



## Benign Paroxysmal Positional Vertigo Following Electroconvulsive Therapy

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### ABSTRACT

Patients with severe major depression most commonly undergo electroconvulsive therapy (ECT). Although this procedure is usually safe and the complication rates are very low, some side-effects and medical complications are observed. The reported adverse effects are generally minor in severity, and vertigo is one of these complications. We presented a case of benign paroxysmal positional vertigo (BPPV) occurring after ECT and aimed to discuss the pathophysiological mechanism of this condition. To the best of our knowledge, there are no other case reports regarding the co-occurrence.

**Keywords:** Electroconvulsive therapy, complication, benign paroxysmal positional vertigo

### INTRODUCTION

Electroconvulsive therapy (ECT) is a medical treatment most commonly used in patients with severe major depression, schizophrenia, or bipolar disorder who are unresponsive to other types of treatments. In the United States, the numbers of these treatments exceed those of coronary bypass, hernia repair, and appendectomy (1). The complication rate of this therapy is very low and frequently involves minor complications.

Benign paroxysmal positional vertigo (BPPV), which causes peripheral vestibular dysfunction, is the most common type of vertigo. It was described first time by Barany in 1921 (2). BPPV is typically characterized by brief attacks of positioning rotatory vertigo with nystagmus, which is evoked by changes in the head position (3). The etiology may be idiopathic (50–70% of all cases) or secondary to infections, migraine, Meniere's disease, extended travel, otologic, non-otologic surgery, prolonged bed rest, trauma, magnetic resonance imaging, vascular and metabolic pathologies, and hormonal changes (4).

We present a case of BPPV that occurred following ECT and discuss the pathophysiological mechanism of this condition.

### CASE REPORT

A 58-year-old woman was referred to the otolaryngology department with a complaint of vertigo lasting 2 days. She reported that her complaints had begun following an ECT treatment for major depressive disorder. It was noted that her disease did not improve with various medical treatments. After the thirteenth ECT, during the awakening from anesthesia, the patient experienced intense vertigo with nausea, which was provoked particularly with head rotation. She was referred to the emergency service. Dimenhydrinate (Dramamine; Ali Raif İlaç San. A.Ş., Istanbul, Turkey) was administered in the emergency department. Computerized brain tomography (CT) imaging was performed for excluding intracranial hemorrhage. The CT was within normal limits. When she presented to our department, there was no disease that could affect her balance. No additional otological problems, including hearing loss, ear fullness, or pressure sense, were reported. There was no history of systemic comorbid diseases or neurotologic pathologies including migraine. Her otorhinolaryngological examination revealed no significant finding. Her neurologic examination and laboratory analyses were within normal limits. An audiotympanometric evaluation was within normal limits. We performed the positioning nystagmus test (Dix–Hallpike test) for the diagnosis to BPPV. During the right swing, the patient had severe vertigo and a torsional right-sided, up-beating nystagmus. Nystagmus started after approximately 3–5 seconds and lasted up to 20–30 seconds. An Epley maneuver was performed on the right side for canalith repositioning treatment. In this maneuver, the patient was seated with the operator behind. The head was placed over the end of the table and turned to the affected ear at 45 degrees. While the head was tilted downward, it was rotated 45 degrees to the unaffected ear. The head and body were rotated until they faced downward at 135 degrees from a supine position. While the head was turned to the unaffected side, the patient was brought to a sitting position. The head was turned forward, with the chin down to

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20 degrees. The patient was advised to sleep in a slightly elevated position and avoid sudden head movements or rest on the affected ear for 3–5 days. Neither medication nor cervical collar was used. After 3 days of the repositioning maneuver, the patient was asked to visit for a control. There were no complaints of vertigo, and the Dix–Hallpike test was negative.

Written informed consent was obtained from the patient participating in this case study.

