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Anton syndrome due to bilateral posterior cerebral artery stroke with polyneuropathy in a patient with uncontrolled diabetes mellitus

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SUMMARY

Anton syndrome comprises of visual loss from cortical damage with patients not perceiving their own blindness in the absence of psychiatric illness or underlying cognitive impairment. Most commonly it results from bilateral posterior cerebral artery stroke.

In 1920, Meyer first reported occipital lobe infarction and postulated compression of branches of the posterior cerebral artery as the causal factor for Anton syndrome.

Joseph Babinski (1857-1932) used the term "anosognosia" for the first time, to describe the unawareness of the deficit in patients with hemiplegia.

We present an elderly man with uncontrolled diabetes mellitus who was admitted with symptoms of sudden blurring of vision and repeated fall but he denied having visual loss. On evaluation he was found to have bilateral posterior cerebral artery ischemic stroke with hemorrhagic conversion leading to cortical blindness as well as distal demyelinating motor sensory polyneuropathy.

Keywords: Anton syndrome; Hemiplegia; Motor sensory polyneuropathy.

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INTRODUCTION

An elderly diabetic and hypertensive male of age 61 was brought to our hospital by his relatives who declared that the patient was complaining of dizziness for previous 2 days and fell several times while moving but denied having any impairment of his vision [1-3].

CASE REPORT

On physical examination, blood pressure was 176/100 mm Hg.

On neurological examination, his higher functions were not significantly altered but his insight of illness was impaired so that he confabulated while asked about his visual impairment. He could not visually recognize any of his relatives. He also could recognize the examining doctor by his conversation. He was unaware of his blindness and was confabulating about his surroundings when asked about.

His speech was fluent with normal comprehension and repetition.

RESULTS

Cranial nerves: Absence of visual perceptions with normal pupillary light reaction.

Motor system: Decreased power in lower limbs with decreased knee and ankle jerks bilaterally.

Babinski sign+bilaterally

Sensory system examination revealed diminished vibration and position sensation in both lower limbs without any impairment of pain sensation.

His gait was high stepping but couldn't walk without support.

Investigations: Routine CBC wnl.

FBS, PPBS: 180 and 350 mg /dl.

Serum cholesterol was 230 mg/dl, LDL 175 mg/dl.

CT brain (**Fig. 1A**): Hypodense lesions in bilateral occipital cortices, with a small hyperdense area in right occipital cortex.

MRI brain (**Fig. 1-4.**): T1W hypointense signal in occipital cortices, diffusion restricted lesion in left occipital

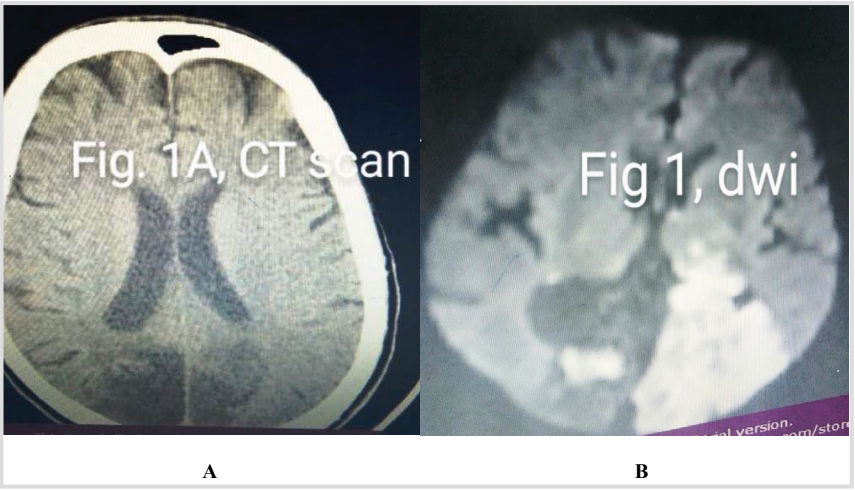


Fig. 1. A) CT scan of brain and B) a small hyperdense area in right occipital cortex.

