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Case Report

# Bilateral Occipital Infarction Presenting as Anton-Babinski Syndrome and Charles Bonnet Syndrome

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## Abstract

Anton-Babinski syndrome describes visual anosognosia, asomatognosia and confabulation in patients presenting after non-dominant brain injury most often secondary to stroke or in some cases head injury.

Charles Bonnet syndrome describes visual hallucinations observed in patients presenting with blindness.

We present a case of a 67 YO gentleman who had a history of left occipital stroke, who presented with diffuse headache of four days' duration. CT scan head and MRI of brain showed an acute onset right ischemic occipital infarction and an old left occipital infarction he suffered a year prior to current presentation. Patient was witnessed to be hallucinating and confabulating during his admission.

Various theories, which we will discuss, have been proposed to explain the reason behind the symptoms seen in Anton-Babinski and Charles Bonnet syndrome.

We believe this a rare case of Anton-Babinski and Charles Bonnet syndrome presenting in one patient after having suffered infarction to both occipital lobes.

***Keywords:*** *Anton-Babinski Syndrome; Charles Bonnet Syndrome; Bilateral Occipital Infarction*

## Introduction

Whilst we acknowledge from out literature review that case reports have been written about Anton-Babinski syndrome in patients who have suffered brain injury to both occipital lobes as a result of stroke and other causes it is not common to see a patient presenting with both Anton-Babinski Syndrome and Charles Bonnet Syndrome after a bilateral occipital injury. In the case of our patient who presented with bilateral occipital infarction a year apart this was a particularly rare occurrence which we thought was worth writing a report on.

Our patient had comorbid conditions that predisposed him to peripheral vascular disease. Atherosclerosis of the vertebrobasilar artery and cardioembolism are the commonest causes of posterior circulation stroke which is the blood supply for the occipital lobes. Damage to the visual cortex and nearby visual association pathways results in anosognosia, asomatognosia, blindness, confabulation and hallucinations in some patients.

We present a case of a patient who presented with most of the above symptoms.

## Case Report

A 67-year-old Spanish-speaking male was admitted to the medical intensive care unit after he presented with headache of four days' duration with associated blurry vision in both eyes and generalized weakness. Medical intensive care unit stay necessitated because of hyperglycemia, 505, on admission and CT scan of the head without contrast showing large acute to subacute right occipital infarct with a small focus of hemorrhagic transformation. His past medical and surgical history included hypertension, type 2 diabetes mellitus, peripheral arterial disease, dyslipidemia, open heart surgery for aortic valve replacement in 2003, left occipital stroke in 2014 with blurred vision in the left eye, right below knee amputation with left 1st to 5th toe amputation in 2014.

His vitals on admission at the emergency care area and the medical intensive care unit were within normal limits.

Patient had presented in 2014 with blurred vision in left and was subsequently found to have had a left occipital stroke. There was no neurological deficit except for the blurred vision in the left eye.

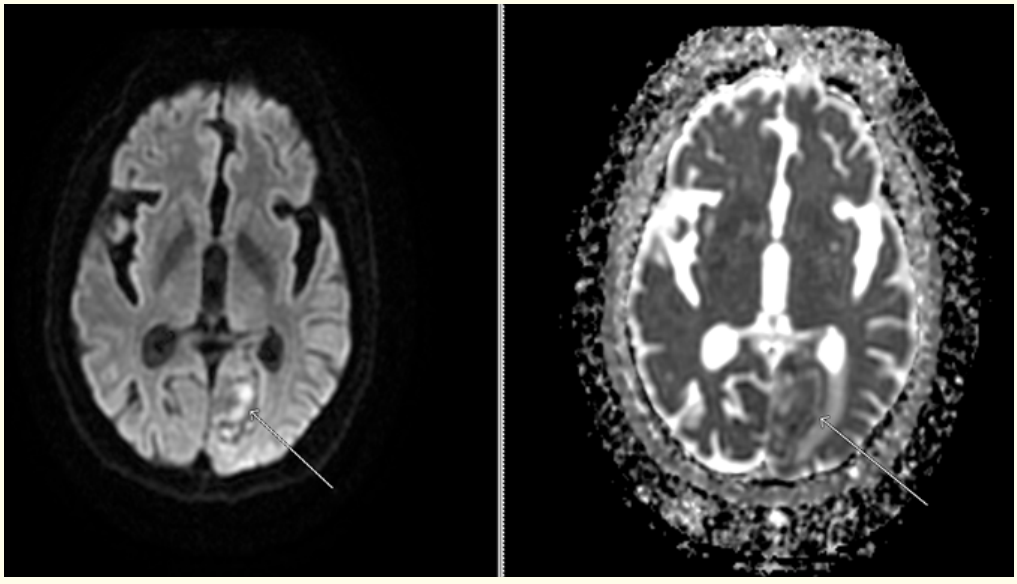
MRI done then showed acute left PCA territory infarction with hemorrhagic transformation in the left occipital lobe and left aspect of the corpus callosum splenium, with small foci of intraventricular extension (Figure 1, A and B).

