

Peri-lead idiopathic delayed-onset bilateral edema after implantation of deep brain stimulation in a GBA mutation carrier: a case report

*Pietro Antenucci*¹, A. Gozzi¹, F. Morgante², C. Sorbera³, M.A. Cavallo⁴, M. Sensi⁵

¹Department of Neurosciences and Rehabilitation, University of Ferrara, Cona, Ferrara, Italy

²Motor Control and Movement Disorders Group, Institute of Molecular and Clinical Sciences, St George's University of London, London, UK

³Neurorehabilitation Unit IRCCS Centro Neurolesi "Bonino Pulejo," Messina, Italy

⁴Neurosurgery Unit, Ferrara University Hospital, Department of Morphology Surgery and Experimental Medicine, Ferrara, Italy

⁵Department of Neuroscience and Rehabilitation, Azienda Ospedaliero-Universitaria S. Anna, Ferrara, Italy

Peri-lead idiopathic delayed-onset edema (IDE) is a self-limiting edema along a single intracranial lead, occurring ≥ 72 h after surgery in the absence of trauma that could rarely complicated deep brain stimulation (DBS) implantation for Parkinson's disease (PD) treatment [1]. No risk factors for IDE predisposition have been identified and switching-off the stimulation has been proposed as the safest managing options for IDE [1-2]. We report the case of a 39-year-old male carrying a GBA genetic mutation (G202R) with an early onset of tremor dominant PD who underwent the DBS intervention and a few days later developed symptomatic and bilateral synchronous edema along the leads. Due to the presence of severe parkinsonism, stimulation was switched on the 16th day after implantation once measured normal impedance values even though the edema was still present, with a satisfactory improvement on motor symptoms. According to the literature, G202R mutations have been associated with a subtype of Gaucher Disease [3] characterized by nonimmune hydrops with abnormal nonimmune interstitial fluid collections [4]. Regarding the absolute rare incidence of bilateral and synchronous IDE, we assumed a possible correlation between genotype variants of GBA mutation and the predisposition to the occurrence of IDE [5-6-7].

References:

- [1]. de Cuba, et al. Idiopathic delayed-onset edema surrounding deep brain stimulation leads: Insights from a case series and systematic literature review. (2016).
- [2]. Hooper, S. & Cameron, T. Neurotoxicity screening test for deep brain stimulation leads. *J Biomater Sci Polym Ed* 18, 1309–1320 (2007).
- [3]. Grace, M. E., et al. Identification and expression of acid beta-glucosidase mutations causing severe type 1 and neurologic type 2 Gaucher disease in non-Jewish patients (1997)
- [4]. Al - Kouatly, H. B. et al. Lysosomal storage disease spectrum in nonimmune hydrops fetalis: a retrospective case control study. *Prenat Diagn* 40, 738–745 (2020).
- [5]. Giraldo, P. & Andrade-Campos, M. Novel Management and Screening Approaches for Haematological Complications of Gaucher's Disease. *J Blood Med* Volume 12, 1045–1056 (2021).
- [6]. Vitner, E. B., et al. Contribution of brain inflammation to neuronal cell death in neuronopathic forms of Gaucher's disease. (2012)
- [7]. Pal, G. et al. Parkinson Disease and Subthalamic Nucleus Deep Brain Stimulation: Cognitive Effects in GBA Mutation Carriers. (2022).