

Patterns of Apperceptive and Associative Visual Agnosia amongst Patients with Neurological Disorders in a Teaching Hospital Annex in Nigeria

Dr Henrietta Olubusayo Osoba

Master of Applied Neuroscience Research Project, School of Advanced Education Research and Accreditation, Universidad Isabel 1, Spain.

Corresponding author email: nidesoby@gmail.com

Affiliation: General Hospital Gbagada, 1 Hospital Road, Gbagada, Lagos, Nigeria.

ABSTRACT

BACKGROUND: Visual agnosia is a significant cause of visual morbidity affecting some patients with neurological disorders. These disorders could range from vascular, degenerative, traumatic amongst others. It is usually visually disabling for the sufferers, who are usually being managed by the neurologist, psychiatrist, or a combination of both. Depending on the area of the brain involved, agnosia could vary in presentations. Furthermore, visual agnosia specifically may also vary in type, and may and may not occur in the presence of other neurological signs and symptoms. Knowledge of the presentation patterns of visual agnosia, could help in early recognition and prompt treatment and/or rehabilitation for the individuals affected.

AIM: This study aims to find out the presentation patterns for visual agnosia amongst the sufferers in patients with neurological diseases

METHOLOGY: A hospital based observational study (case series/report).

RESULTS: Results showed diverse neurological presentations for patients with visual agnosia with all the cases having abnormality of the optic nerve 8(100%). Different areas of the brain were involved. However, the majority, 3 out of the 8 (cases (37.5%) had affectation of the occipital lobe. The other associated neurological signs include hemiparesis, tremors, involuntary muscle movement, cranial nerve abnormalities amongst others. The neurological diseases in this study presenting with visual agnosia include stroke, multiple sclerosis, Parkinson's disease, degenerative dementia, and traumatic brain injury.

DATA ANALYSIS: Data was entered and analyzed using IBM SPSS version 25 software. Student T test would be used for the numerical variables obtained in the study while ANOVA would be used for the different categorical variables. The mean, standard deviation and percentages of the variable would be determined. Confident interval of 95% and a p value of <0.05 would be considered significant.

CONCLUSION: Visual agnosia may occur in patients with diverse neurological diseases. It may be more common than previously diagnosed as patients may not complain of it unless asked or elicited yet could severely affect the quality of life of the affected individuals. The presentation patterns may also vary depending on the underlying neurological pathology and type of visual agnosia. Adequate recognition and management of visual agnosia is key in ensuring the best prognosis for those affected.

Keywords: Apperceptive visual agnosia; associative visual agnosia; visual agnosia;neurological disorders

INTRODUCTION

Agnosia is an inability to identify and recognize objects, sounds or persons using either one or more of their senses even though the senses have a normal function (Kumar, 2023). Visual agnosia has been described as a group of neurological disorders in which the affected individual cannot recognize some or all aspects of an object by sight but by other means such as sound or touch (Barton, 1999). It occurs due to the involvement of certain regions of the brain in a pathological process. Although visual agnosia is a rare occurrence, it is usually grossly disabling. The deficit in vision is unexplainable by language problems, memory, attention, or unfamiliarity with the stimuli in question. It is an impairment of the ability to recognize using vision despite an otherwise normal visual acuity, visual field, color vision, language memory and brightness discrimination (Kumar, 2023). One of the earliest experiments was observed by Munk, who described some dogs who avoided some objects in the environment although they could not recognize it. This dog had partial ablations of its left and right occipital lobe (Barton, 1999; Biran 2003). The term Agnosia was however introduced in 1891 by Freud of which he noted that this failure of visual recognition was not due to a poor acuity of vision (Baton,1999). It is a problem of the ventral stream of visual processing and a problem of recognition restricted to the realm of vision (Barton, 1999; Tranel, 2001). Patients with visual agnosia usually have deficits in both retrograde and anterograde memories (Tranel, 2001).

It is usually categorized as general or selective visual agnosia. In the former, every object is affected, and sufferers make gross problems with identification. In the latter however, only some specific objects are affected. Broadly speaking, general agnosia could be distinguished into associative, where affected individuals find it difficult to link what they see with the knowledge stored about the object, and apperceptive form where objects are not perceived. This distinction was done by Lissauer. Other subdivisions of general agnosia include integrative agnosia, form agnosia, and transformational agnosia. Selective agnosia is divided broadly into topographic agnosia, prosopagnosia, and pure alexia. Other subdivisions include: apperceptive, amnesic, associative, landmark agnosia among others. Further subtypes of visual agnosia include those affecting the face, objects amongst others (Humphrey, 2013). Several parameters help in object recognition besides visual form, this includes the role of color and other surface characteristics (Righi, 2018). When the brain finds it difficult to produce a single coherent image from the visual characteristics of the object

presented, it is called apperceptive visual agnosia. On the other hand, if the image cannot be associated with any familiar thing, it is called associative visual agnosia (Perez, 2021).

