Improvement of Astatikopsia (Riddoch's phenomenon) after correction of vertebral stenoses with angioplasty

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Abstract

Visual perception disorder detection may be challenging with several dozen different syndromes identifiable. These may range from hypofunction to hyperfunction in the topological as well as hodological dimensions of cerebral structure. We report here a case of a 61-year-old white man presented with dizziness and visual impairment.

Case Report

Visual perception disorder detection may be challenging with several dozen different syndromes identifiable. These may range from hypofunction to hyperfunction in the topological as well as hodological dimensions of cerebral structure, as recently reviewed in an excellent treatise on the subject.¹ Motion detection is a key sensory attribute and when impaired can be remarkably disabling to people with the syndrome of akinetopsia, most dramatically evidenced by crossing a busy road. In this case report, we describe the opposite to akinetopsia. This has been variously termed statokinetic dissociation² or Riddoch's phenomenon³ and refers to appreciation of objects only once they move, whereas when static they are not seen or appreciated.

Index patient

A 61-year-old white man presented with dizziness and visual impairment. He was an ex fighter pilot and until recently a bank CEO. He described his visual impairment as a loss of appreciation of stationary objects referring primarily to people but being able to see them as soon as they moved. He cited specific examples such as when in a room filled with people, he would not be able to acknowledge a motionless person. He also had difficulty identifying several people at a distance of 10-20 ft away as they would disappear and he described the scene *like a fiery haze*. He was nevertheless able to see fine detail. For example he related that he was able to perceive and accurately

portray minuscule insects once they were in motion but not when stationary. These symptoms had been present for over a year without further improvement.

Relevant past history, included risk factors of hypertension, diabetes mellitus type II, coronary artery disease, hypercholesterolemia, hyperhomocysteinemia and remote smoking history.

Examination

Pertinent cognitive evaluation revealed an alert, rational man with normal language function save for occasional dysfluency. He had a number of posterior brain syndromes such as acquired alexia as evidenced by letter-by-letter reading, features of visual agnosia, prosopagnosia in that he was unable to recognize the same examiner visually as well as simultanagnosia. He had subtle achromatopsia only for the color pink. In addition dysmemory and dyscalculia were evident. Cranial nerve testing revealed no hyposmia, with pupils equal in diameter, round and reactive to light with no afferent pupillary reflex defect. Dilated fundal examination showed healthy retinal structures with no pathology noted. Visual acuity testing revealed some fluctuation in acuity and ranged from OD 20/200-20/400 and OS 20/100-20/400. A left quadrantanopic inferior nasal visual field loss was noted in addition to bilaterally concentrically restricted fields that were documented by ophthalmological consultation and field plots. External ocular movements were full without nystagmus. There were no sensorimotor deficits, reflex asymmetries or imbalance with tandem walking and Romberg's test normally performed.

Laboratory

Complete blood count, basal metabolic panel, lipid panel, B12, folate, ESR, CRP, TSH and liver function tests were normal with elevations noted in BUN 28, creatinine 1.9, glucose 171, triglycerides 291, homocysteine 22.4 (4.1-13.1), hemoglobin A1C 7.8 (3.9-6.7). Serological tests included a negative RPR.

Cardiology

Echocardiogram normal and ECG revealed sinus rhythm with 1st degree heart block and old inferior infarct.

Neuroradiology

MRI: Bilateral posterior cerebral artery and posterior inferior cerebellar artery territory regions of infarction within both occipital lobes, the right parietal lobe, posterior right medial temporal lobe, and right cerebellar hemisphere. Lacunar infarcts in right frontal, basal ganglia and left thalamus (Figure 1). The MR angiogram revealed absent posterior communicating arteries bilaterally.

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Angiography

The magnetic resonance angiogram revealed high grade tandem stenosis of left internal carotid artery and high grade bivertebral origin stenosis. The catheter four vessel cerebral angiogram revealed 75-80% stenosis of right vertebral artery at the C6 level and 40-45% long segment of stenosis at the origin (Figure 2) and 85-90% stenosis at the origin of left vertebral artery and 55-60% at the C6 level. The left internal carotid artery showed 60-65% stenosis at the bulb, 75-80% at the C2 level, 50-60% involving the presellar segment, and 60-65% involving the supraclinoid segment (Figure 3).