He was managed in the medical intensive unit with insulin drip and put on stroke protocol.

Throughout his stay in the hospital patient was alert and oriented with Glasgow coma scale of 15/15. There was no motor or sensory deficit in the extremities. The was no cranial nerve deficit except cranial nerve II which on examination showed perception to light in both eyes with an inability to perceive hand motion, count fingers or see numbers on the near card with +250 correction.

Despite this physical examination findings on admission patient subsequently claimed to see people walking around in his room though he was the only patient in the room. He was then showed a pen but he could not name object.

MRI/MRA 05/16/2015 (Figure 1, C and D) done after patient was transferred to the medical floor showed Subacute right occipital and medial temporal lobe infarct with hemorrhagic transformation. This included a 1.1 cm acute hemorrhage in the right hippocampus. Chronic left occipital infarct. Loss of the right distal ICA petrous segment flow void (Figure 1, E and F) was also noted.



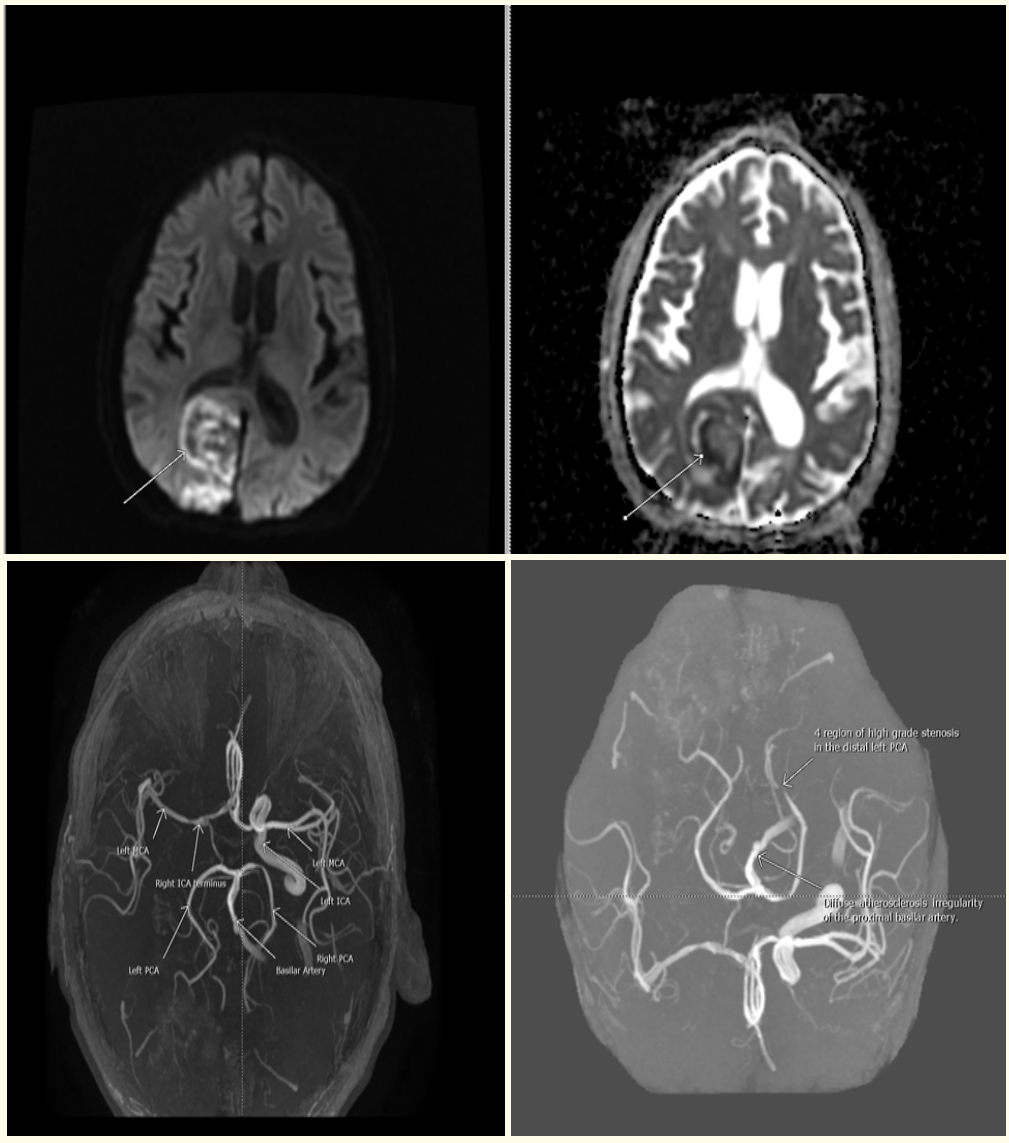



***Figure 1:*** *A (DWI) and B (ADC) Acute left PCA territory infarction with hemorrhage transformation in left occipital lobe as depicted by the arrows.*

***MRI of Brain C (DWI) and D (ADC):*** *There is a subacute right occipital and medial temporal lobe infarct with hemorrhagic transformation as depicted by the arrows. This is new since prior MRI of 1/28/2014.*

***MRA of Head and Neck (E and F):*** *Time-of-flight images demonstrate absent flow related enhancement in the distal ICA. There is reconstitution of the right ICA terminus via collateral flow through the anterior and posterior communicating arteries. Diffuse atherosclerotic irregularity of the proximal basilar artery. 4 mm region of high-grade stenosis in the distal left PCA.*

*PCA: Posterior Cerebral Artery; MCA: Middle Cerebral Artery; ICA: Internal Carotid Artery*

Prior to being discharge he was evaluated by ophthalmology and was assessed to have normal eyes structurally with visual problems likely originating posterior to the retina with the greatest chance of this being due to the bilateral occipital lobe infarcts.