The ventral cortical pathway that determines the ‘what’ play a vital role in object recognition, whereas the dorsal ‘where’ pathways are involved in motion processing and actions necessary for visual processing (Huberly, 2012). Thus, pathologies involving the ventral pathways can result in visual agnosia. These pathologies could range from a wide range of neurological disorders including stroke, Parkinson disease, traumatic brain injury amongst others. There could be involvement of the ventromedial occipito-temporal cortex including the lingual and fusiform gyrus, with preservation of color and contrast analysis in the pathological process (Huberly, 2012). The dominant hemisphere for facial recognition is the right hemisphere (Haque 2018). There are separate processing of texture and form on the ventral stream with evidence from fMRI and visual agnosia (Carvena-Pratesi, 2010). Furthermore, patients with visual agnosias presents usually with ventral pathway damage and a dissociation between vision and biologic motion is suggested (Huberle, 2012). On a general note, only one sense is affected for example vision, touch or hearing in agnosia (Righi, 2018).

Patients with visual agnosia usually have no deficits with elementary neurosensory functions nor do they have impairment of their global cognitive function (Carlesimo, 1998). Regarding the clinic- anatomical description of visual agnosia, the lateral occipital complex is involved in object recognition, while facial recognition areas include the fusiform face area, an occipital face area in the inferior occipital cortex, which is more posteriorly located and just anterior to V4. These areas are the main facial recognition system, although other areas such as amygdala, ventral anterior temporal lobe may be involved (Haque, 2018). Lesions involving the posterior cerebral arteries destroy the left medial occipital area, corpus callosum and inferior longitudinal fascicle in visual agnosia patients, lateral part of the occipital lobe with or without the ventral part of the temporal lobe (Oliver, 2012; Humphrey, 2013). Visual agnosias are said to be more common in posterior cerebral artery infarcts than reported (McCarthy, 1986).

In a case report study on associative visual agnosia involving a patient with ischemic stroke, that developed significant visual agnosia with associated color anomia, amnesia, and alexia with agraphia, the area of involvement was in the left posterior cerebral artery territory. It was concluded that the cortical locus of deficit was a disconnection between the normal functioning visual memory storage and the semantic system and that his deficits in visual facial processing was in parallel to his

anomaly of recognition of the subject categories (Carlesimo, 1998). Also, in a documented case study of a patient named John, who had perceptual difficulties including face processing, color, motion, and depth following post stroke visual agnosia (Humphrey, 2013).

In another case report by Charnallet et al. (2008) on associative visual agnosia, where a right-handed man named JPM presented with visual agnosia following cerebral anoxia from cardiac arrest. Results revealed severe constitutional apraxia, deficits in executing symbolic gestures and mild impairment of memory. He had mild difficulty finding words but fluent skills. Although the MRI was normal, result from tests conducted showed he had severe impairment of object recognition in all categories of visual stimuli. The visual recognition for inanimate objects was 7/48, 3/48 for living objects, 4/45 of popular faces, 3/22 of buildings that are famous. The visual perception was normal in all the five tasks assigned, and regarding nonvisual input, the access to semantic knowledge from other categories were better preserved, with a score of 48/52 for the verbal form and a normal range for sound. It was concluded that based on the current model of semantic memory, an anomaly of the stored structural descriptions would give meaning to JPM's complete visual agnosia and why visual knowledge was absent (Charnallet et al., 2008).

In a single case study of a visual agnosia patient with language, visual, spatial, and perceptual abilities being well preserved but still had difficulties recognizing visually presented objects. A conclusion that the left hemisphere is critical in recognizing meaning of objects that are common was drawn (McCarthy, 1986).

Visual agnosia could sometimes be both apperceptive and associative depending on the area involved and is usually because of impairment of higher processing functions (Baugh, 2010). This was described in a case study by Shelton et al. (1994). It was revealed that a 66-year-old man with bilateral infarction of the occipital and inferior temporal association cortex, and sparing the primary visual cortex, had a visual agnosia resulting in an impairment of his visual recognition. While his gesture recognition impairment seems to be due to associative agnosia, his defective object recognition abilities was related to apperceptive agnosia. Results made suggestions that although internal representations were normal, he had inability to form adequate perceptual representations (Shelton, 1994). It may not necessarily affect every aspect of visual stimuli but some categories, for example, face, color, environment amongst others (Berti et al., n.d.). Four subdivisions of

apperceptive visual agnosia were proposed viz: apperceptive agnosia, dorsal simultagnosia, ventral simultagnosia and perceptual categorization deficit (Sydney Cognitive Development Centre).