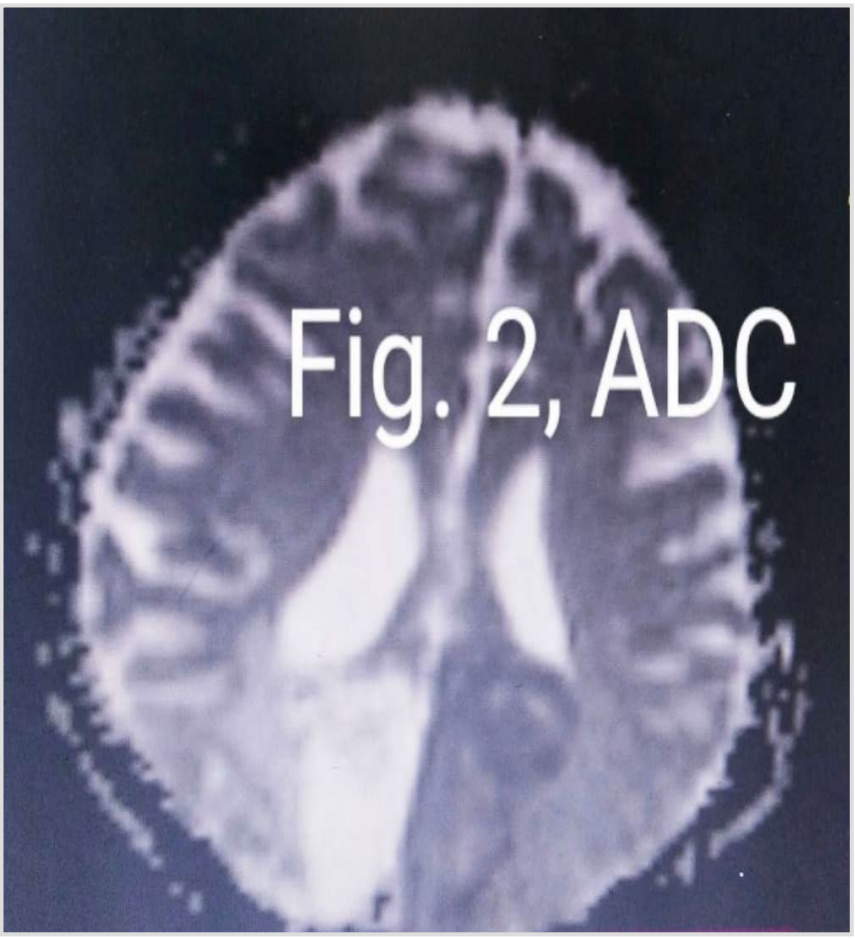


Fig. 2. Diffusion restricted lesion in left occipital cortex with corresponding ADC dark mapping.

cortex with corresponding ADC dark mapping. GRE blooming was present in right occipital cortex.

NCV (**Fig. 5A.**): Decreased CMAP amplitudes in bilateral peroneal and tibial nerves with delayed conduction velocities.

Sensory NCS (**Fig. 5B.**): Demyelinating sensory neuropathy of upper limbs and axonal degeneration in bilateral sural nerves.

VEP (**Fig. 5C.**): Absence of any potential.

Provisionally diagnosed as Anton syndrome with bilateral occipital lobe stroke (bilateral homonymous hemianopia) and visual anosognosia [4,5].

Treatment: Antihypertensives, inj insulin and oral hypoglycemics, physiotherapy and counseling. After about 10 days, he did not have confabulation and verbally admitted his visual loss.

DISCUSSION

Bilateral posterior cerebral artery stroke presenting with visual anosognosia with denial of blindness in this hypertensive, diabetic patient was diagnostic of Anton syndrome.

Fig. 3. GRE blooming was present in right occipital cortex.

