

CASE REPORT

The clinical potential for serial visual field assessment in patients with parietal lobe lesions: an illustration with a ruptured left parietal cerebral arterio-venous malformation

*Olufunmilola A OGUN¹, Abiodun A ADEYINKA²,
Atinuke M AGUNLOYE², Temitayo SHOKUNBI³*

AFFILIATIONS

Departments of:

¹Ophthalmology

²Radiology

³Surgery

College of Medicine

University of Ibadan /

University College Hospital

Ibadan, NIGERIA

CORRESPONDING AUTHOR

Temitayo SHOKUNBI

Division of Neurological
Surgery, Department of

Surgery, College of Medicine

University of Ibadan /

Department of Neurological

Surgery, University College

Hospital, Ibadan, NIGERIA

Email: temitayoshokunbi@yahoo.com

Phone: +234 802 291 2220

ABSTRACT

Background: The parietal lobe plays an important role in integrating visual, auditory and somatic sensory information to create an awareness of the body in space, which guides the planning and execution of movements. Parietal lobe lesions therefore present with deficits of sensory, visual and executive functions.

Methodology: Serial visual field assessment was performed in a patient with acute dominant parietal lobe haematoma following the rupture of an arteriovenous malformation.

Results: This case report is presented and concepts are discussed, exploring the use of serial visual field assessment as a surrogate means of monitoring neurological improvement where serial neuroimaging may not be possible.

Conclusion: In practice settings with limited access to neuroimaging, the potential for the use of serial visual field assessments in conjunction with clinical observation, as a simple means of monitoring lesion resolution or progression and functional recovery or deterioration is proposed. Future research is recommended to confirm this.

Keywords: Haematoma, intracerebral, neuroimaging, visual recovery

Received: 15th February, 2015

Accepted: 15th March, 2015

DISCLOSURES: NONE

INTRODUCTION

The parietal lobe plays an important role in integrating visual, auditory and somatic sensory information to create an awareness of

the body in space, which guides the planning and execution of movements (e.g. reaching out to pick up an object), as well as the detection and analysis of motion.¹ Lesions of

this area may, therefore, present with visual field defects along with visuo-spatial syndromes epitomized by the Gerstmann's syndrome.² The latter is characterised by finger agnosia, agraphia, right-left disorientation and acalculia.³

Two discrete pathways, both arising from the primary visual cortex, process visual information in the brain. The dorsal stream conveys visuospatial information and localizes objects in space. The ventral stream mediates colour perception and object and form recognition. There is considerable cross talk between the two systems. Visual field assessment is potentially valuable for localization of the lesions in the parietal lobe but its value as a surrogate measure of progression of or recovery from lesions of this part of the brain has not been extensively studied.

We present a patient who suffered a left parietal intracerebral haemorrhage and who presented with an incongruous right homonymous hemianopia with Gerstmann's syndrome. We tracked clinical improvement by serial measurements of visual field, and postulate that this can be used as an objective measure of lesion evolution in the parietal lobe.

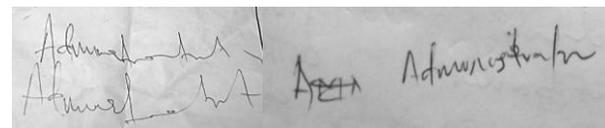
CASE REPORT

A 37-year old right-handed man was referred for neurosurgical evaluation. He suffered sudden loss of consciousness, right-sided weakness associated with severe headache and vomiting, two weeks prior to referral. There was no history of hypertension, diabetes mellitus or head trauma. However, there were recurrent afebrile seizures in early childhood and adolescence, and two episodes of altered consciousness in the five years preceding his presentation. He was not on any medication at the time of the present attack and there were no visual complaints.

On examination, he was conscious and alert), but had slurred speech with delayed response times and evidence of dysgraphia (Figures 1a & 2a), dyscalculia, right-left disorientation and finger agnosia. There was no evidence of anomia (anomic aphasia).

A provisional diagnosis of acute intracranial haemorrhage in the territory of the left middle cerebral artery, secondary to an arteriovenous malformation was made. The features of Gerstmann's syndrome were noted.

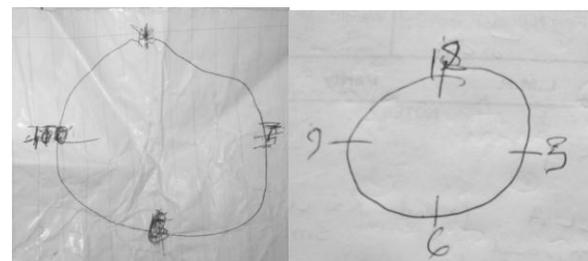
Figure 1(a): Dysgraphia [Patient was unable to write the word "Administrator" to describe his profession at presentation. (b) Resolved after 2 months: Patient was able to write the word "Administrator"]



1(a)

1(b)

Figure 2(a): Dysgraphia and right/left disorientation [Patient was unable to draw and correctly label the face of a clock at presentation (b) After resolution, patient's depiction of the face of a clock with appropriate labels