A case report on DJ, who had many task impairments. had both naming and gesture deficits but otherwise normal object processing in other modalities. There were no impairments in stored structural descriptions and patients could build new visual representations (Fery, 2003).

Also, a case of JS, who had visual form agnosia after a stroke. The fusiform and lingual gyrus adjacent to the cingulate gyrus was damaged in the two hemispheres. It showed dissociation between the visual perception of orientation and shape on one side and preserved between visual perception of orientation and shape on one side (Karnath, 2009).

A case was described of a 70-year-old right-handed man, who complained of visual problems and headache. He had no history of paralysis nor sensory disorder. He however had homonymous hemianopia, disorder of memory, slight aphasia, and visual agnosia. He had fluent language and had only slight difficulty with finding words but could have daily conversations well. He had a short- term memory disorder and impaired visual perception. He could not explain the use of objects nor name them. He could only recognize objects on the table with touch, could eat independently and maintained personal hygiene (Tanak, 2021).

As earlier mentioned, varying neuro-ophthalmic pathologies could result in visual agnosia depending on the area involved and this could occur at any age, involving both adults and children. However, when this occurs in children it is often labelled as cerebral visual impairment. van Iterson et al. (2021) in a case study on a 6-year-old boy who developed inability to recognize familiar faces and in reading numbers and letters following seizures. With the use of a neuro-psychological test and an electroencephalogram which was done concurrently during his treatment period. Results revealed no clinical seizures during period of treatment, normal intelligence, normal to high level for retention and learning, but severe difficulties with visual memory which was persistent (van Iterson et al., 2021). Selective visual perception abnormalities such as visual agnosia could result from a damage to the brain with preservation of other visual functions (Matha, 1990).

In a patient with visual agnosia, after an acute hypertensive episode, a residual visual abnormality was noticed by a patient. The patient had homonymous hemianopia as the only neurological deficits

asides changes in behavior. He had writing difficulties although he could read. Color agnosia, prosopagnosia and object recognition difficulties were the other abnormalities (Rogers, 2021).

A man with brain damage who had inability to appreciate the nature of objects, presents vision problems despite being able to match, draw and see the objects. Neurophysiological mechanisms were responsible viz: specific defect of categorization of visual non-verbal stimuli and inter hemispheric visual verbal disconnection (Silverberg, 1975).

Although visual agnosia may be rare, many cases are usually left undiagnosed or sometimes misdiagnosed for other anomalies, making the sufferers far from receiving optimal care. A study on the patterns of presentation of visual agnosia amongst patients with neurological diseases would help to recognize and manage this condition more effectively. Although the medical community has agreements on the cortical areas and neuronal circuits involved in vision processing, more studies utilizing functional neuro imaging techniques would give further details (Bermann, 2010).

AIM

To determine the presentation patterns for visual agnosia amongst sufferers in patients with neurological diseases.

OBJECTIVES

To determine the different patterns of presentation of visual agnosia amongst patients with neurological diseases

To determine the areas of the brain involved in this anomaly.

To determine other associated neurological signs and symptoms in this condition
To determine the types of neurological diseases presenting with visual agnosia.

METHODOLOGY

STUDY LOCATION

This study was carried out at the department of ophthalmology and medicine of the Lagos State University Teaching Hospital Annex, Gbagada general hospital, Lagos, Nigeria. This is a Lagos state owned general hospital that serves as an annex to the Lagos State University Teaching Hospital, Lagos.

STUDY DESIGN

A hospital based observational case series.

STUDY POPULATION

The study was conducted amongst eight patients with visual agnosia visiting the ophthalmology, General Outpatient Department (GOPD) and the Medical Outpatient Department (MOPD) from the onset of the study till the end of the study period. See Ethics committee approval in the annex.

STUDY DURATION

This study was carried out for a duration of three months from the onset of the study.

SAMPLING TECHNIQUE

A purposive sampling technique was done, using the eligible participants during the period of study. All neurological patients with visual agnosia on follow up or new cases visiting the neurology clinic during the period of study were recruited if eligible and willing to participate in the study. Eight eligible participants were recruited and fully studied.

SAMPLE SIZE DETERMINATION

A total population of neurological patients with visual agnosia that are eligible and willing to participate in the study during the study period were recruited into the study.

SELECTIVE CRITERIA

The inclusion criteria were the following: patients must be 18 years of age or above, they must have visual agnosia and give consent for the study.

The exclusion criteria were the following: patients unwilling to participate in the study, unconscious patients and patients unable to communicate in writing.

