

**A RARE CASE
COVID INDUCED BILATERAL
OCCIPITAL LOBE INFARCT –
ANTON SYNDROME**

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HISTORY

- o NAME : SHANKAR
- o AGE : 44
- o SEX : MALE
- o OCCUPATION : LABOURER
- o INFORMANT : WIFE (reliability good)

CHIEF COMPLAINTS

- o C/O fever for 1 week
- o C/o cough for 3 days

H/O PRESENTING COMPLAINTS

- o Presented with the complaints of fever for the past 1 week
 - sudden onset
 - continuous
 - mild grade
 - not associated with chills, rigor
 - not @ evening rise of temperature
 - not @ sweating
 - no diurnal variation

c/o cough for the past three days

- intermittent
- non productive
- not @ hemoptysis
- no diurnal variation

H/o headache +

h/o breathlessness on exertion for the past 3 days .

- o Given RT PCR throat swab for covid and reported as covid positive on 7/11/20 and admitted on 8/11/20
- o His wife noticed he was struggling to do his routine activities on 8/11/20 morning ,she asked him regarding any blurring of vision but he denied his loss of vision and told he was alright .
- o No h/o difficulty in seeing objects as history given by patient .
- o His wife noticed sometimes he was bumping into objects while walking
- o Patient able to move his eyeball in all direction

- o No h/o loss of smell
- o No h/o eye pain / double vision
- o No h/o dropping of eyelids
- o No h/o difficulty in chewing the food
- o No h/o difficulty in appreciating the sensation over the face
- o No h/o deviation of angle of mouth to right or left side

- o No h/o dryness or abnormal watering of eyes
- o No h/o drooling of saliva while drinking or eating
- o No h/o hard of hearing/tinnitus/vertigo
- o No h/o difficulty in swallowing the foods
- o No h/o choking sensation while eating
- o No h/o hoarseness of voice

- o No h/o difficulty in turning side to side
- o No h/o difficulty in protruding tongue

MOTOR SYSTEM :

- o No h/o weakness of both upper and lower limbs
- o No h/o flail ness or thinning of limbs
- o Able to hold objects and comb the hair
- o Able to hold slippers and walk upstairs

SENSORY SYSTEM :

- o Able to perceive cloth sensation

- o Able to perceive hot and cold like sensation while taking bath .
- o no h/o bowel and bladder disturbances
- o No h/o unsteadiness while standing
- o No h/o speech disturbances
- o No h/o involuntary movements
- o No h/o speech or memory disturbances
- o No h/o loss of consciousness

- o No h/o behavioural or emotional disturbances
- o No h/o sleep disturbance
- o No h/o vomiting
- o No h/o trauma

PAST HISTORY

- o Patient was recently diagnosed as type2 diabetes mellitus 3 months back not on medication
- o No other co morbid illness of CVA / CAD / RHD / TUBERCULOSIS / SEIZURE DISORDER
- o No other significant treatment history

Personal history

- o Normal bowel bladder habits
- o Chronic alcoholic
- o Non smoker
- o No substance abuse

SUMMARY

- o A 44 years old male patient who was diagnosed as covid 19 positive presented with the complaints of fever and cough ,wife noticed that patient had vision loss which was denied by the patient with no other cranial nerves , motor , sensory or autonomic involvement .

GENERAL EXAMINATION

- o Moderate built and nourished
- o Conscious
- o Oriented to time & place
- o No pallor/ icterus / cyanosis/
clubbing/lymphadenopathy/pedal edema
- o Jvp not elevated
- o No neurocutaneous /tuberculosis/ischemic
heart disease markers .

