CASE REPORT

Silent Lacunar Infarct With Peduncular Hallucinosis

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Abstract

Objective: Lacunar infarct is a small infarct in the distal distribution of deep penetrating vessels. A silent stroke is not associated with outward symptoms. The objective of this report is to highlight a case of a silent young stroke presenting with peduncular hallucinosis. Methods: This gentleman was thoroughly investigated and was found to have a lacunar infarct of the Pons and Occipital Lobe. A further referral to Neurology thereafter; for an in depth investigation into the cause of this silent young stroke. Results: Treatment with low dose antipsychotic resolved the complaints; while the episode being transient. Conclusion: Organic psychosis is responsive to antidopaminergic agents, via action on the Ponto-Geniculo-Occipital pathways.

Keywords: Lacunar Infarct, Pons, Young Stroke, Peduncular Hallucinosis, Organic

Introduction

Young strokes are generally accepted as sufferers who are of less than 45 years old. We report on a rare case of a young gentleman, who presented with formed visual and auditory hallucinations. He responded promptly to a low dose atypical antipsychotic within a fortnight. Investigations to rule out organic causes showed a silent lacunar infarct, which the gentleman was asymptomatic to.

The incidence of stroke for those under the age of 35 occurring is less than 1 in 100,000 (<0.01%) and increases with age¹. In Western countries, the prevalence of stroke in young adults (aged 20-40) is less than 5% and the readings go up to between 19% and 30% in developing countries².

Causes of young stroke are commonly due to cardioembolism, premature atherosclerosis, hematological and immunological causes⁵. Rarer causations are hereditary connective tissue disorder, genetic factors such as CADASIL (Cerebral Autosomal-Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy) and mitochondrial cytopathies³.

A silent stroke, commonly ischemic and lacunar type, is symptom-free, and patients are unaware that they have suffered a stroke. Despite that; silent strokes still carry
significant risk of transient ischemic attack (TIA) or major strokes in the future, vascular dementia and cognitive decline. Young strokes also account to 20 odd years of potential life lost.

The pons plays a role in dreams and sleep, has a pneumotaxic center, and is the point of origin for a few cranial nerves. Occipital lobe damage has been touted responsible for visual hallucinations with visual field deficits in mentally stable patients. It has been proposed to be secondary to inappropriate activation of neurons at the affected region. Charles Bonnet syndrome is one such, with characteristic liliputian hallucinations.

Post-stroke psychosis prevalence rates are estimated to be around 3-4%. Psychotic symptoms arise from various regions of the brain post-stroke; however, the most common locations are Temporo-Parieto-Occipital lesions. Consideration of overt brainstem lesions have to be considered in new onset visual hallucinations.

Case report

A 24 year old Muslim gentleman, Mr A., presented to our Psychiatric clinic, with complaints of vivid auditory hallucinations (AH) for three weeks. He describes it as hearing the voices of a famous Ustaz (religious preacher). Also present were visual hallucinations (VH). At different moments, he saw an Ustaz walking in the city, dressed extravagantly and accessorized with religious ornaments, bearing no regards from the surrounding public, nor did he interact with Mr A. He brushed it off, but with a sense of unrest as in to why he had seen with such particularity.

Also present was a bizarre delusion; whereby the patient believed that he had the ability to communicate with colleagues and friends via ultrasonic radio waves. Closing his eyes gave him symbolistic answers to his internal questions, in the form of a word ‘yes’ or ‘no’ visible at the back of his eyelids. Mr A also became hyperreligious; reading religious transcripts and sharing sermons.

This was his first contact with Psychiatry. There was no family history of psychiatric illness. He denied use of illicit drugs, nor smoking or alcohol consumption in the past. He has had no previous medical conditions, or admissions to a hospital. Birth history was fairly insignificant, with no antenatal and perinatal complications. He remained physically active, with cardiovascular endurance exercises 2 to 3 times weekly. He was an above average student.

Physical examination including neurological exam was insignificant. The results of a full blood count, serum biochemistry, urinalysis were all within normal limits, with a negative urine toxicology screen. A low dose atypical antipsychotic; using Risperidone at 1mg twice daily was prescribed, which he tolerated well without any extrapyramidal side effects.
Fig. 1 and 2 shows the pre- and post-contrast CT films. The red circles indicate the hypodensities over the right occipital lobe and right side of pons.

Within two weeks, he returned and attested to the resolution of his psychotic symptoms. Brain computer tomography revealed a Right-sided lacunar infarct involving the Occipital Lobe and Pons.

Discussion

Strokes are mainly ischemic or hemorrhagic; usually presenting with unilateral weakness, with or without paresthesiae, slurred speech, facial drooping, and occasionally loss of gag reflex, ataxia, signs and symptoms of increased intracranial pressure, vertigo or visual defects. A lacunar infarct is when there is an occlusion of deep penetrating arteries of the brain, and generally has 5 syndromic variants. Vertebrobasilar insufficiency is an inadequacy in the supply of blood to the posterior circulation of the brain; including the pons and occipital cortex; the two regions implicated in this patient.

Post stroke neuropsychiatric sequelae are common, such as depression, anxiety, pseudobulbar affect and psychosis. But there has been a lack of data and publications pertaining to young silent strokes with peduncular hallucinosis, making this case unique.

Post stroke psychosis can be due to anatomical lesions involving several regions, namely the cortex (frontal and occipital) and subcortex; midbrain, thalamus, basal ganglia and brainstem\textsuperscript{11,13}.

The gentleman in this case did not have a premorbid history or a family history of psychiatric illness. The onset of psychotic symptoms was abrupt, with no preceeding delusional mood or perplexity, unlike the typical prodrome of Schizophrenia. Also, his
VH were formed and distinct; all these pointing towards an organic diagnosis.

Peduncular hallucinosis is a rare cause of VH. It was described by Frenchman, Jean Lhermitte; touting the brainstem with a release phenomenon, mainly the cerebral peduncles to be involved. It is reported to be vivid, dream-like, and bizarre. The images are formed and are of people mostly. Insight is commonly preserved. Such a description fits in line with what was stereotypical of Mr A’s experience.

There are two main mechanisms thought to be responsible for such a phenomena due to stroke. The first is a dysregulatory loss of inhibition of Ponto-Geniculo-Occipital (PGO) connecting waves causing VH similar to rapid-eye movement (REM) sleep. The second would be direct damage to primary visual cortex causing misreading of undamaged visual association areas.

Atypical antipsychotics that regulate serotonergic pathways, and also dopaminergic antagonism are of benefit in such patients.

**Conclusion**

Psychosis is due to a myriad of causes, functional and organic in nature. The presence of VH should alert a differential diagnosis of a probable organic cause. VH quality and characteristics will differ based on the location of lesion. Atypical presentation in such a case where there were VH, a lack of preceding delusional mood, no risk factors apart from being male; may very well be a common clinical presentation. It is of utmost importance that a thorough assessment be done, to avoid diagnostic labeling and stigmatization associated with psychopathology. Further research into the role of PGO waves in psychosis and psychopathology holds promise; where there is a common pathway of aminergic neurons.

**References**


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