ETHICAL CONSIDERATION

Ethical approval was sort and obtained from the Health Ethics and Research Committee of Lagos State University Teaching Hospital. After explaining the nature of the study to the participants, a written informed consent will thereafter be obtained from the participants.

STUDY MATERIALS

Pen torch, writing materials, Snellen's chart for distance, Illiterate E chart, occluder, pinhole, trial frame, patients consent form, mydriatic eye drop, 78D, and slit lamp biomicroscope.

STUDY PROCEDURE

All neurological patients with visual agnosia during the study period who are willing and eligible to participate in the study. The case notes for the patients were checked to confirm eligibility. All eligible patients were given the informed consent. A questionnaire was administered to the recruited participants. History taking, general examination and neurological exam was done using the questionnaire as a template. All necessary additional information were obtained from the case note. Information on the imaging done to show the area of the brain affected, the neurological condition the patient is being managed for, other associated neurological symptoms were also obtained. Additional ocular features associated with the visual agnosia and the type of visual agnosia were noted.

General examination was done for the participants noting their gait, speech pattern amongst others.

Neurological examination was done, noting the activity of the 12 cranial nerves, consciousness and alertness, orientation in time, place, and person as well as the tests for muscle bulk, tone, power and reflexes.

Mental state examination was done noting the cognition, memory affect/mood amongst others.

Psychoanalytical test was done noting their ability to draw a circle, match two similar shapes, respond to a gesture and to name a familiar object.

Ocular examination was done noting the visual acuity, anterior and posterior segment examination. Other adjunctive tests such as light brightness test, colour vision test and depth perception would also be done.

Neurological examination was also done, noting the activity of the 12 cranial nerves, the mental state examination, orientation, as well as the tests for muscle bulk, tone, power, and reflexes.

DATA COLLECTION AND ANALYSIS

Data was entered and analyzed using IBM SPSS version 25 software. The tables were drawn using SPSS and the frequencies and percentages of the parameters were obtained.

RESULTS

Below are tables showing the summary of case reports and the positive findings by each of the eight studies.

CASE REPORTS

Table 1. Description of cases

CA SE	CLINICAL HISTORY	EXAMINATION	MENTAL EVALUATION	PSYCOANALYTICAL TESTS	VISUAL FIELD	IMAGING
CA SE1	A 62-year-old male who presented with visual blurring, tremors, involuntary body movement, and has difficulty recognizing people	Hypertonia and hyperreflexia globally, cranial nerves 2 and 8 affected	Fair cognition, judgement and insight, good affect, thought, attitude, appearance and speech and motor activity	Could only understand gestures partially	Binasal hemianopia	No abnormality detected
CA SE2	A 78-year-old male who presented with blurring of vision worse in the left eye, generalized body weakness, inability to walk, involuntary muscle movement and has inability to recognize people	Stooped posture, poor speech, not oriented in time and place but oriented in person, hypertonia, and hyperreflexia globally, and cranial nerve 1,2,5,8 affected	Poor thoughts and perception, poor cognition, poor attitude, poor memory and poor speech and motor activities, fair appearance	Could not draw a circle, could not match objects, could not understand gestures and could only name objects partially	Generalized depression with peripheral constriction in the right eye, and total scotoma in the left eye	Bilateral periventricular T2/Flair signal lesions, confluent and partially confluent configuration suggestive of Fazekas grade 2/3 white matter lesion in the right hemisphere. The basal ganglia show blurring of the globus pallidus suggestive of iron related dystrophic changes.

CA SE3	A 78-year-old woman who presented with visual blurring and has difficulty recognizing objects	Cranial nerve2 involvement	Poor attitude, fair mood, good judgement and insight, cognition, appearance, memory, speech and motor activity.	Could only partially draw a circle	Homonymous Hemianopia	Large acute non-hemorrhagic infarct involving the temporal lobe, adjacent corpus callosum. Age related cerebral atrophy mild small vessel ischemic changes. Ischemic changes in supratentorial white matter.
CA SE4	A 72-year-old who presented with visual blurring and has difficulty recognizing faces	Hemiplegic gait, slurred speech and left sided weakness. Power is reduced on the left compared to right, left hypotonia and hyporeflexia	Poor mood, appearance, speech and motor activities, fair cognition and memory and good attitude, judgement and insight,	Cannot draw a circle, cannot match	Homonymous quadrant anopia	Large infarct in the occipital and temporal
CA SE5	A 45-year-old female who presented with inability to recognize inanimate things associated with difficulty naming things.	Cranial nerve2 involvement	Poor memory, good mood, thoughts, perception, attitude, judgement and insight and speech and motor activities	Difficulty naming objects	Superior altitudinal scotoma bilaterally	Extensive cerebral white matter, thalamic, cerebellar T2 flair hyperintensity with mild atrophy of right cerebral peduncle and the midbrain and right
CA SE6	A 54-year-old who presented with sudden onset of left droopy eyelid with involuntary eye movement months after road traffic accident. Has associated difficulty	Slurred speech, cranial nerves 2,3,5,6,7 palsy.	Poor thoughts and perception, cognition, judgement and insight, memory and speech and motor activities fair mood and appearance	Can only partially draw a circle, does not understand gestures, finds it difficult	Generalized depression	No abnormality detected