- o Ht -168 cms
- o Wt - 56 kgs
- o Bmi -19.85kg/m²
- o Vitals : BP - 120/80 mmhg ,measured in right arm of supine position . No postural variation in BP

- o Pulse rate – 82/min regular rhythm, normal volume ,no specific character, no radio femoral or radio radio delay
- o RR – 18cycles / min
- o Temp -98.4 degree celsius
- o Spo2 – 96 % in room air

EXAMINATION OF CNS

- o HIGHER MENTAL EXAMINATION :
- o Right handed person
- o Conscious
- o Oriented to time ,place & person
- o Memory – immediate, recent and remote intact
- o Language – comprehension, fluency and repetition intact
- o No emotional liability

CRANIAL NERVE EXAMINATION:

CRANIAL NERVE	RIGHT	LEFT
I) OLFACTORY	Able to perceive smell	Able to perceive smell
II) OPTIC NERVE		
1)visual acuity	Able to appreciate hand eye movement	Not able to appreciate
2)field of vision	absent	Absent
3)colour vision	Absent	Absent

CRANIAL NERVE	RIGHT	LEFT
4) Fundus		
Media	clear	Clear
disc / vein	Normal	Normal
macula (foveal reflex)	Present	Present

3, 4 & 6: 1)extra ocular movements	Full range	Full range
2)direct & indirect light reflex	Present	Present
3)Ptosis	No	No
4)accommodation reflex	Not able to perform	Not able to perform

TRIGEMINAL NERVE		
1)sensation over face & buccal mucosa	Present	Present
2)clenching of teeth	No deviation	No deviation
3)corneal & conjunctival reflex	Present	Present
4)jaw jerk	Absent	Absent

FACIAL NERVE

1)Taste in ant 2/3 of tongue

Present

Present

2) wrinkling of forehead

3)able to open both eyes against resistance

Yes

Yes

4)deviation of angle of mouth

No deviation

No deviation

5)blowing of cheeks & holding of air in mouth

able to do

able to do

7)corneal & conjunctival reflex

Present

Present

8)salivation & lacrimation

Present

Present

VESTIBULO-COCHLEAR NERVE		
1) Rinne's test (BC > AC)	AC > BC	Ac > BC
2) Weber's (lateralisation)	No lateralisation	No lateralisation
GLOSSOPHARYNGEAL & VAGUS:		
1) Sensation over post 1/3 of tongue	Present	Present
2) position of uvula	Midline	Midline
3) palatal & pharyngeal reflex	Present	Present
SPINAL ACCESSORY NERVE:		
1) Shrugging of shoulder against resistance	Able to do	Able to do
2) turning head against	Able to do	Able to do

**HYPOGLOSSAL
NERVE:**

1) Deviation of tongue
on protrusion

No deviation

No deviation

MOTOR SYSTEM

- o No Generalised /localised muscle wasting
- o BULK

	CIRCUMFERENCE	RIGHT (CM)	LEFT(CM)
UPPER LIMB	Mid arm	25	23.5
	Mid forearm	21.5	20.5
LOWER LIMB	Mid thigh	35.5	35.5
	Mid leg	27	27

	RIGHT	LEFT
Tone :		
UPPER LIMB	Normotonia	Normotonia
LOWER LIMB	Normotonia	Normotonia
Power :	5/5 in all joints of both upper and lower limb	5/5 in all joints of both upper and lower limb
Hand grip	100 %	100 %

REFLEX

SUPERFICIAL	RIGHT	LEFT
Corneal (5,7)	Present	Present
Conjunctival (5,7)	present	present
Pharyngeal (9,10)	Present	Present
Palatal(5,10)	Present	Present
Abdominal (T8-T12)	present	present
Cremastric (L1,L2)	present	present
Plantar(L5,S1	Withdrawal	withdrawal

DEEP	RIGHT	LEFT
Jaw jerk(C5)	absent	absent
Biceps(C5,C6)	Present	Present
Triceps(C6,C7)	Present	Present
Supinator(C5,C6)	Present	Present
Knee (L2,L3,L4)	Present	Present
Ankle(S1)	Present	Present

Finger flexor reflex , Hoffmann's reflex , Wartenberg's reflex: present

- o Sensory system :
- o Touch ,pain ,temperature - able to appreciate .
- o Pressure ,vibration and position sense – present
- o Tactile localisation ,two point discrimination streognosis and graphesthesia -intact

- o Bowel bladder : Normal bowel bladder function .

