Case Report

Wernicke's aphasia as the primary presenting feature in a young stroke female with antiphospholipid syndrome – The first ever case report from India

ABSTRACT

Wernicke's aphasia is a language disorder characterized by fluent speech with impaired comprehension and is traditionally associated with damage to the left posterior superior temporal gyrus. This article aims to explore the unique aspects of Wernicke's aphasia in the context of stroke in young patients. To the best of our knowledge, this is the first case report of Wernicke's aphasia as the primary and solo presentation in a young stroke patient with antiphospholipid syndrome.

Keywords: Antiphospholipid syndrome, autoimmune disease, stroke, Wernicke's aphasia, young female

INTRODUCTION

Wernicke's aphasia, also known as receptive aphasia, is referred to a condition, in which there is trouble in understanding spoken and written language. In roughly 95% of right-handed people and 70% of left-handed people, the most frequent cause of occurrence is damage to the posterior superior temporal gyrus, a part of the dominant cerebral hemisphere (Brodmann's area 22).[1] Unlike Broca's aphasia, in Wernicke's aphasia, patients may exhibit fluent speech, along with proper grammar and normal sentence structure. However, they may also use extended, meaningless sentences, extra words, or even make up their own words. Neurological symptoms depend on the size and location of the lesion that include visual field deficits, trouble with calculation (acalculia), and writing (agraphia). In most patients, the root cause of this kind of aphasia is an embolic or ischemic stroke affecting the inferior division of the middle cerebral artery (MCA), supplying the temporal cortex.

CASE REPORT

A 29-year-old female visited our hospital with a chief complaint of sudden onset of difficulty in speaking and comprehending words, repetition of irrelevant words and sentences, difficulty in understanding and following orders or commands, and also reading and writing and restlessness for the last 6 h. There was no history of headache, loss of consciousness, nausea or vomiting, limb weakness or deviation angle of the mouth, vision disturbance, or transient ischemic attack (TIA). There was no history of fever, night sweats, loss of appetite, or weight loss. She had no history of hallucinations or abnormal behavior before and no record of any psychiatric illness.

On examination, there was no pallor, cyanosis, clubbing, pedal edema, lymphadenopathy, or thyroid swelling. Vitals were within the normal limits (pulse rate – 78/min and blood pressure – 124/80 mmHg). Central nervous system

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examination – the patient was conscious but irritable and agitated. Her speech was evaluated as spontaneous speech with good fluency (repetitive talks and neologism present) repetition – impaired, comprehension – impaired, naming – impaired, and reading and writing – not cooperative.

Pupils bilateral – 3 mm reactive, fundus – normal, extraocular muscle – full, no ptosis, no facial lag, other cranial nerves examination – normal, motor system: tone – normal, power 5/5 all 4 limbs, deep tendon reflexes – 2+, plantar – flexors, sensory – could not be assessed, no cerebellar signs, no meningeal signs/no neck stiffness, other systemic examination – normal.

There was no significant medical history except the history of spontaneous abortion (first trimester) 2 years back. Routine blood investigations and two-dimensional echo were normal. Vitamin B12 level was significantly low at 80 pg/ml corresponding to her purely vegetarian diet. Magnetic resonance imaging (MRI) brain was suggestive of acute infarct in the left temporoparietal region involving the inferior branches of the M2 segment of left MCA [Figure 1a and b]. However, MRI angiography and venogram were normal.

Considering the young age of the patient and history of spontaneous abortion, antinuclear antibody (ANA) blot and thrombophilia profile with antiphospholipid antibodies (APLAs) antibody were sent. Homocysteine levels were found to be >50. As she presented after 6 h of the onset of symptoms (beyond the window period), thrombolysis was not considered in management. Meanwhile, she was treated conservatively with anti-coagulants and anti-platelets and was given the support of speech therapy. The patient showed improvement and started recognizing, understanding, and partially obeyed commands. Reading and writing were significantly improved within 3 days of starting treatment. Workup for the previously sent autoimmune disorder revealed a high titer of anti-β2 glycoprotein I (β2GPI) IgG (79 U/mL) shown in Figure 2a and anti-cardiolipin IgG was negative (1.5 U/mL) antibodies, and a low titer of anti-β2GPI (2.3 U/mL); whereas antiplatelet antibodies, ANA, anti-double stranded-DNA, anti-mitochondrial antibodies, antineutrophil cytoplasmic antibodies, anti-liver-kidney microsomal antibodies, and anti-smooth muscle antibodies were negative. lupus anticoagulant (LAC) was also found to be positive [Figure 2b]. The ultrasound abdomen examination was normal. A cardiology workup was performed by an experienced cardiologist of our institute and was found to be within normal limits. The anticoagulation therapy was carried on with good response and then shifted to oral anticoagulation therapy. Anti-β2GPI IgG and LAC were repeated after 12 weeks and were found to be positive, hence confirming our diagnosis [Figure 3].

