

Abnormal Movements Secondary to Anoxic Brain Injury – Namdi Nwasike, MD and Shanthi P. Lewis, MD

1.) Case Report

An elderly female presented to the ED unresponsive after cardiac arrest. This was caused by long QT with Torsades or pause dependent Torsades, thought to be induced by Quetiapine. She was initially intubated and on a ventilator. She was seen by neurology, had CT-H and brain MRIs done, which revealed hyperintensities in the bilateral thalami, caudate nucleus, and dentate nucleus. She was diagnosed with anoxic brain injury. After being extubated, she was non-verbal and exhibited chorea-like movements, likely due to anoxic brain injury, although Tardive Dyskinesia was considered as the patient scored 21 out of 28 on the AIMS. The ICU team managed her movements with Dexmedetomidine. Psychiatry was consulted to manage medications, and Valproate was started in lieu of Quetiapine.

The patient's cognition slowly improved over the course of 2 weeks, and eventually she was able to speak in whispers. One day she expressed suicidal ideation, without intent or plan, because she believed her health was not improving. She developed short-term memory loss and couldn't remember when her family visited her, thus she felt isolated. A suicide risk assessment was performed, using the CAIPS format. Patient was deemed to not require a 1:1 for SI, given that she did not remember making those statements, and continuously denied SI thereafter. Psychiatry was reconsulted to do a capacity assessment regarding medical decision-making for a surgery, and patient was deemed to lack capacity. Her family agreed on the operation. She was eventually discharged to inpatient rehab after a month-long hospitalization.

2.) Outcome

Patient's chorea-like movements significantly lessened, and she regained her ability to speak. However, she retained deficits in attention/concentration, delayed recall, and short-term memory by the time she was discharged.

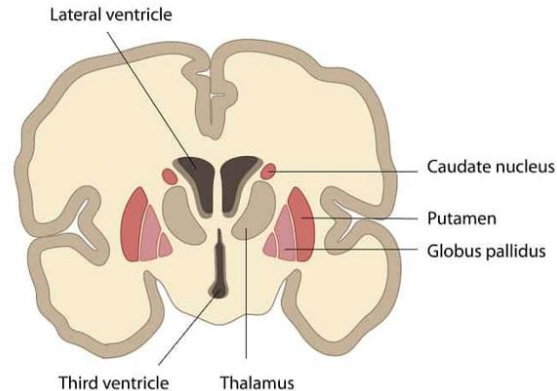


Figure 1. The caudate nucleus and the thalamus.) Hunt, Will, and Yuri Sugano. "The Basal Ganglia - Direct - Indirect - Nuclei- TeachMeAnatomy." *Teachmeanatomy.info*, 2018, teachmeanatomy.info/neuroanatomy/structures/basal-ganglia/. Accessed 26 Oct. 2023.

4.) References

- 1.) "How to Write a Suicide Risk Assessment That's Clinically Sound and Legally Defensible." *Www.mdedge.com*, www.mdedge.com/psychiatry/article/97395/depression/how-write-suicide-risk-assessment-thats-clinically-sound-and-
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- 5.) Ziad Ali, Autumn Roque, Rif S. El-Mallakh, A unifying theory for the pathoetiologic mechanism of tardive dyskinesia, *Medical Hypotheses*, Volume 140, 2020, 109682, ISSN 0306-9877, <https://doi.org/10.1016/j.mehy.2020.109682>. (<https://www.sciencedirect.com/science/article/pii/S0306987719314501>)

3.) Discussion

The caudate nucleus is involved with the execution of movement, learning, and memory. The dentate nucleus is involved with fine control of voluntary movements, cognition, language, and sensory functions. Given that the patient had hyperintensities in both nuclei, we hypothesize that the damage here was responsible for her deficits in speech, memory, and her chorea-like movements. Her movements resembled Tardive Dyskinesia (TD), which is characterized by facial/oral movements (such as grimacing, tongue probing), extremity movements (such as choreic or athetoid movements), and trunk movements (such as rocking or twisting.)

The AIMS is a 12-item tool that assesses TD. We were initially suspecting TD, and our patient scored 21 out of 28 on the AIMS. However, given the patient's medication history, anoxic brain injury was more likely to be the cause of her movements. The CAIPS format was chosen for suicide risk assessment due to its thoroughness, as it involves looking at chronic risk factors, acute risk factors, imminent warning signs, protective factors, and writing a summary statement.