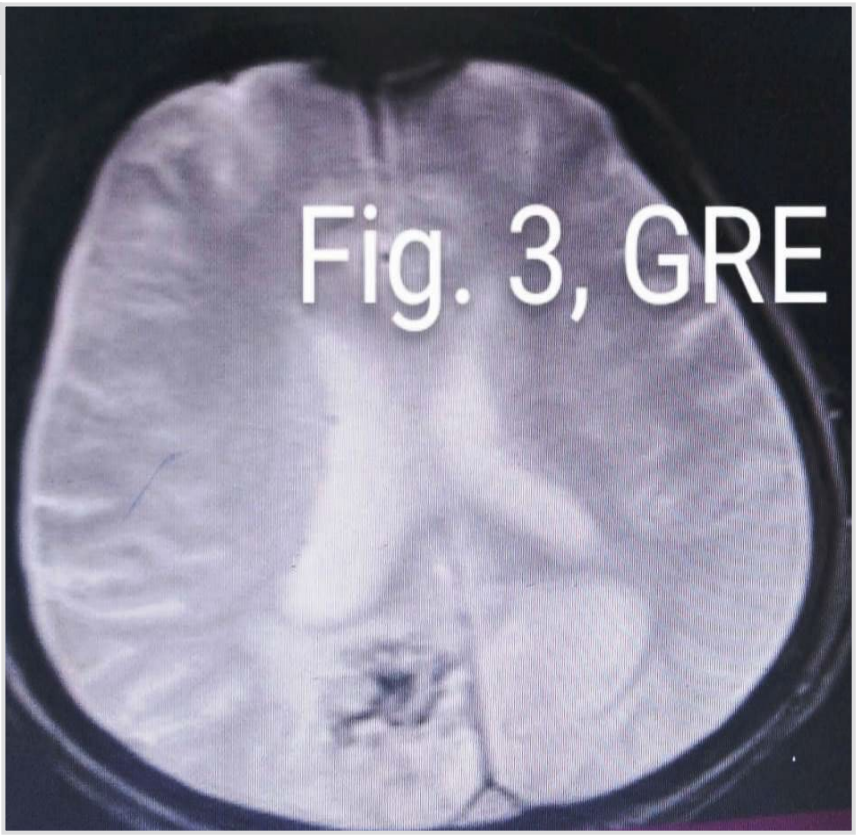


Fig. 4. T1W hypointense signal in occipital cortices, diffusion restricted lesion in left occipital cortex with corresponding ADC dark mapping.