DISCUSSION

This case underscores the complexity of young-onset ischemic stroke. Ischemic stroke and TIAs are the most common neurologic complications in patients with APLA. Although the neurologic presentation of patients with antiphospholipid syndrome (APS) may vary, many patients have striking similarities, such as initial memory loss, aphasia, cognitive dysfunction with progressive cerebral deterioration, and even dementia. The identification of anti-β2GPI, IgG, and LAC helps in identifying the APS. APS is an autoimmune disorder recognized in its association with recurrent thrombotic events and pregnancy-related complications.[2] Elevated homocysteine levels are the risk factors for the hypercoagulability. The response to the anticoagulation

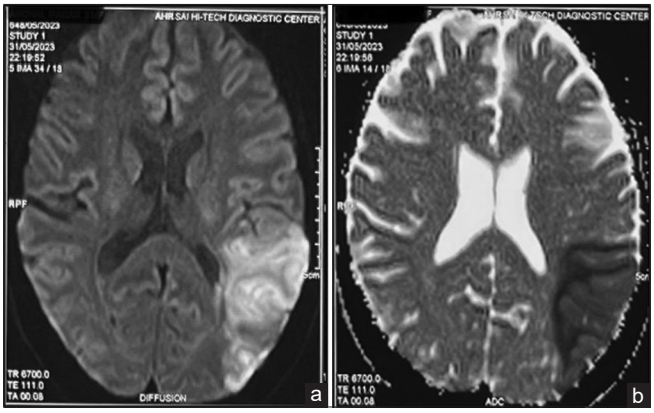
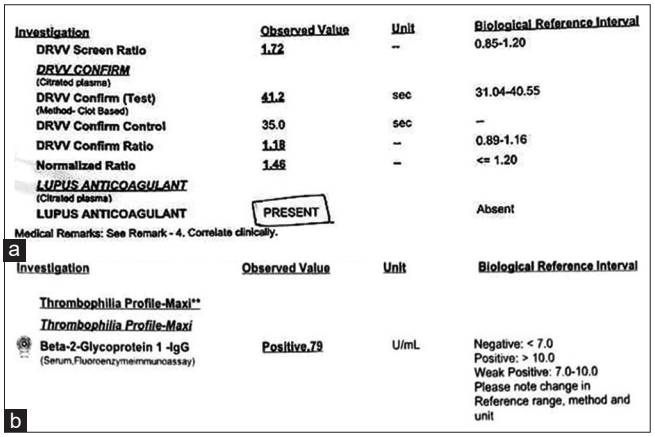


Figure 1: (a) Magnetic resonance imaging showing diffusion restriction in inferior division of M2 of left middle cerebral artery, (b) Apparent diffusion coefficient sequence confirms acute infarct in same area



Investigation	Observed Value	Unit	Biological Reference Interval
DRVV Screen Ratio	1.72	--	0.85-1.20
DRVV CONFIRM (Citrated plasma)			
DRVV Confirm (Test) (Method- Clot Based)	41.2	sec	31.04-40.55
DRVV Confirm Control	35.0	sec	--
DRVV Confirm Ratio	1.18	--	0.89-1.16
Normalized Ratio	1.46	--	<= 1.20
LUPUS ANTICOAGULANT (Citrated plasma)			
LUPUS ANTICOAGULANT	PRESENT		Absent

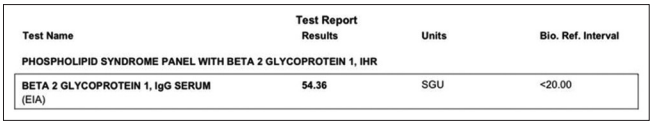
Medical Remarks: See Remark - 4. Correlate clinically.

a

Investigation	Observed Value	Unit	Biological Reference Interval
Thrombophilia Profile-Maxi**			
Thrombophilia Profile-Maxi			
Beta-2-Glycoprotein 1 -IgG (Serum,Fluoroenzymeimmunoassay)	Positive,79	U/mL	Negative: < 7.0 Positive: > 10.0 Weak Positive: 7.0-10.0 Please note change in Reference range, method and unit

b

Figure 2: (a) Lupus Anti-Coagulant Positive. (b) Beta 2 Glycoprotein 1 IgG Positive



Test Report

Test Name	Results	Units	Bio. Ref. Interval
PHOSPHOLIPID SYNDROME PANEL WITH BETA 2 GLYCOPROTEIN 1, IHR			
BETA 2 GLYCOPROTEIN 1, IgG SERUM (EIA)	54.36	SGU	<20.00

Figure 3: Follow-up investigations

therapy with the established management approach to APS helps to prevent recurrent thrombotic events.[3,4] Low Vitamin B12 levels might have been one of the causes for her neurological symptoms.[5]

According to several studies, in young (<45 years of age), only a handful of 20% of strokes are potentially associated with APS. A multitude of neurological problems such as convulsions/epilepsy, dementia, cognitive deficits, headaches/migraine, chorea, multiple sclerosis-like symptoms, transverse myelitis, ocular symptoms, Guillain–Barré syndrome, and even peripheral neuropathy can be presenting features of APS. Wernicke's aphasia is almost never the first presenting symptom in such cases. However, our timely diagnosis and prompt intervention allowing a multidisciplinary approach to incorporating anticoagulation therapy and speech therapy to the patient helped in significant improvement in a short time. In such cases, regular follow-ups are essential to adjust the management plan and to monitor the patient's response to the treatment. The long-term use of anticoagulation therapy aims at preventing recurrent thrombotic events in these patients.[6,7]

CONCLUSION

On the basis of medical history, clinical and laboratory features, and ruling out associated autoimmune diseases, a diagnosis of primary APS was made. To the best of our knowledge, this is the first case report of Wernicke's aphasia as the primary and solo presentation in a young stroke patient with APS in the Indian population.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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