Clinicoradiological assessment

Extensive cervicocephalic stenotic atherosclerotic disease on the basis of multiple significant cerebrovascular risk factor and in particular involvement of the posterior circulation with likely artery to artery embolism to the posterior circulation.

Procedures

Percutaneous transluminal angioplasty was performed during 2 separate visits and included the cervical segment right vertebral artery stenosis with no evidence of residual stenosis. Angioplasty and stenting was performed, with a distal embolic filter device with the stent extending from the distal left common carotid artery to the left internal carotid artery with no evidence of residual stenosis within the stent. After the first revascularization procedure of the right vertebral origin stenosis (with no residual stenosis and good angiographic result) he reported a *dramatic clearing of his head* notably the vision abnormality of seeing people only when they moved.

Discussion

Neuroanatomical understanding of the visual system is key to understanding the clinical features of this patient. This report reflects the differential involvement of the visual association cortices and a brief tour of the pertinent neuroanatomy is relevant. The visual system consists of several parallel pathways, notably the dorsal and ventral stream from the occipital region to the parietal and temporal regions, that, amongst other features, process motion and form respectively. The processing of visual motion however begins at the retina where the M cells project to the magnocellular layers and P cells project to the parvocellular layers of the lateral geniculate nucleus of the thalamus. Cells in these layers project to different sub-layers in 4C of the primary visual cortex (V1), where they feed into parallel pathways extending to cerebral cortex. The M cells are sensitive to stimuli with lower spatial and higher temporal frequencies and the P cells are essential for color vision. V1 combines visual information from the two eyes. The primary visual (striate) cortex is comprised of columnar units, each with their own receptive field containing simple and complex cells. Simple cells respond to illumination and orientation and complex cells respond to sharp, dark and light contrasts. V2 is the largest visual association area from which cells respond to orientation, motion, wavelength and depth. V3 region neurons respond mainly to spatial orientation, direction and depth and lesions here can impair motion and depth perception and form analysis presenting clinically as visual agnosia. The V4 area is in the lingual sulcus and fusiform gyrus and involved in hue perception, intermediate form vision and visual attention in the contralateral superior quadrant. Dominant V4 activation is seen with word processing as measured by f-MRI. The dorsal area of V4 (V4d) may be involved in size estimation and V4v (ventral) is activated by objects identity. V5 is the motion area located near the junction of the lateral temporo-parietal-occipital junction and responds both speed and direction of a moving stimulus. V6 is activated by visual analysis of 3 dimensional shapes, as well as visual search and arm reaching movements. Finally V8 represents the color area in the fusiform gyrus and V7 in humans remains uncharted.⁴

With particular reference to the dorsal and ventral streams, what has not been resolved, is the so called *binding problem*; a final common path that brings together all the various elements of a complex percept.⁴ Motion perception deficits can be caused by any kind of disconnection within the motion processing stream. Stimulation of V5 is a much more potent way of inducing akinetopsia than stim-

Figure 1. MRI Brain T-2 FLAIR sequence showing the bilateral occipital infarcts.

Figure 2. Right vertebral artery.

Figure 3. Left internal carotid artery.

ulation of V1 which abolished motion perception only marginally.⁵ In accounting for his residual vision in terms of active cortex, we hypothesize that the asymmetric loss of V1 to V2 bilaterally has affected object and form detection with relative preservation of V5 motion sensitive. As a consequence, the perception of stationary *versus* motion objects is altered causing astatiksopsia⁶⁻⁷ Riddoch's phenomenon pertains to the ability to perceive the motion while being unable to detect any other features of that object such as its color or its shape of an object,³ also called statokinetic dissociation.² Other similar syndromes include blindsight (collicular vision may guide non-conscious behavior) and agnosopsia.⁸

Our case has some differing features to previous descriptions of this phenomenon in that our patient clearly defined and was able to distinguish different people and recognize them by sight. A post procedure angiogram was notable for remarkable improvement in the right vertebral stenosis without demonstrable

residual stenosis. Ideally a pre and post SPECT or PET brain imaging would have been an optimal surrogate test to reveal brain perfusion improvement.

Considerable improvement of this complex visual disorder was possible by appropriate revascularization procedures, underscoring that precise clinical measurement and ascertainment of deficits remain pivotal in managing patients. Alternatively a second pathway reaching V5 from the retina, a fast one which bypasses V1 has been proposed and may have played a role in the recovery.⁹

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