Direct Effect of Amantadine on Aphasia during Inpatient Rehabilitation after Left Middle Cerebral Artery Aneurysmal Subarachnoid Hemorrhage

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Case Diagnosis

Fluent Aphasia secondary to Left Middle Cerebral Artery Aneurysmal Subarachnoid Hemorrhage

Case Description

A 61-year-old female with no past medical history presented with slurred speech and altered mental status to a gas station on her way to work. She was found down and intubated by paramedics in the field. On CTA she was found to have left middle cerebral artery aneurysmal subarachnoid hemorrhage with left-to-right midline shift and severe edema. The patient underwent aneurysmal clipping, external ventricular drain placement, left frontotemporal hemicraniectomy and cranioplasty, tracheostomy and gastrostomy tube placement. Her hospital course was complicated by ventilator-associated pneumonia treated with vancomycin and an acute pulmonary embolism requiring inferior vena cava filter placement. On admission to the acute rehabilitation facility five weeks post-injury, the patient was nonverbal, not gesturing to answer questions, and not following commands. Amantadine 100mg daily was initiated to improve arousal and increased to 200mg twice daily over the next month. Amantadine was discontinued due to psychomotor agitation and speech subsequently declined as demonstrated by Western Aphasia Battery-Revised (WAB-R) scores. With CT head stable, amantadine was restarted at 100mg twice daily and aphasia rapidly improved over the following week. WAB-R Aphasia Quotient scores gradually improved on amantadine until discharge seven weeks later.

Background

Amantadine is a NMDA receptor antagonist and indirect dopamine agonist with antiviral properties. Amantadine has been shown to accelerate pace of functional recovery as measured by Disability Rating Scale scores during active inpatient rehabilitation in patients with post-traumatic disorders of consciousness.[1]

Methods

Aphasia was assessed with the WAB-R, an instrument with high internal consistency, test-retest reliability, and validity as well as the ability to determine the presence, type, and severity of aphasia.[2] All WAB-R assessments were performed by the same speech language pathologist.

Results

WAB Subtest	8 weeks	11 weeks	14 weeks	16 weeks	20 weeks
Yes/No	36/60	39/60	30/60	39/60	42/60
Repetition	4/100	8/100	-	44/100	50/100
Object Naming	0/60	36/60	8/60	41/60	36/60
Auditory Word Recognition	30/60	39/60	17/60	51/60	48/60
Sequential Commands	-	12/80	-	14/80	34/80
Sentence Completion	0/10	6/10	2/10	8/10	4/10
Responsive Speech	0/10	8/10	2/10	4/10	6/10

Fig. 1. WAB-R subtest scores at time intervals in weeks after the initial injury. The dash indicates tests that were not administered or recorded.

Discontinuation of amantadine at 13 weeks post-injury was followed by significant decline in WAB-R Aphasia Quotient scores with subtest scores demonstrated above (Fig. 1). Per SLP report, decreased object naming at week 14 (36/60 to 8/60) was associated with semantic paraphasia and no benefit from cueing.

Discussion

Evidence has shown amantadine can improve functional recovery in post-traumatic disorders of consciousness but its efficacy in treating in post-stroke aphasia is a promising area of study. This patient's rapid decline in speech after discontinuation of amantadine and rapid improvement after restarting amantadine demonstrates a direct effect of amantadine on aphasia, as captured by Western Aphasia Battery-Revised assessments performed by the same speech language pathologist. To our knowledge, this is the only case report demonstrating the aforementioned direct effect regarding object naming.

Conclusions

This case highlights the benefits of amantadine in treating post-stroke aphasia. Future studies should determine Amantadine's effects on fluent vs. nonfluent aphasia, ischemic vs. hemorrhagic stroke, proper dosing, and benefits of long-term use.

References

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2. Shewan CM, Kertesz A. Reliability and validity characteristics of the western aphasia battery(Wab). J Speech Hear Disord. 1980;45(3):308-324.

Acknowledgments

Poster produced with Faculty & Curriculum Support, Georgetown University School of Medicine