He was discharged with follow up appointment with Neurology, Endocrine and Ophthalmology.

## Discussion

Anton-Babinski syndrome is a rare condition described by Austrian Neurologist and psychiatrist, Gabriel Anton, and French Neurologist, Joseph Jules Francois Felix Babinski [1]. It describes visual anosognosia and asomatoagnosia in patients presenting after brain injury most often secondary to stroke or in some cases head injury.

This occurs in patients with injury to the visual cortex in both occipital lobes and damage to nearby structures. These patients usually mask their neurological deficit by confabulating often deceiving those around them. This deficit is found out only after careful neurological exam that is directed at picking out this deficit.

Charles Bonnet syndrome which is visual hallucination in patients with blindness was first described by naturalist, biologist and philosopher Charles Bonnet in 1769 after he witnessed and documented the visual hallucinations his 89-year-old grandfather, Charles Lullin, who had cataracts was experiencing. In 1936, Georges de Morsier, a neurologist, coined the eponym Charles Bonnet syndrome in recognition of Charles Bonnet [2].

We present this case because it not so common to find both Anton-Babinski syndrome and Charles Bonnet syndrome presenting in one patient.

The above syndromes described above occurred in our patient because of bilateral occipital stroke which occurred a year apart.

The occipital cortex receives it blood supply via the Vertebrobasilar circulation. Most cases of posterior circulation stroke are caused by local arterial atherosclerosis, arteriolosclerosis in lacunes and cardiogenic embolism [3-5].

With our patient having evidence of peripheral arterial disease his bilateral occipital infarcts and lacunar infarcts was most likely due to atherosclerosis and arteriolosclerosis.

Figure 2 depicts the posterior circulation and the various segments of the vertebral artery. The most common segments of atherostenosis in the vertebrobasilar circulation are in the V1 and V4 segment of the vertebral artery [4].