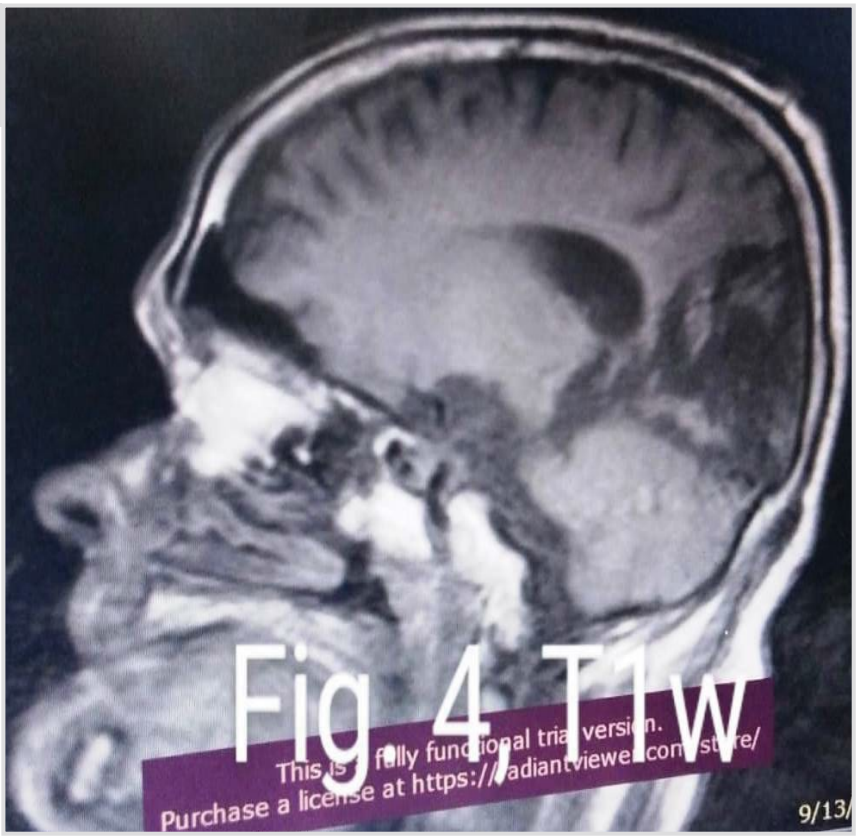
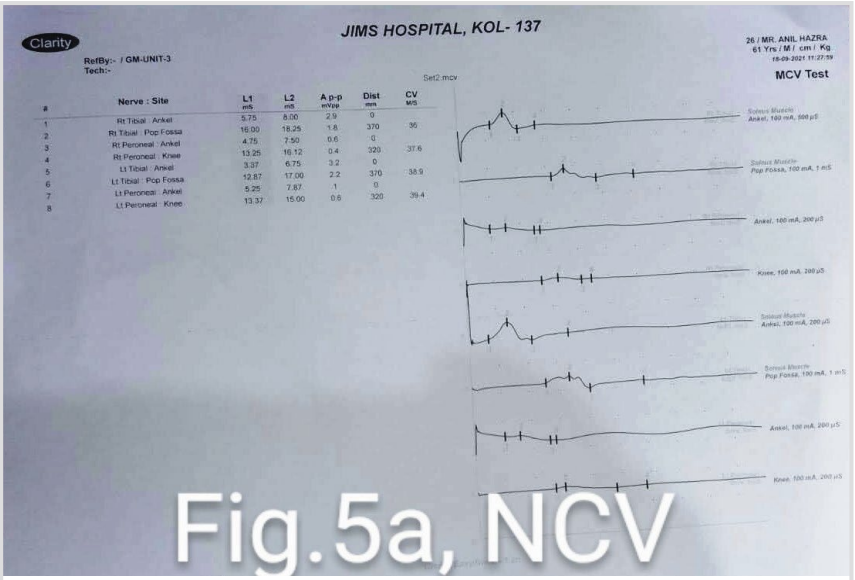
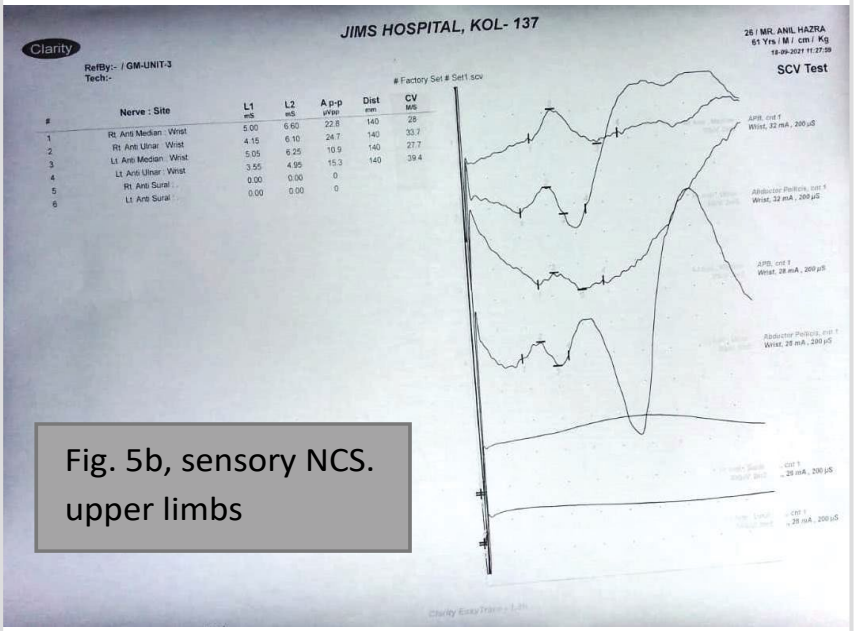
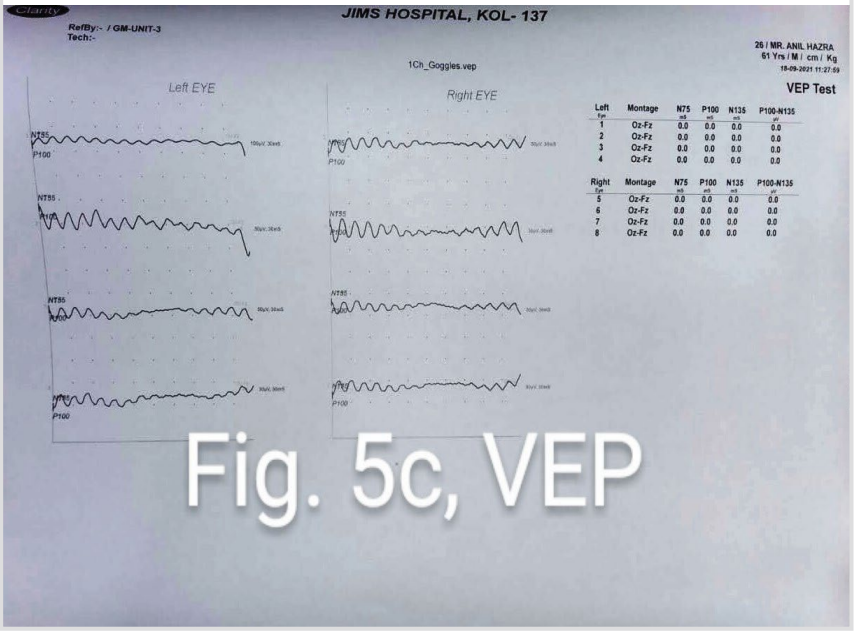


Fig. 5. A) Decreased CMAP amplitudes in bilateral peroneal and tibial nerves with delayed conduction velocities, **B)** demyelinating sensory neuropathy of upper limbs and axonal degeneration in bilateral sural nerves and **C)** absence of any potential.







He also had distal symmetric axonal polyneuropathy in lower limbs with some associated demyelination suggested by NCV study. This was most likely related to his uncontrolled diabetes mellitus for prolonged period.

CONCLUSION

Cerebrovascular disease is the most common cause of Anton's syndrome, and can be a potential complication of uncontrolled diabetes mellitus. Any patient with diabetic cerebrovascular stroke should also be evaluated for associated polyneuropathy.

CONFLICT OF INTEREST

The authors declare no competing interests.

ACKNOWLEDGMENTS

None.

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