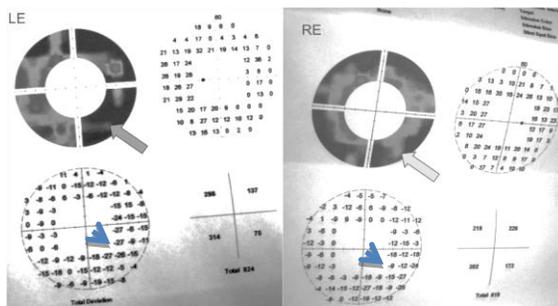
2 (a)

2 (b)

Automated perimetry of the peripheral 30-60° of visual field was done (shown in Figure 3a) as the density of the central 30° (CVF 30-2) field loss was too severe to plot initially. Block

arrows indicate that inferior quadrants were more severely affected, also, represented on the total deviation plot showing a higher degree of visual loss in the right-sided inferior quadrant in each eye (smaller arrows).

Figure 3(a): Right homonymous hemianopsia depicted on automated perimetry of the peripheral 30-60° of visual field. [Block arrows indicate that the inferior quadrants of the right hemifields of both eyes were more severely affected than the left hemifields].



This is also represented on the total deviation plot, which shows a higher degree of visual loss (lower sensitivities) in the right-sided inferior quadrant in each eye (smaller arrows)

Figure 3 (b): Improvement of symptoms enabled a plot of the central 30° at 5 months post-incident, with almost complete resolution of the hemianopsia leaving only a few relative scotomas within the right hemifield of each eye as shown on the pattern deviation plots (thick arrows). [Fixation losses, across the vertical midline, in the left eye, reflect the patient's easy fatigability and occasional wandering gaze]

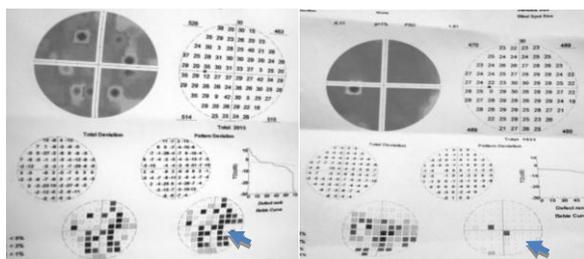


Figure 4: (a) Signal void in substance of the arteriovenous malformation (AVM) in the left parieto-occipital region (yellow arrow); (b) and (c) are lower cuts showing the features of chronicity

developing within the haematoma on T₁, axial MRI scans (yellow arrows); (d) MRA of the Circle of Willis is shown, demonstrating the abnormal distal 1/3rd segment of the left MCA, compressed and embedded within the haematoma (thin arrows). The faint outline of a tortuous venous pool of vessels is indicated by the thick white arrow.

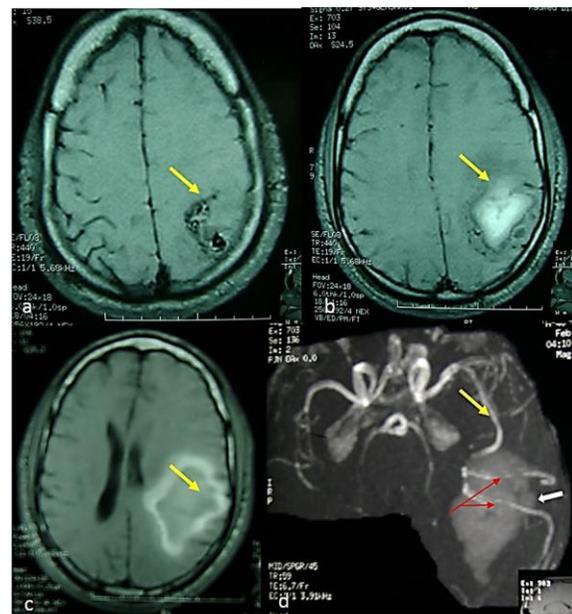


Figure 3a shows the right-sided incongruous homonymous hemianopsia worse in the lower quadrants, indicative of a left parietal lobe lesion; though vision remained 6/5 bilaterally and the ocular examination was, otherwise, normal.

Low-field magnetic resonance imaging of the brain (two weeks after patient's initial collapse) showed serpiginous signal void intensities in the left parieto-occipital region (figure 4a); lower cut (shown in figure 4b) showed a diffuse hyperintense lesion with a peripheral rim of hypointense signal, indicating chronic haematoma. Subsequent lower cut at the level of the body of the lateral ventricle, (figure 4c) showed central hypointensity within the haematoma.

Magnetic resonance angiography (MRA), of the Circle of Willis (figure 4d), showed a well-

defined, normal proximal 2/3rd of the left middle cerebral artery, MCA (yellow arrow). However, the distal 1/3rd showed irregularity and effacement (thin red arrows), and was embedded in a pool of tortuous venous circulation (thick white arrow) surrounded by a well-defined oval-shaped, hyperintense haematoma in the left parieto-occipital region. These features were indicative of an arteriovenous malformation with chronic haematoma.