Cerebellum :

- o Finger nose test - intact
- o Finger finger nose test – intact
- o Dysdiadochokinesia - intact
- o Heel knee test - able to perform
- o Speech – normal

- o No dysmetria ,nystagmus
- o No intentional tremor
- o No pendular knee jerk
- o No titubation
- o No rebound phenomenon

MENINGES :

- o No neck rigidity
- o Kernig & brudzinski sign – negative

- o Romberg's test - negative
- o Gait - normal
- o Spine - no tenderness
- o Peripheral nerve - no thickening or tenderness

Other systems

- o CVS – S1 S2 heard , no murmur
- o RS – Bilateral air entry +
b/ l crepts +
- o PER ABDOMEN : Soft , no organomegaly

Provisional diagnosis

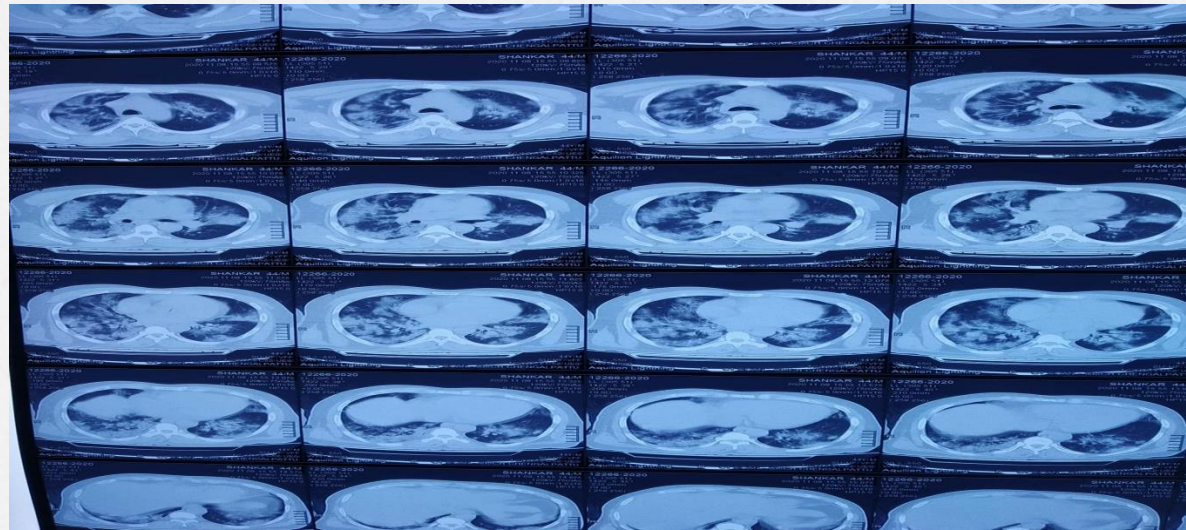
COVID 19 POSITIVE / VIRAL PENUMONIA /
ACUTE CVA WITH BILATERAL SUDDEN
PAINLESS LOSS OF VISION

DIFFERENTIAL DIAGNOSIS

- o BILATERAL OCCIPITAL LOBE LESION
- o CENTRAL RETINAL VEIN / ARTERY OCCLUSION
- o CENTRAL SEROUS RETINOPATHY
- o RETINAL DETACHMENT
- o VITREOUS OR RETINAL HEMORRHAGE
- o ANTON SYNDROME
- o CHARLES BONNET SYNDROME

INVESTIGATIONS

CT chest :bilateral ground glass opacities with 50 - 70 % lung involvement s/o covid pneumonia



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C.T. SCAN CENTRE

CT SCAN CHEST

Name: Shankar Age/Sex: 47/M Reg No. _____ Date: 9/11/2020

(without/with IV contrast)

CLINICAL HISTORY:

Serial Axial sections of the Thorax, following Volumetric Data Acquisition on Multi Slice CT from thoracic inlet to the level of the domes of diaphragm, were studied, along with Coronal and Sagittal Reformations, and show-

SCANOGRAM:

Trachea normally placed. Cardiac shadow is normal. Both hemi thoraces are of equal and normal volume. Lung zones appear normal. Bony thorax and Diaphragmatic domes appear normal.