	recognizing inanimate things and difficulty naming things			naming objects		
CA SE7	A 51-year-old with visual blurring and painful eye movements. He has associated difficulty recognizing people.	Cranial nerve 2 involvement	General appearance, mood, thoughts and perception. Attitude, judgment and insight, memory, speech and motor activities all good	Can only partially name objects	Right superior altitudinal scotoma and left temporal hemianopia	Few areas of subcortical small vessel ischemic changes noted
CA SE8	A 78-year-old who presented with multiple areas of paresthesia and associated dizziness. Ocular complaint was poor vision. Has associated difficulty recognizing people	Hemiplegic gait, slightly slurred speech and left sided weakness. Power is reduced on the left, left hypotonia and hyporeflexia. Cranial nerves 2,5,7 involvement	General appearance fair, good mood, thought and perception, cognition, and judgement and insight	Cannot draw a circle	Right hemianopia, left total scotoma	Multiple areas of infarction involving the right parietal and occipital lobes

ANALYSIS OF RESULTS

Table 2. Age and sex distribution of the cases

VARIABLES	FREQUENCY	PERCENT (%)
Age group (years)		
18-40	0	0.0
41-60	3	37.5
>60	5	62.5
Sex		
Male	3	37.5
Female	5	62.5

Majority were of the age group greater than 60 and majority were females.

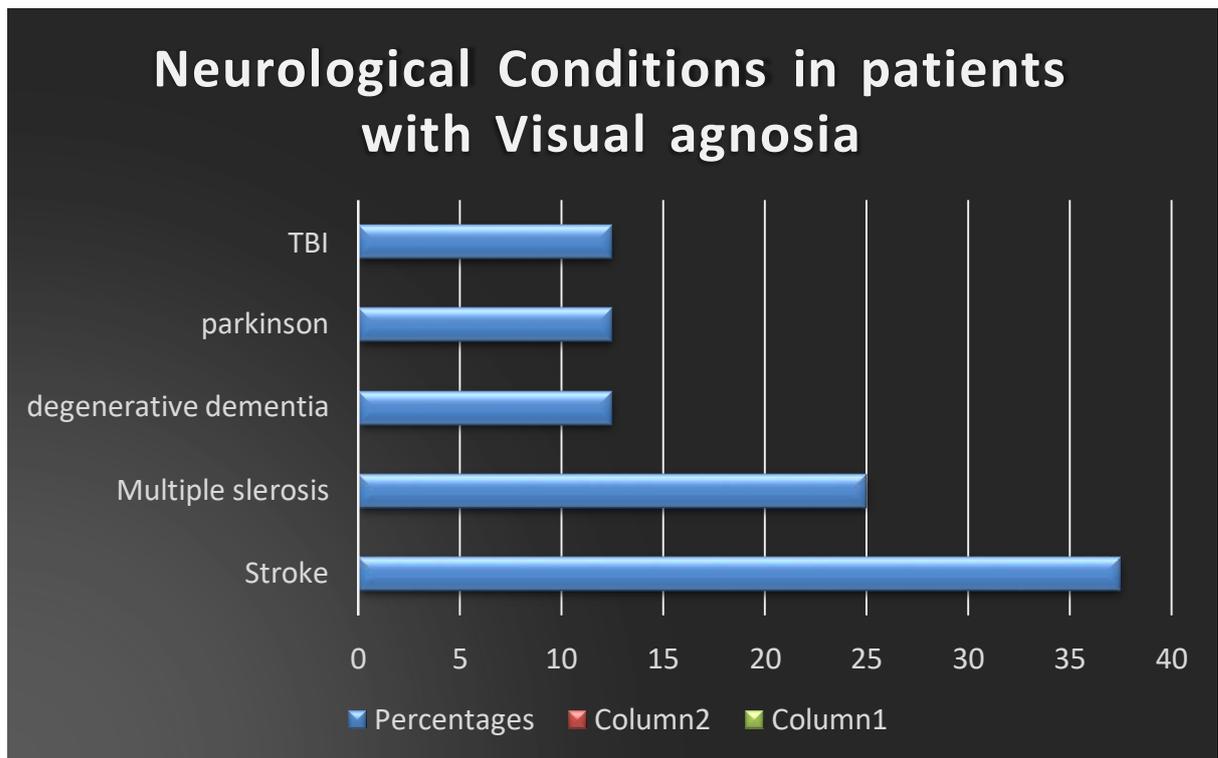


Figure 1. Neurological diagnosis of the patients with visual agnosia

Figure 1 shows the percentage of each neurological condition presenting with visual agnosia. Most of the cases 3(37.5%) had stroke, followed by multiple sclerosis in 2(25%) and the others including Parkinson's disease, degenerative dementia and traumatic brain injury seen at the same frequency of 1(12.5%).