He was managed conservatively, with mannitol and dexamethasone for the cerebral oedema and raised intracranial pressure; and subsequently, had Gamma-knife irradiation of the left-sided parieto-occipital arteriovenous malformation in India, four months later.

Repeated evaluation showed a slow recovery of speech, calculation skills, handwriting/drawing (figures 1b & 1b) and resolution of the finger agnosia and right-left disorientation associated with a concurrent improvement in the visual field maps (figure 3b), over about seven months. Though there was recorded improvement in subsequent visual field assessments, this was still marred by the patient's inability to concentrate for prolonged periods at a stretch. There have been no more episodes of seizures or loss of consciousness; and the patient has returned to work.

DISCUSSION

Language is the comprehension and communication of abstract ideas. Reading and writing are important tools for language expression. As language deficits are rare, we lack well-standardized and validated methods of assessment. When the dominant hemisphere is affected, which is the left hemisphere in majority of people, language deficits do occur.

Visual field defects can also affect reading and writing; complicating the clinical presentation of language deficits. Parenchymal brain haemorrhage presents with visual field defects even when non-"eloquent" brain areas are affected. It was, therefore, relevant in this patient to exclude any co-morbidity affecting the visual fields, visual processing speed or attention and cognitive disorders, in order to establish a neurological cause for his expressive language deficit.

His right hemianopsic visual field loss, denser in the inferior quadrants was consistent with the parietal lobe haematoma and this type of field loss could present reading and writing difficulties (as it becomes more difficult to see the full extent of a word or sentence). However, his difficulty in reading could not be attributed to his visual field abnormalities alone, as such a diagnosis does not account for the disorder of orientation or apraxia. It was, therefore, concluded that the visual field defects were a result of the primary pathology, as were the associated reading difficulties, dysgraphia, dyscalculia and agnosia. Although the patient's general condition and easy fatiguability may have contributed to his relatively poor performance (high fixation losses) especially in the right eye. Nevertheless, over the following weeks, as his apraxia and aphasia improved and his clinical condition stabilized, so did his visual field maps.

Various studies have explored the recovery of previously 'lost' visual fields following cerebral vascular events and other brain lesions and have discussed possible means of stimulating recovery at the transition zone between 'seeing' and 'non-seeing' fields.^{7,8,9,10} This technique is referred to as vision restoration therapy. In this case, however, the recovery and improvement observed following visual field assessment was spontaneous, and mirrored the clinical

improvement accompanying resolution of the parieto-occipital haematoma.

We recommend that future research should explore the potential of serial visual field assessment in patients with neurological language deficits, as a means of evaluating clinical improvement and clot resolution and determine if there exists a potential for the evaluation of the rate of improvement in visual field recovery as a prognostic sign in lieu of repetitive cost-intensive neuroimaging; in resource-challenged environments, where cost-effective clinical care is needed.

CONCLUSION

In conclusion, we have described a patient with a left parieto-occipital lobe cerebral haematoma with corresponding neurological deficits and visual field defects, who demonstrated reversal of visual field deficits which mirrored clot resolution and clinical recovery.

We, therefore, propose that serial visual field assessment may mirror progressive neurological and radiological improvement in parietal lobe lesions and that serial visual field assessment may be an inexpensive alternative to serial neuroimaging in monitoring clinical progression. This needs to be proven in a large sample of patient population.

REFERENCES

1. St. Peter J. Clinical insights into the parietal lobe. *Clinical Eye and Vision Care* 1998; 10: 113-118.

2. Gerstmann J. Zur Symptomatologie der I-lirniasionenim Obergangsgebiet der unteren Parietal- md mittleren Occipital windung. *Nervenarzt* 1930; 3: 691-695.
3. Gerstmann J. Fingeragnosie und insolierte Agraphie: einneues Syndrom. *Z ges Neurol Psychiatr* 1927; 108:152-177.
4. Mayer E, Martory M, Pegna A, *et al.* A pure case of Gerstmann syndrome with a subangular lesion. *Brain* 1999; 122: 1107-1120.
5. Bassan H, Limperopoulos C, Visconti K, *et al.* Neurodevelopmental Outcome in Survivors of Periventricular Hemorrhagic Infarctions. *Pediatrics* 2007; 120: 785-792.
6. Glisson C. Capturing the benefit of vision restoration therapy. *Current Opinion in Ophthalmology* 2006; 17: 504-508.
7. Muellera I, Mast H, Sabela B. Recovery of visual field defects: A large clinical observational study using vision restoration therapy. *Restorative Neurology and Neuroscience* 2007; 25: 563-572.
8. Schmielau F, Wong E. Recovery of visual fields in brain-lesioned patients by reaction perimetry treatment. *Journal of Neuro-engineering and Rehabilitation* 2007; 4: 31. (doi:10.1186/1743-0003-4-31)
9. Jones S, Roger S. Improving outcome in stroke patients with visual problems. *Age and Ageing* 2006; 35: 560-565.
10. Reinhard J, Schreiber A, Schiefer U, *et al.* Does visual restitution training change absolute homonymous visual field defects? A fundus controlled study. *British Journal of Ophthalmology* 2005; 89: 30-35.