## DISCUSSION

ECT is generally a safe procedure. Hermida et al. (5) analyzed 766,180 ECT treatment cases from 32 studies involving different countries, and they found that the ECT-related mortality rate was 2.1/100,000. According to a study by Nuttall et al. (1), the complication rate was found 0.9% per patient and most of these were the occurrence of prolonged seizures, which are relatively benign.

The complications and adverse effects of ECT can be seen at the peri- or post-treatment period. The peri-treatment mortality of ECT is approximately 0.002%, mainly from cardiac arrhythmias, myocardial infarction, aspiration, laryngospasm, or sepsis (5). The risks and side effects after ECT treatment are confusion, memory loss, and physical side effect(s). Confusion can be seen immediately after treatment. Patients cannot recollect events that occurred right before the treatment or in the weeks or months before the therapy. These memory issues usually improve within few months after the treatment ends in most cases. Some patients experience headache, nausea, jaw ache, or muscle pain on the days of the ECT. These side effects can be improved with medications (1, 5). To the best of our knowledge, BPPV as a side-effect of ECT has not been previously reported in English literature.

There are two possible pathophysiologic mechanisms that lead to BPPV: canalolithiasis or cupulolithiasis (6, 7). Canalolithiasis is the most commonly accepted mechanism, wherein the otoliths that detach from the utricle or saccule and floating freely in the endolymph system of the semicircular canals cause neuronal stimulation. They are replaced through changing head positions. Parnes and McClure (6) in 1992 demonstrated this phenomenon in vivo. The pathophysiology of the cupulolithiasis was described by Schuknecht (7), wherein particles detached from the otoconial membrane are accumulated in the cupula of the semicircular canal. Until the detached otoconial particles enter the semicircular canal, they are floating freely in the utricle (7).

The possible pathophysiology of developing BPPV after ECT is not known clearly. We believed that it could be similar to the mechanisms mentioned in BPPV cases following maxilla–facial and dental surgeries. Drill- or hammer-induced vibrations on the maxilla are propagated throughout the bony structures, finally reaching the posterior labyrinth. In this situation, the mechanical energy would travel through the endolymphatic fluids. The membranous structures of the inner ear, which are located in the bony chambers, are particularly vulnerable to the traumatic effects of the transmitted mechanical waves. The vibrations may dislodge otoliths, which then enter the canal and cause BPPV (8, 9).

According to stimulus parameters, there are two ECT pulse widths: brief (0.5–2.0 millisecond) and ultrabrief (0.25–0.3 millisecond). It

is known that the stimulus dosage is affected for cognitive side-effects (5). The possible pathophysiology of developing BPPV after ECT is related to the electrical stimulation, which may be sufficient to detach the otoconia into the labyrinth and cause canalolithiasis. In our patient, bitemporal brief pulse ECT was applied.

The electrode placement varies markedly among clinics (10). In the current ECT practice, there are three commonly used electrode placements: bitemporal, bifrontal, and right unilateral (5). The principle of maximizing the efficacy while minimizing the cognitive side effect is based on the decision of the location of the electrode placement. Most studies compared the differences between bitemporal and unilateral electrode placement. The long term consensus is that bitemporal placement provokes side-effects that are more cognitive, while unilateral electrode placement is less effective, with fewer remissions but high relapse rates (10). In our patient, bitemporal electrode placement was used in ECT. We thought that electrode placement on the temporal region dislodged otoliths, which caused vertigo and nausea. However, the etiology of developing BPPV following ECT remains obscure, making this case more featured.

ECT is often a treatment required in severe persistent psychiatric disorders. An awareness of the morbidity associated with the procedure is essential. Although ECT is generally safe, it possesses some risks and side-effects. Patients should not experience vertigo and nausea after ECT; thus, both psychiatrists and otorhinolaryngologists must consider that a BPPV attack may be the etiology.

**Informed Consent:** Written informed consent was obtained from patient who participated in the case study.

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**Conflict of Interest:** The author has no conflict of interest to declare.

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