Clinical presentation among cases

The most common ocular presentation by the participants was visual blurring seen in 5 (62.5%) cases followed by dropping of the eyelid and redness of the eye seen in 1 (12.5%) case each. The most common associated general complaint was headache, seen in 6 (75.0%), followed by memory loss and generalized weakness in 1 (12.5%) case each. Most of the patients 6 (75.0%) had difficulty with drawing or copying objects, 5 (62.5%) had difficulty in recognizing places, 1 (12.5%) had difficulty in recognizing color while none had difficulty in recognizing landmark places. Three of the participants (37.5%) had difficulty in recognizing places, while 5 (62.5%) had difficulty in using vision as a guide. Three of the participants had neglect for one part of the body, while all the participants could recognize objects by their sound, taste, or smell. Two of the participants (25.0%) had associated visual hallucinations. Other associated neurological symptoms include tremors in 4 (50.0%), as well as slurred speech, memory loss, muscle palsy, involuntary eye movement seen in a case each.

Table 3. Ocular Examination. Findings among cases

VARIABLES	FREQUENCY	PERCENT (%)
Visual acuity right eye		
<6/36	4	50.0
6/36	3	37.5
>6/36	1	12.5
Visual acuity left eye		
<6/36	6	75.0
6/36	1	12.5
>6/36	1	12.5
Cranial nerve 3		
Full	6	75.0
Restricted	2	25.0

Cranial nerve 4

Full	8	100.0
Restricted	0	0.0

Cranial nerve 6

Full	7	87.5
Restricted	1	12.5

Strabismus (right eye)

Present	1	12.5
Absent	7	87.5

Strabismus (left eye)

Present	2	25.0
Absent	6	75.0

Gaze abnormalities

Present	4	50.0
Absent	4	50.0

Saccades

Normal	4	50.0
Abnormal	4	50.0

Smooth Pursuit

Normal	2	25.0
Abnormal	6	75.0

Nystagmus

Present	1	12.5
Absent	7	87.5

Diplopia

Present	0	0.0
Absent	8	100.0

The extra ocular muscle cranial nerve examination, as well as findings on strabismus, gaze abnormalities, saccades, smooth pursuit, nystagmus, and diplopia are all displayed. The commonest abnormalities are problems with smooth pursuit, saccades and gaze abnormalities.

Table 3. Ocular Examination Findings among cases (continuation)

VARIABLES	FREQUENCY	PERCENT (%)
Pupil		
Reactive	5	62.5
Sluggishly reactive	3	37.5
Pallor		
Present	7	87.5
Absent	1	12.5
Vessel calibre		
Normal	3	37.5
Attenuated	3	37.5
Dilated	2	25.0
Retinal		
Exudate		
Haemorrhage		
Atrophy		
Light brightness sensitivity		
Normal	3	25.0
Abnormal	5	75.0
Colour desaturation		
Normal	3	25.0
Abnormal	5	75.0
Depth Perception		
Normal	2	25.0
Abnormal	6	75.0

The examination findings of the pupil and posterior segment findings as well as the light brightness, colordessaturation and depth perception findings are shown. Majority of the participants have abnormal depth perception.

RESULT OF DATA ANALYSIS

The above data analysis results were obtained using SPSS version 25 and are displayed in frequencies and percentages.

DISCUSSION

Eight participants were recruited for the case series. Majority, 5 (62.5%) were over 60 years (as shown in Figure 1) and male to female ratio was 3:5.

With regards to the first objective which was to determine the patterns of presentation of visual agnosia, all had neurological pathologies, with three being due to stroke, two to multiple sclerosis, and one each to Parkinson's disease, degenerative dementia, and traumatic brain injury. They all presented with diverse neurological complaints except one, as well as visual complaints. Visual blurring, besides visual agnosia was the major ocular complaint. While all the participants had visual agnosia, it is important to note that none except one gave a complaint of visual agnosia. The other seven patients however had the history of visual agnosia elicited by directly asking about it. This emphasizes the role of directly asking about visual agnosia in patients with neurological diseases in order to allow for timely detection of this condition and ensure that the underlying cause is addressed adequately.

