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# Isolated metamorphopsia: A rare presentation of splenial infarction of the corpus callosum

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## ABSTRACT

Metamorphopsia is the visual misperception of the shape of objects. Retinal diseases, especially those affecting the macula, are responsible for the majority of cases. However, it can also be seen during migraine attacks, epileptic seizures and damage to visual pathways in the brain.Infarction in the splenium of the corpus callosum may cause visual agnosia, prosopometamorphopsia, alexia and amnesia. We describe here a case of isolated metamorphopsia caused by splenial infarction in the absence of any other neurological symptoms.

**Keywords:** splenium, ischemia, visual distortion

## INTRODUCTION

Metamorphopsia is commonly seen in macular and retinal disorders. However, it can also be related to diseases of the brain. The general complaint of patients with metamorphopsia is visual misperceptions such as changes in size, boundaries and angles of objects while visual acuity is not always affected. However, visual impairment may accompany metamorphopsia in patients with stroke, tumor, migraine attacks, epileptic seizures, exposure to certain toxic substances and some infectious conditions (1-4). In the literature a number of cases with lesions of the optic chiasm, optic radiation, corpus callosum, temporo-parietal cortex and occipito-parietal cortex have also been reported (5-6). We describe here a case with bilateral metamorphopsia caused by brain ischemia resulting from bilateral splenial infarction who first applied to an ophthalmology outpatient clinic.

## CASE REPORT

A 62 year old, right-handed woman attended an ophthalmology outpatient clinic 3 days after the sudden onset of visual discomfort. She complained of blurriness and dizziness when she tried to focus on details of television scenes or newspaper lines. Her visual acuity at distance was 20/20 without correction and color vision was within normal limits in both eyes. Pupils were equal and bilaterally reactive to light. Extraocular muscle motility was normal and no diplopia was detected in gaze. Fundoscopic examination was found in normal limits. During an Amsler grid test, the patient stated that she felt dizzy and experienced very mild distortion on the central lines of the grids in both eyes that suggested metamorphopsia. She was referred to a neurology outpatient clinic for further investigation about extraocular causes of metamorphopsia.

During the neurological examination, she was oriented and cooperative. The cranial nerves were normal. No abnormal signs were detected during piramidal, extrapiramidal, cerebellar or sensory system examinations. She could read and write numbers and letters properly. Blood pressure was 180/110 mm/Hg. After 24 hours tension holter evaluation. Magnetic resonance imaging (MRI) showed a hyperintense area in the T2-weighted se-

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quence and restricted diffusion in the splenium of the corpus callosum (Fig. 1). The doppler ultrasound of the carotis and vertebral arteries showed minimal atherosclerosis but no occlusion. Echocardiography showed mild left ventricular hypertrophy. No cardiac arrhythmia was detected by the 24 hour rhythm holter test.

Our diagnosis has been established as hypertensive ischemic stroke resulting in splenial infarction of the corpus callosum. Lisinopril 5 mg/day and acetylsalicylicacid 300 mg/day were started. Her visual discomfort and dizziness recovered six weeks later. Also, Amsler grid test was found within normal limits bilaterally.

## DISCUSSION

Metamorphopsia is an ophthalmological symptom that is difficult to recognise in neurological examinations, especially when it is the patient's sole symptom (7). We present here a rare case of metamorphopsia related to splenium of corpus callosum (CC) infarction. The clinical findings regarding splenial infarcts are very complicated and this may lead to misdiagnose (7). Clinical signs and symptoms such as confusion, ataxia, dysarthria, headache, hemiparasis, spastisty and seizure are variable. Furthermore, some cases with prosopometamorphopsia (isolated interruption of facial recognition) have been described in splenial infarcts (8,9). Our case is an exceptional clinical presentation of splenial CC infarction because metamorphopsia and related dizziness are not usual symptoms in these patients.

CC infarction is not commonly seen because of its rich blood supply. However, isolated infarction of the splenium is seen more often than in other parts of the CC because the other parts such as the body and genu have more collateral vascularisation (10). The blood supply of the splenium is provided via posterior cerebral circulation. It is attributed to this that the incidence of posterior cerebral artery infarcts are greater when it is compared with anterior cerebral artery infarcts (10,11).

Splenium have heterogeneous homotopic and heterotopic connections with other cortical areas such as visual cortex, language areas and Papez circuit. However, there is marked interindividual variability in terms of splenial connectivity (12-15).