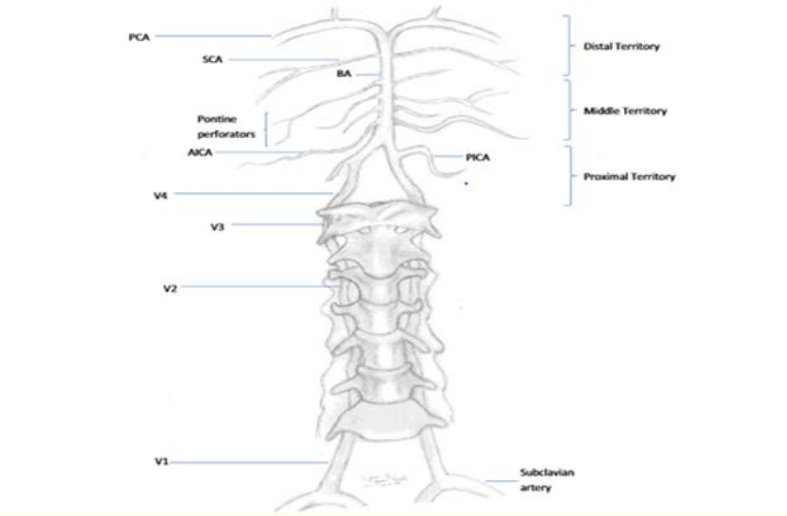



***Figure 2:*** *Vertebrobasilar system (Adapted with permission from Amre Nouh): PCA: Posterior Cerebral Artery; SCA: Superior Cerebral Artery; BA: Basilar Artery; AICA: Anterior Inferior Cerebellar Artery; PICA: Posterior Inferior Cerebellar Artery; V1–V4: Segments of the Vertebral Artery. Proximal territory, areas supplied by the intracranial VAs and PICAs up to the VB junction; middle territory, BA and AICAs up to the SCAs; distal territory, rostral BA, SCAs, and PCAs.*

Various theories have been proposed to explain the unawareness of deficit seen in patients presenting with the symptom complex of Anton-Babinski syndrome, one being the disconnection phenomenon. The theory proposes that a conscious awareness system (CAS) located on the parietal lobes serves as a monitor for all the information received from the senses. It functions by connecting with other independent executive systems located in the frontal lobes. These systems work in tandem to enable an individual to perform complex cognitive tasks. Damage to the visual cortex may be responsible for the symptom complex see in Anton-Babinski syndrome.

Other theories proposed in addition to the disconnection phenomena include a neuropsychologic mechanisms that involves signal transmission to the visual monitor from a secondary visual system, located on the superior colliculus, pulvinar and temporoparietal regions. In the absence of transmission on the geniculocalcarine pathway, this secondary visual pathway would allow blind patients to confabulate. This serves as a false feedback to the visual monitor.

The other theory states that damage to the visual monitor stimuli leads to incorrect interpretation of images [6-8].

For the visual hallucination seen in our patient for which he was diagnosed of having Charles Bonnet Syndrome (CBS), there is no concrete evidence to explain why these patients with bilateral cortical blindness hallucinate. Just like Anton-Babinski Syndrome several theories have been proposed to explain why patients with Charles Bonnet syndrome experience these recurrent visual hallucinations. Some the theories proposed include the phantom-limb syndrome and release hallucinations, sensory deprivation, creating consistency, perceptual release, irritative hallucinations, and senescence.

These theories although explained in a different way tend to have one common factor in that there is lack of sensory input leading to visual hallucinations [2].

A study conducted by FFytche., *et al.* in 1998 looking at the anatomy of visual hallucinations using functional Magnetic Resonance Imaging (fMRI) compared individuals with CBS who had experienced visual hallucinations in the past and individual who had never experienced visual hallucinations. They noticed increased signal intensity in Ventral occipital lobe and within and around fusiform gyrus of patients experiencing visual hallucination and also observed increased signal intensity before the onset of the visual hallucination. Whilst in individuals without visual hallucination there was delay in signal intensity visual stimulation [2].

These findings led to FFytche., *et al*. concluding that visual consciousness is a product of complex neuronal sequences that are influenced by top-down processing that may be located in specialized areas of the brain influencing lower sensory pathways. This is opposite to the usual bottom up sensory processing [2].

Depending on the etiology, patient's symptoms might be reversible but in the case of our patient the etiology was cardiovascular in origin hence the likelihood of reversal of symptoms is minimal to almost nonexistent.

## Conclusion

This is an interesting of Anton Babinki Syndrome and CBS presenting in the same patient. So far, the explanation for these symptom complex remains theoretical. With the rise of atherosclerotic disease these symptom complex might become a common presentation. Physicians need to be on the lookout for these symptoms.

## Author Contributions

Nii Kwanchie Ankrah: Resident for case writing and discussion.
Yongxing Zhou: Resident for patient care, case writing and discussion.
Roger Weir: Attending supervising patient care and case discussion.
Annapurni Jayam-Trouth: Attending supervising patient care and discussion.

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## Author Disclosures

The authors report no disclosures.

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