgical treatment [10]. Population aging will probably increase the incidence of ECT indications in patients with a history of SDH. Moreover, in the elderly, in whom SDH

remains frequently asymptomatic during long periods of time, the SDH rate associated to ECT may be underestimated. There are very few published articles that report ECT after SDH treatment [4,11], and it is therefore not possible to define the risk of recurrence or potential predictive factors of recurrence in patients who have a previous history of SDH and require ECT. Moreover, the rate of patients in whom ECT was performed and did not have any SDH recurrence remains unknown. When ECT is the last therapeutic option, an intracranial bleeding history may thus not be considered as an absolute contraindication, but a systematic neurosurgical follow-up, including brain imaging (CT-scan), may be considered in addition to the psychiatric supervision.

### **Conclusion**

- A previous history of SDH may not be an absolute contraindication to ECT but a clinic-radiological neurosurgical follow-up is recommended during the treatment. In this case, a brain imaging (CT-scan) should be performed (at least in the two weeks) before ECT.
- The appearance of delirium, focal neurological deficit or consciousness disorders during ECT must immediately suspend the procedure until a new brain CT-scan is performed.
- ECT in patients with hemostatic drugs and patients with cortical atrophy should be extremely careful and cautious, because principle of “shaken baby” syndrome is applied.

The patient gave her informed and signed consent for the writing and publication of this article.

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**Figure 1:** a. Brain CT scan was performed three months before ECT. b.& c. Right chronic subdural hematoma associated with an acute bleeding. The brain shift was about 10 mm.

**Abbreviations:** CT-scan: Computed Tomography scan; ECT: Electroconvulsive therapy; MRI: Magnetic Resonance Imaging; PD: persistent depression; SDH: subdural hematoma.



Table 1 : Summary of cases

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5
Sex	Male	Male	Female	Male	Female
Age (years)	76	63	38	42	64
Psychiatric diagnosis	PD	BAD	BAD	PD	PD
Anticoagulant / antiplatelet drug	-	-	-	No	No
Number of ECT sessions	6	10	12	1	4
Neuroimager y before ECT	Left Chronic SDH *	*	*	No cortical atrophy	Cortical atrophy
Neurologic symptoms - GCS	Reduced consciousness	GSC12 E3V3M6	Right hemiparesis	GCS8 E2V4M2	GCS7 E1V2M4
Neuroimager y after ECT	Left Chronic SDH *	Left Chronic SDH	Left Chronic SDH	Bilateral Acute SDH	Right Chronic SDH
Neuroimager y: brain shift	No *	Yes	Yes	Yes	Yes
Treatment of SDH	Medical	Surgery: burr-hole	Surgery	Surgery: burr-hole	Surgery: burr-hole
References	Wijeratne et al. 1999	Awasthy et al. 2005	Saha et al. 2012	Kulkarni et al. 2012	Our case

PD: Persistent Depression; BAD: Bipolar Affective Disorder; ECT: Electroconvulsive Therapy; SDH: Subdural Hematoma; GCS: Glasgow Coma Scale

- : no information about bleeding drugs was published

\* : no imager y was published