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Anton's syndrome- the neurologic mystery of denial

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ABSTRACT

Anton's syndrome is a neurologic condition in which patients deny their blindness despite objective evidence of visual loss. It is a rare extension of cortical blindness in which, in addition to the injury to the occipital cortex, other cortical centres are also affected. Ironically the patients confabulate and stick to their stance that they are normal.

1. INTRODUCTION

Visual anosognosia, that is, denial of blindness, associated with confabulation in the setting of obvious visual loss and cortical blindness is known as Anton's syndrome. In Anton's syndrome the anterior visual tracts are intact, the visual association centres in the occipital cortex may be compromised. Patients with Anton's syndrome strongly believe they aren't blind and behave and talk as though they were normal. We describe a case of Anton's syndrome due to cerebrovascular accident.

2. CASE REPORT

A 65 year old male patient was admitted to department of medicine with complains of weakness of right side of the body since 1 day. Patient was a known diabetic and hypertensive and was on treatment for the same. Patient's Glasgow Coma Scale (GCS) was 15 out of 15 and he had normal power in all four limbs with no sensory loss

The most striking clinical feature on examination was severe impairment of visual acuity with the patient only having PL and PR on examination of vision.

Ocular movements and pupillary reflexes were intact suggesting that anterior visual pathways were not damaged. Fundoscopy was normal. The patient was not aware of the sight loss. In particular, the sight loss was observed for the first time when the patient asked for a window to be opened, even though it was already standing wide open. When asked about the position of the window, the patient pointed to the obviously wrong direction. Also when asked to describe the attending relative, the patient provided a completely wrong visual description. In addition, he was unable to reach examiner's extended hand. Despite this obvious blindness, the patient suffered a visual anosognosia, since he was unaware of his blindness and was confabulating about his surroundings when asked about it.

In addition to the above findings, on further examination, cerebellar signs were also positive.

MRA Brain showed Acute Infarct in Bilateral Cerebellum and Bilateral Occipital Region (posterior Circulation) with Chronic Lacunar Infarct in body of left caudate nucleus and left thalamus.

The above report along with clinical examination confirmed a diagnosis of Anton's Syndrome.

Patient was started on anti-stroke therapy and management for hypertension and diabetes was continued as before.

3. DISCUSSION

Anton's syndrome was first described in the fifteenth century and then was scientifically described by Austrian neuropsychiatrist Gabriel Anton (1858-1933). In Anton's syndrome, patients have objective blindness and deafness but show a lack of self-perception of their deficits. Joseph François Babinski (1857-1932) later used the term anosognosia to describe this phenomenon [1,2].

Neurological visual impairment encompasses a broad spectrum of conditions. These include conditions such as cerebral visual impairment, visual neglect, visual agnosia, various visual perceptual disorders, homonymous hemianopia, lack of facial recognition, delayed visual development and cortical blindness.

In patients with total cortical blindness secondary to bilateral damage to the occipital cortices, movement of objects may nonetheless be perceived, either consciously or unconsciously (blindsight) 3,4. This is due to the presence of projections from the lateral geniculate nucleus, both to the visual cortex (V1) via the optic radiations and to the motion-selective middle temporal area (MT or V5), a cortical area.

Anton's syndrome is the denial of loss of vision (visual anosognosia) associated with confabulation in the setting of obvious visual loss and cortical blindness. Frequently, patients with damage to the occipital lobes bilaterally also have damage to their visual association cortex, which may account for their lack of awareness [5]. Additionally, as suggested by Anton, damaged visual areas are effectively disconnected from functioning areas, such as speech-language areas. In the absence of input, functioning speech areas often confabulate a response [6].

Two other likely neuropsychological mechanisms have been postulated for Anton's syndrome. One suggests that the monitor of visual stimuli is defective and is incorrectly interpreting images. The other suggests the presence of false feedback from another visual system. Cerebrovascular disease is the most common cause. Some other causes are, hypertensive encephalopathy with pre-eclampsia, obstetric haemorrhage with hypoperfusion, and trauma. [7,8,9]

Our patient with bilateral occipital infarcts causing cortical blindness and visual anosognosia, fulfilled the classical description for Anton's syndrome. Good recovery of visual function has been noted in conditions causing Anton's syndrome such as hypertensive encephalopathy and cortical hypoperfusion. In these conditions, correction of the causative factor may lead to resolution of symptoms.

4. CONCLUSION

Cortical blindness and Anton's syndrome should be considered in patients with atypical visual loss and evidence of occipital lobe injury. Cerebrovascular disease is the most common cause of Anton's syndrome, as in our patient suggesting lesser chances of complete recovery. Management ultimately focuses on secondary prevention and rehabilitation.

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