MEDIASTINUM:

Trachea is normally placed and appears normal in caliber.
Both major bronchi appear normal.
Ascending, arch and descending thoracic aorta appear normal.
Main pulmonary artery and its branches appear normal.
The pericardium appears normal.
There is no evidence of mediastinal lymphadenopathy.

LUNG:

The lung parenchyma from the apex down to the base appears normal.
The parenchymal vascular structures show normal distribution and caliber.

PLEURAL SPACES:

Pleural cavity on RT/LT sides appear normal.

IMPRESSION:

Patchy Pneumonitis Ground glass
Opacities Seen in both lung fields.

Verified by _____

Typed by _____

[RADIOLOGIST]

Dr. E. ELAVARASU, DMRD

Reg No: 52536

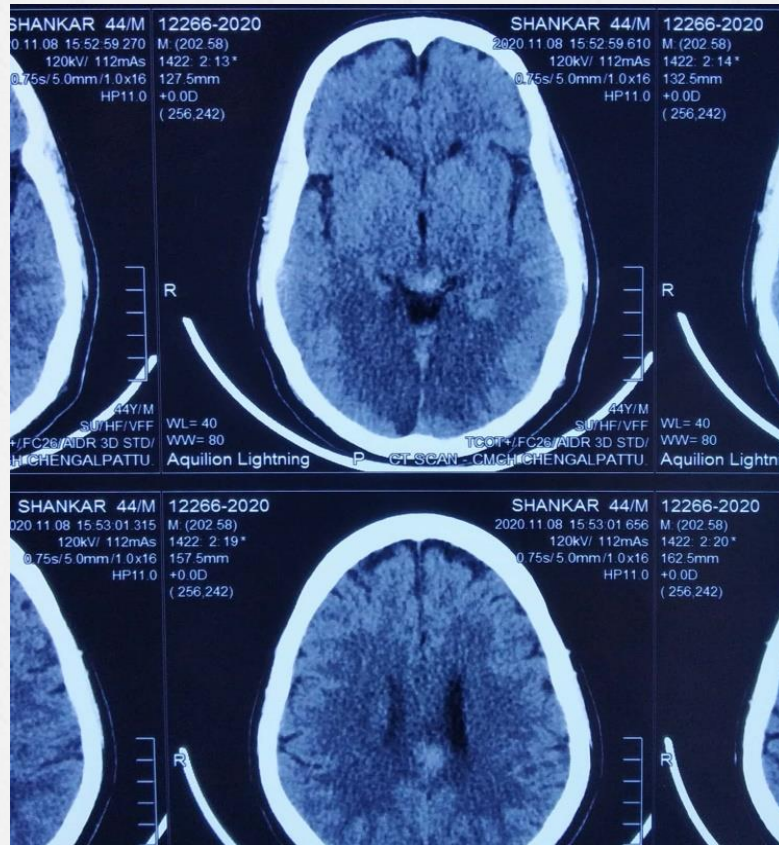
Radiologist (Senior Resident)

Chengalpattu Medical College and Hospital

Imp:-

COVID Pneumonia 70-75%.

CT brain -- bilateral posterior cerebral artery infarct (right - complete, left - partial)



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C.T. SCAN CENTRE

CT SCAN BRAIN

Name: Shankar Age/Sex: 49/M Reg No. _____ Date: 9/11/2020

Serial axial and sections of the brain from base of skull to vertex
(with/without IV contrast)

Eyeballs optic nerves, orbital fat extra ocular muscles appear normal.
Paranasal (ethmoid, sphenoid, frontal sinuses) appear normal.

POSTERIOR FOSSA:

Medulla oblongata, pons and midbrain appear normal.
Cerebellar hemisphere and vermis appear normal.

SELLA :

Sella and suprasellar regions appear normal.
Basal cisterns appear normal.
Ventricular System appears normal.

CEREBRUM :

Cerebral sulci and gyri appear normal.
External capsule, lentiform nucleus, internal capsule, caudate nucleus and thalamus appear normal.
Corona radiata and centrum semiovale appear normal.
No evidence of SOL. No evidence of hemorrhage / infarct.
No evidence of midline shift.