With regards to the second objective which is to determine the areas of the brain involved in this anomaly. The areas identified include occipital lobe, temporal lobe, parietal lobe, pons, thalamus, corpus callosum, cerebellum and globus pallidus. Two of the cases had normal findings on MRI. The neurological manifestations in each of the patients however differed. Two of the cases had affection of the occipital and temporal lobe. This is comparable to the work of Carlesimo et al. (1998) in a case of associative visual agnosia in an ischemic stroke patient, in which the left posterior cerebral artery territory was the area involved. However, other areas were identified in this study.

For the third objective, which is to determine the other associated neurological signs and symptom in this condition. The neurological manifestations varied depending on the neurological condition. One of the cases did not have any other associated neurological condition. Out of the seven others, the neurological signs varied from cranial nerve palsies, hemiparesis, gait abnormalities, tremors, gaze abnormalities, synkinesis, memory loss. Abnormalities in psychoanalytic tests such as gesture and naming abnormalities which is usually found in associative agnosia, as well as drawing abnormalities found in apperceptive

agnosia. One of the cases had features suggestive of both associative and apperceptive agnosia. The commonest cranial nerve abnormality was in the second cranial nerve (ophthalmic nerve), with all the participants having affection of this nerve. Seven with pale disc and one with disc edema. The gesture abnormality as well as the impairment of memory found is like that seen in the study by Charnallet et al. (2008), in which the affected individual had mild memory impairment and abnormalities in gesture execution.

Regarding the last objective, the neurological diseases noted were stroke, multiple sclerosis, degenerative dementia, and traumatic brain injury. Stroke was the most common neurological disorder amongst the participants followed by multiple sclerosis. The occurrence of visual agnosia in stroke is like that in the study by Carlesimo et al. (1998) involving a patient with ischemic stroke. However, unlike in the case with Loretta et al. (2008) in which the visual agnosia occurred following seizure, none of the participants recruited had a history of seizure.

The cases of stroke cases seen in case 4 were like findings by Shelton (1994) in terms of the area of involvement (occipital and temporal areas) but were different from those of Karnath (2009) in which the area of involvement was fusiform and lingual gyrus adjacent to the cingulate gyrus. Case 8 had affection of the occipital and parietal lobe. Other findings seen in the stroke cases were color abnormalities, amnesialike findings by Carlesimo et al. (1998) with amnesia, color anomia, alexia and agraphia.

Regarding the case of traumatic brain injury seen, it contrasts with the study by Silverberg et al in which the patient could not match and draw although he could see, as found in apperceptive visual agnosia. In this study however, the patient could match, could draw only partially, and could not understand gestures as seen in associative visual agnosia but also having some characteristics of apperceptive visual agnosia.

In contrast to McCarthy (1986) that said the left side of the brain seems critical in processing object recognition, three patients had affection of the right cerebral hemisphere instead, one in the left, one bilateral, one unspecified and the others had normal findings on MRI. This shows that both side of the brain might be responsible for object recognition processing, and handedness of the individual may also play a key role in this.

In terms of the presentation, the commonest general presentation noted was left sided weakness although majority had normal gait. The most common ocular presentation was visual blurring. The commonest neurological abnormality was cranial nerve palsy, with universal affection of cranial nerve two in all cases

seen. In terms of the visual field findings, the most common was homonymous hemianopia like findings by Rogers et al. (2021). Other patterns seen were one sided hemianopia, homonymous quadrantanopia amongst others. Four of the cases had associative visual agnosia, three had apperceptive while one had both. Mental state examination was good in half of the case and poor in the other half. Majority of the patients had abnormal depth perception. All the participants had abnormal psychoanalytical tests although it varied in severity and type.

LIMITATIONS

The history of visual agnosia had to be elicited from the patients presenting with a different pathology in all the cases except one. This is because none of the patients (except one) gave a history of visual agnosia and so could have been missed.

Some participants with visual agnosia were not stable enough to participate in the study.

FUTURE LINES OF RESEARCH

Future research in functional imaging of the brain might be needed in patients with visual agnosia to find out more precisely possible areas involved in this pathology.

RECOMMENDATIONS

All neurological patients should be asked about the occurrence of visual agnosia in order to allow for adequate diagnosis and management of this neurological presentation.

Psychological and vision support should be given to sufferers of visual agnosia in order to ensure the best possible outcome and quality of life for these individuals.

Clinical acumen should be heightened amongst clinicians in identifying the presence of visual agnosia even with the most subtle features in order to allow for early recognition of any neurological pathology present. This is particularly so if visual agnosia is the only neurological feature present.