Our patient stated that her vision was blurred and she felt dizziness when she tried to focus on the details of objects. Ophthalmological examination was normal except for the Amsler grid test. The Amsler grid test suggested that the patient was ex-

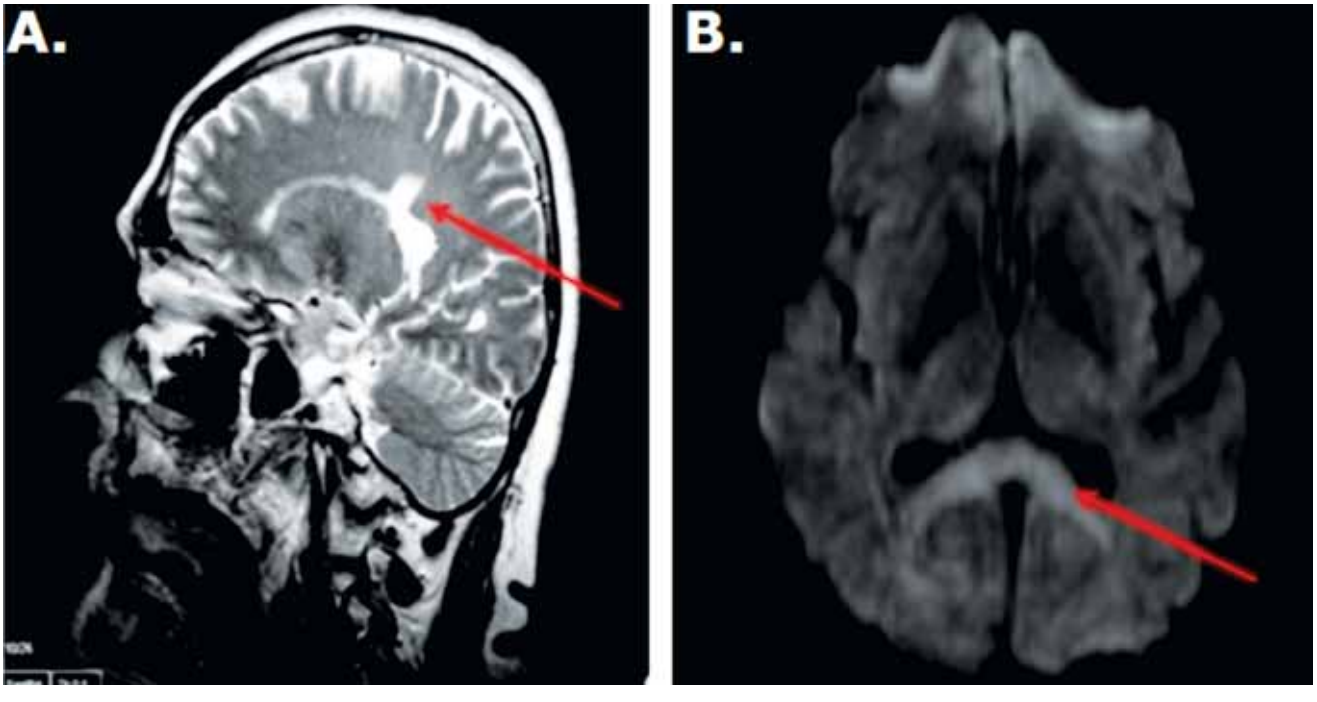


***FIGURE 1.*** *Arrows show hypertensities*
***A.*** *in a T2-weighted sagittal and* ***B.*** *diffusion-weighted axial slice of the brain MRI, suggesting acute infarction producing cytotoxic oedema involving the splenium of the CC.*

periencing metamorphopsia. We also suspect that her dizziness was triggered by disturbed vision.

Pure alexia was reported due to the splenial lesions that intercepted both the association fibers from the occipital cortex to the left angular gyrus (16). Amnesia can be seen in the retrosplenial cortex lesion due to disconnections between the Papez circuit and the splenium. The collosal fibers are damaged between the homologues corticalareas of 17-18 that may result in visuo-spatial disorientation (17). No alexia, amnesia or visuo-spatial disorientation was detected despite bilateral splenial infarction in our patient.

To the best of our knowledge, this is the first case of isolated metamorphopsia caused by bilateral splenium infarction in the absence of any other neurological symptoms. Physicians must keep in mind that splenial infarction of the corpus callosum could be the causative etiology in patients with metamorphopsia.

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