BONES :

Bones of skull appears normal.
Extra cranial soft tissues appear normal.

IMPRESSION :

No significant abnormalities detected in brain

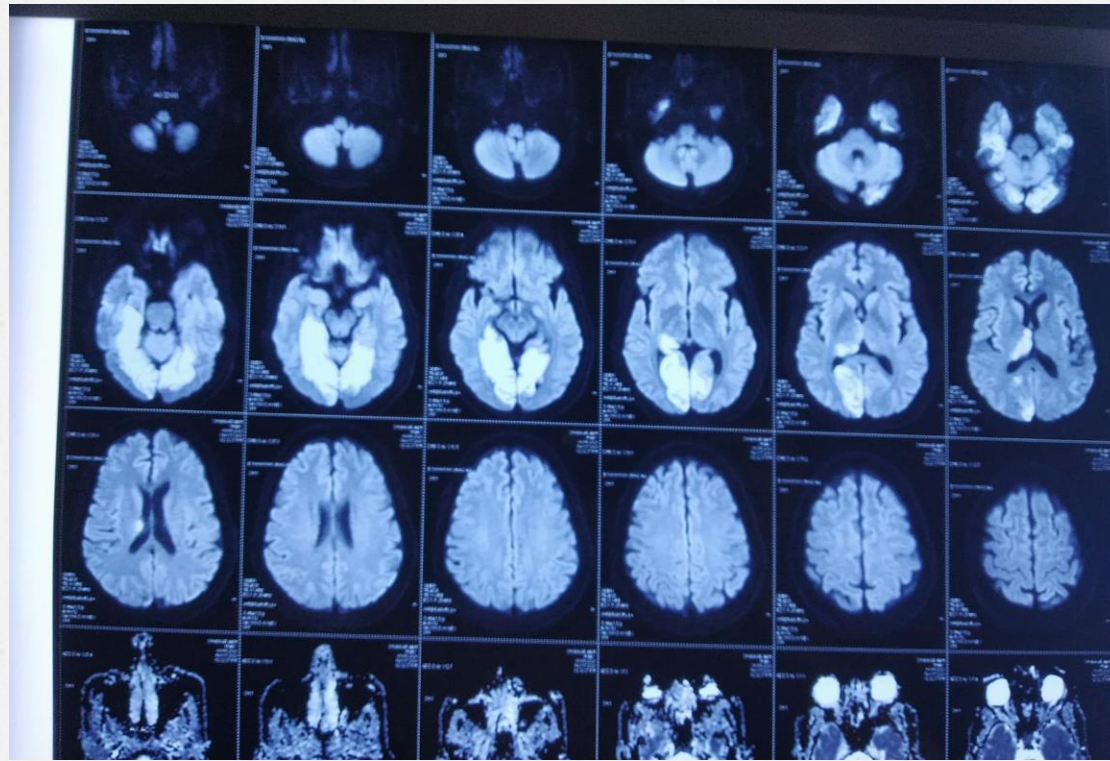
B/L PCA territory infarct

[Signature]
[RADIOLOGIST]

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MRI brain – acute infarct bilateral occipital region ,
acute infarct right thalamus





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MRI - SCAN CENTRE
CHENGALPATTU MEDICAL COLLEGE HOSPITAL
CHENGALPATTU

NAME : Mr. SHANKAR
REF.DR:

AGE & SEX : 44 / M
DATE : 17.11.2020

MR IMAGING OF BRAIN

TECHNICAL DATA

Without IV MR contrast media administration. Spin echo, Fast spin echo, Gradient echo and inversion recovery techniques.
SE T1W, FSE T2W GREW sagittal, T1WI, STIR, FSE T2W coronal, FSE T2W axial.

OBSERVATION

Acute infarct bilateral occipital region.

Acute infarct right thalamus.

Cerebellar hemispheres, vermis and peduncles are normal in morphology and signal. The medulla, pons and mid brain show normal MR features.

