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Brief Communication

Anton's Syndrome in Occipital Lobe Infarction

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ABSTRACT

Anton's syndrome implies the situation when affected patient contradicts blindness in spite of objective documentation of loss of vision, and often confabulate to assert their bearing. It is an infrequent sequel of cortical blindness affecting both occipital cortex and other cortical centers, while patients genuinely act as if they are sighted. Our case report is of a lady, 55 years old, who presented to us with history of uncontrolled diabetes, hypertension and visual loss as a consequence of occipital lobe infarction bilaterally. In patients with evidence of occipital lobe injury and atypical visual loss, an assessment for cortical blindness and Anton's syndrome must be included. Anton's syndrome is most frequently caused by cerebrovascular disease. Any condition that causes cortical blindness may, however, lead to Anton's syndrome. Improvement in visual function after occipital lobe infarction due to cerebrovascular events is limited. Therefore, the management should be focused on rehabilitation and secondaryprevention.

Keywords: Anton's syndrome, Occipital lobe infarct, Diabetes mellitus, Hypertension.

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INTRODUCTION

Anton's syndrome, also known as visual anosognosia, is defined as "negation of visual loss, accompanied by confabulation in the presence of evident loss of vision and cortical blindness".[1]in this condition, visual association centers of the occipital lobe is affected but the anterior visual tracts are intact. Patients affected with Anton's syndrome are confirmed that they are seeing what they actually cannot. They act as if they have vision.[1] However, awareness to the likelihood of the syndrome is drawn with accidents such as walking into walls, falling over furniture and describing things, which may not be present.[2]Wepresent a case of Anton's syndrome caused by un-controlled diabetes mellitus and hypertension, ultimately leading to occipital lobe infarct.

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CASE PRESENTATION

A 55-year old woman presented to Chaudhary Muhammad Akram Teaching and Research Hospital, Lahore with the complaint of decreased vision from last 2 days. She had history of uncontrolled diabetes and hypertension from last 4 and 3 years respectively. On presentation, her Glasgow coma scale was 15/15 with normal power in all groups of muscles of 4 limbs and down going plantars. There was peripheral neuropathy as well. On examination of eyes, her pupillary reflexes were preserved bilaterally but patient was not able to perceive light and threatened response. The most conspicuous clinical sign was marked limitation of visual acuity. According to her, she would walk into things as if plainly blind. In spite of an objective attenuation of her vision, the patient asserted she could 'see' objects surrounding her but with blackish haze. Pupillary reflexes were normal (highlighting an intact anterior visual pathway), along with fundoscopic findings of non-proliferative diabetic retinopathy and macular edema in both eyes. Her random blood sugar was 390mg/dl and blood pressure of was 160/90 mmHg.