The basal (including CP angle), supra/para sellar and sylvian cisterns are normal. The fourth, third and the lateral ventricles are of normal size, shape and position.

The thalami, basal ganglia and internal capsules are normal on both sides. The pituitary gland and optic chiasm are normal.

The orbits and their contents appear normal.

MRA: Thinning of bilateral posterior cerebral artery. P7C

MRV: Normal Study.

Orbital Screening: Normal Study.

IMPRESSION:

- ❖ **Bilateral acute posterior cerebral artery infarct.**

Dr.
Dr. ANNALAKSHMI
Radiologist

Dr. R. ANNALAKSHMI, DMRD
Reg No: 67037
Radiologist (Senior Resident)
Chengalpattu Medical College and Hospital

Dr. KASIVISALAKSHI
HOD

- o D dimer – more than 10000 mg/ml
- o S.ferritin – 995.67mg/ml
- o CRP - 12
- o PT - 20.36
- o INR - 1.2
- o RBS -304
- o UREA – 25
- o CREATININE – 0.7

o ECHO :

NO RWMA

EF-60 %

NORMAL STUDY

PERIPHERAL SMEAR : normal study

S.HOMOCYSTEINE : 17micromol /litre

o CBC :

TC -10,600

Hb- 14.3

Plt -2.5 lakhs

Neutrophil - 80

Lymphocyte - 05

Nlr ratio of 16

Ophthal opinion

9/11/2020

S/B Asst. Ophthalm

Thank for referral
 H/o sudden bilateral loss of vision since last night
 H/o CRA/Ischemic stroke

O/E	OD	OS
Bestside vi sm	Hm+	Hm+
Conjunctiva	clear	clear
Cornea	clear	clear
Ac	nt	nt
Iris	ncp	ncp
pupit	3mm, round, sluggishly FR	3mm, round, sluggishly FR
lens	clear lens changes	clear lens changes
fund	free/full	free/full

fundus: (BE) Media clear
 Dr - Normal
 Macula FR of

To R/V MRI Reports
 2 MR

A - Defective vision due to stroke/
 CRA infarct

SC 9/11/20

Revised diagnosis

- o Covid 19 positive / viral pneumonia / Acute CVA / ischemic stroke / covid induced bilateral PCA infarct



- o ANTON SYNDROME

ANTON BABINSKI SYNDROME

- Visual anosognosia
- Rare neurological condition
- Syndrome was named after Anton and Babinski in 1914
- Hypoperfusion of occipital cortex is the usual cause of cortical blindness.
- Commonly results from ischaemic vascular injury but also from haemorrhage and other causes .

- o Cerebrovascular disease is the most common underlying aetiology
- o Damage to the visual association cortex (brodmann area 18 & 19) and the primary visual cortex (brodmann area 17) is responsible for cortical blindness.
- o Damaged visual areas are effectively disconnected from functioning areas such as speech and language areas.

- o In the absence of input ,functioning speech areas often confabulate a response
- o Recovery of visual function depends on the etiology and occur in some cases of hypertensive encephalopathy ,cerebral hypoperfusion .

CASE REPORT

Bilateral cortical blindness with Anton–Babinski syndrome in an elderly Nigerian woman: Challenges for tertiary prevention

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ABSTRACT

Anton–Babinski syndrome (Anton’s syndrome) is well described in the scientific literature even though it is a rare neurological condition. Most publications have highlighted the anatomy, neurophysiology, and pathology of visual anosognosia, which is the hallmark of the syndrome. We are not aware of any published report of cortical blindness with Anton’s syndrome from Africa. We report a catastrophic complication of severe hypertension in an elderly Nigerian woman who was on follow-up for stroke, chronic heart failure, diabetes mellitus, and glaucoma. She developed bilateral cortical blindness with Anton’s syndrome as a complication of severe hypertension, following 3 weeks of missed medications. This report highlights some challenges of tertiary prevention in this elderly woman without health insurance, who before becoming blind, had been largely dependent on family members’ goodwill for her medical care.

Keywords: Anton’s syndrome, cortical blindness, elderly, Nigeria, tertiary prevention

INTRODUCTION

Anton’s syndrome is bilateral cortical blindness with visual anosognosia and visual confabulation.^[1] The first published description of someone who was blind but denied it was that of a nobleman reported by Montaigne in the 16th century.^[2] Gabriel Anton’s description of the syndrome was published in 1899 with descriptions of three patients who had visual, hearing, and motor impairments, respectively, but who denied these deficits.^[1–3] Joseph François Babinski in 1914 named this phenomenon of blind hemiplegic patients who were unaware of their blindness. “anosognosia.”^[4] Although the syndrome has been

named after Anton and Babinski, it had earlier been described by Wernicke in 1874 and by Westphal in 1882.^[5]

Clinical diagnosis of Anton’s syndrome is based on five criteria: (i) failure to blink in response to threat, (ii) loss of light and dark visual impulses, (iii) preservation of pupillary and accommodation reflexes, (iv) normal fundi, and (v) preserved extraocular movements. The absence of global cognitive deficits was central in Anton’s original description of the syndrome.^[4]

Hypoperfusion of the occipital cortex is the usual cause of cortical blindness. This commonly results from ischemic vascular injury^[6] but could also result from hemorrhage and various other causes, including advanced glaucoma.^[7] Cerebrovascular disease is the most common underlying etiology that is reported in association with Anton’s syndrome.^[2]

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Visual Anosognosia (Anton-Babinski Syndrome): Report of Two Cases Associated with Ischemic Cerebrovascular Disease

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ABSTRACT

Visual anosognosia or Anton-Babinski syndrome is a rare neurological condition related to cortical blindness. The patients deny their blindness and affirm adamantly that they are capable of seeing. The clinical presentation includes confabulations and sometimes confusional states. In this article we report two patients with anosognosia related to ischemic stroke in two different sets of etiology and pathogenesis. We describe the major clinical manifestations of this syndrome and review the current medical literature. Two patients were identified, a 96-year-old male with visual anosognosia secondary to a right posterior cerebral artery thrombosis, and a 56-year-old female with the same syndrome but related to central nervous system angiitis in relation with multiple sclerosis and Hashimoto's thyroiditis. Visual anosognosia or Anton-Babinski syndrome is a rare neurological condition. However the ischemic vascular cerebral disease is a frequent etiology. We believe that this is the first report of this syndrome in relation to angiitis with a clear autoimmune pathogenesis.

Keywords: Visual Anosognosia; Cortical Blindness; Anton-Babinski Syndrome; Stroke; MRI

1. Introduction

Cortical blindness matches several clinical criteria [1]: loss of all visual sensations, loss of menace reflex, preservation of pupillary reflexes, a normal fundoscopic examination and preservation of ocular movements. Visual anosognosia, or Anton-Babinski syndrome is a rare complication of cortical blindness, where the patients deny their visual deficit [2]. Damage of the visual association cortex has been thought as one of the main causes explaining the loss of awareness of the visual deficit [3], along with damage of other pathways connecting the visual cortex with the systems that process the information received from the senses. Ischemic cerebrovascular disease causing cortical blindness is the most common etiology of this syndrome [4].

We herein describe two cases and a review of the literature about the visual anosognosia.

*Corresponding author.

2. Case Presentation

2.1. Patient #1

A 96-year-old man was admitted to Emergency Room with severe headache and sudden loss of vision. He had a cutaneous carcinoma resected about three years ago, and he was currently on treatment for hypertension.

On admission he was awake and oriented. Blood pressure was 180/100, and he had a mild paresia on his left arm. Ophthalmologic exam confirmed a severe vision loss, ocular movements, as well as photo motor reflex, were preserved. Fundoscopic examination revealed changes secondary to chronic hypertension. The patient sustained that he was able to see, despite the objective evidence of vision loss.

The diffusion images on the brain MRI of the brain demonstrated ischemic areas on the occipital lobes, specially on the right side (**Figure 1**). A brain CT angiography showed a stenotic segment on the posterior right cerebral artery (**Figure 2**).

THANK YOU



Anton-Babinski syndrome

Anton's syndrome (cortical blindness plus denial of blindness)