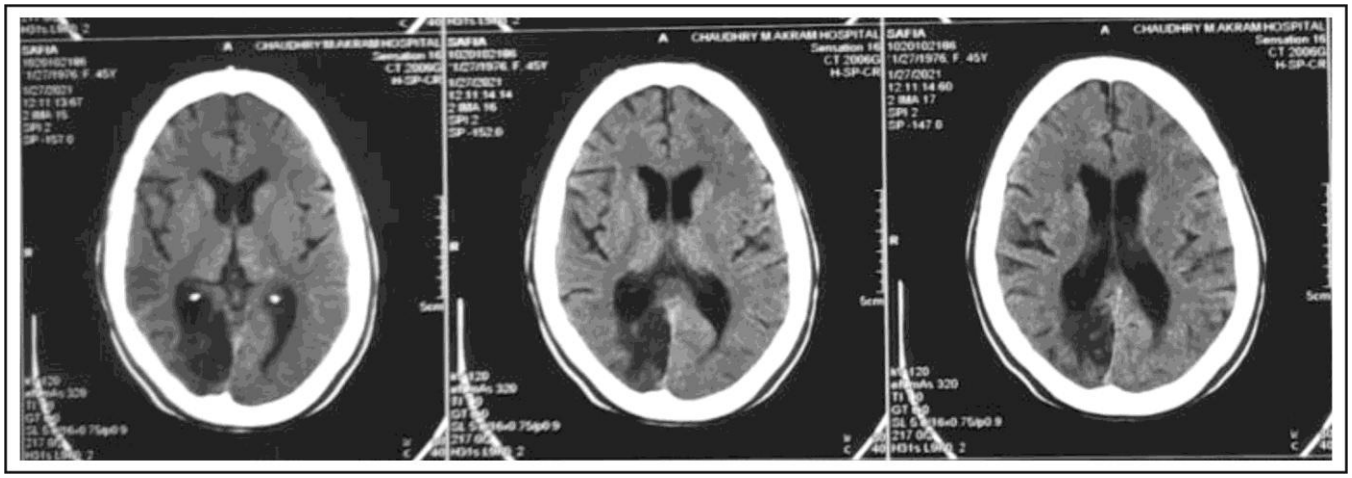

Figure 1: CT scan Brain of the patient showing hypo-dense area in the occipital lobe indicating infarction.

CT of brain (Figure 1) was performed which showed an old infarct of right occipital lobe and new evolving infarct of left occipital lobe. She needed assistance in moving safely due to her impaired vision, and needed assistance for daily routine chores. She would reach out for utensils on the tray and start eating if placed in front of her, however she was unable to see her meals, but help was needed to assist her avoid spillage and to finish the meals. During her stay in the hospital, her blood sugar was controlled and then patient was referred to ophthalmology department for treatment of non-proliferative diabetic retinopathy and macular edema. After the control of blood sugar her macular edema also reduced.

CONSENT

Consent was taken from the patient's attendants regarding sharing all the information.

DISCUSSION

Neurological impairment of vision encompasses a vast gamut of conditions including visual neglect and agnosia, cerebral visual impairment, homonymous hemianopia and numerous perceptual vision disorders, lack of facial recognition, delayed visual development and cortical blindness. Moving objects may nonetheless be perceived in total cortical blindness caused by damage to the occipital cortices bilaterally, whether consciously as in Riddoch's syndrome or unconsciously as in Blindsight.[2] Moreover, motion blindness is also described, where patients are able to see things but are unable to perceive their movements. This may be because of presence of projections from the lateral geniculate nucleus towards the visual cortex (V1) via optic radiations, directed to the middle temporal area.[3,4] Other demonstrations of visual impairment can compromise Charles Bonnet syndrome, when patients may experience elaborate hallucinations, with images of unfamiliar buildings or people although insight is preserved.[5] Anton's syndrome implies the situation when patients contradict their blindness in spite of objective documentation of loss of vision, and often confabulate to assert their bearing.[1] Often patients with damaged bilateral occipital lobes also have injury to the visual association cortex, that can lead to their deficit in awareness. Affected visual areas may be functionally disengaged from monitoring centers, such as speech and language centers, as suggested by Aston and these functioning speech areas may confabulate a response even in absence of input.[1] Another hypothesis suggests that the monitor of visual stimuli is dysfunctional and interprets images incorrectly.[4] The others advocate the presence of false feedback from another visual system. In this aspect, the superior colliculus, pulvinar and temporo-parietal regions can channel signals to the center if the geniculocalcarine system ebbs.[6] This internal imagery convinces the speech areas to make a response, in the absence of visual input.

Anton's syndrome is most frequently caused by cerebrovascular disease.[5] Any condition which may lead to cortical blindness can, however, possibly culminate in Anton's syndrome such as obstetric haemorrhage, hypertensive encephalopathy, pre-eclampsia, hypoperfusion, multiple sclerosis and brain trauma.[7,8] Our patient had occipital infarction bilaterally leading to cortical blindness and visual

anosognosia, and asserted her visual aptitude firmly in spite of an obvious visual impairment. Recovery in visual function is often good in diseases such as hypertensive encephalopathy and cortical hypoperfusion as resolution of the etiologic cause can result in correction of symptoms.[7]Our patient, having occipital lobe infarcts bilaterally, is less likely to gain a substantial improvement in spite of a partial recovery of her vision and therefore it is important to opt for secondary prevention, and guide her rehabilitation.

CONCLUSION

This case report accentuates to the scarce data available on Anton's syndrome. Our case report of a lady, 55 years old, who presented with history of uncontrolled diabetes, hypertension and visual loss as a consequence of occipital lobe infarction bilaterally due to Anton's Syndrome. In patients with evidence of occipital lobe injury and atypical visual loss, an assessment for cortical blindness and Anton's syndrome must be contemplated and investigated.

Conflict of Interest

Authors declared no conflict of interest.

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