CONCLUSION

Visual agnosia, although rare, may be a more common neurological feature than described. There are diverse presentation patterns for patients with visual agnosia and a direct history about visual agnosia and deliberate questions regarding visual agnosia must be asked from patients suffering neurological disease. This is because in the study involving eight cases, only one of the participants presented with a history of visual agnosia, it was however only elicited from the other seven participants by direct questioning. This study has revealed that visual agnosia can be found in a range of neurological conditions and may occur with (more commonly) or without other neurological features. Different brain areas are involved in the pathophysiology of visual agnosia and although both apperceptive and associative visual agnosia could occur, associative type seems to be more common.

ACKNOWLEDGEMENT

I would like to acknowledge God the giver of wisdom who has given us the grace to discern hidden things. I would like to acknowledge the support of my tutors that were tremendous in ensuring that I have a good grasp of the knowledge being instilled. To my dearest family, thank you for all you do.

REFERENCES

-
- Barton, J. S. Visual agnosia. (1999). *Neuro-Ophthalmology and Neuro-Otology* <https://www.medlink.com> Accessed 5/11/2022.
- Baugh, L. A., Desanghere, L., Marrotta, J. J. (2010). Agnosia. *Encyclopedia of Behavioral Neuroscience*. Elsevier Inc, 27-33 <https://experts.nebraska.edu> Accessed 10/06/2023.
- Behrmann, M., Mura, M. (2010) Agnosia. *WIREs Cognitive Science* 1(2), 203-213 <https://doi.org/10.1002/wcs.42>
- Benson, D. F., Segarra, J., & Albert, M. L. (1974). Visual agnosia-prosopagnosia. A clinicopathologic correlation. *Archives of neurology*, 30(4), 307–310. <https://doi.org/10.1001/archneur.1974.00490340035007>
- Berti, A., Neppi-Madon. Agnosia. <https://www.academia.edu/agnosia> Accessed 08/06/2023.
- Carlesimo, G., Casadio, P., Sabbadini, M., Calfagione, C. (1998). Associative Visual Agnosia: Results from a Disconnection between intact visual memory and Semantic systems. *I.R.C.C.S.*
- Cavina-Pratesi, C., Kentridge, R. W., Heywood, C. A., Milner, A. D. (2010). Separate Processing of Texture and form in the ventral stream: Evidence from fMRI and Visual Agnosia. *Cerebral Cortex*, 20(2), 433-446
- Charnallet, A., Carbonnel, S., David, D., & Moreaud, O. (2008). Associative visual agnosia: a case study. *Behavioural neurology*, 19(1-2), 41–44. <https://doi.org/10.1155/2008/241753>

- Fery, P., Morais, J. (2003). A case study of Visual Agnosia with Perceptual Processing or Structural Description impairment. *Cognitive Neuropsychology*. 20(7), 595-618
- Haque, S., Vaphiades, M. S., & Lueck, C. J. (2018). The Visual Agnosias and Related Disorders. *Journal of neuro-ophthalmology : the official journal of the North American Neuro-Ophthalmology Society*, 38(3), 379–392. <https://doi.org/10.1097/WNO.0000000000000556>
- Huberle, E., Rupek, P., Lappe, M., & Karnath, H. O. (2012). Perception of biological motion in visual agnosia. *Frontiers in behavioral neuroscience*, 6, 56. <https://doi.org/10.3389/fnbeh.2012.00056>
- Humphery, G., Goodale, M., Servos, P. (1994). The role of surface information in object recognition: studies of a visual form agnosia and normal subjects. *Sage Journals* 23(12).
- Humphrey, G., Riddoch J. (2013). A case study of Visual Agnosia Revisited. <https://doi.org/10.4324/978020355809>
Accessed 06/06/2023
- Karnath, H. O., Rüter, J., Mandler, A., & Himmelbach, M. (2009). The anatomy of object recognition-- visual form agnosia caused by medial occipitotemporal stroke. *The Journal of neuroscience : the official journal of the Society for Neuroscience*, 29(18), 5854–5862. <https://doi.org/10.1523/JNEUROSCI.5192-08.2009>
- Kumar, A., Wroten, M. (2023). Agnosia. National Library of Medicine.
- van Iterson, L., Vrij, S., Sie, L. T. L., Augustijn, Rooze, A. C. S., & Jansen, F. E. (2021). Acquired visual agnosia as an uncommon presentation of epileptic encephalopathy in a 6-year-old boy with CSWS. *Epilepsy & behavior reports*, 16, 100465. <https://doi.org/10.1016/j.ebr.2021.100465>
- Farah, M. J. (1990). *Visual Agnosia: Disorders of object recognition and what they tell us about normal vision*. MIT Press. <https://philpapers.org>
- Martin L. A., Silverberg, R., Reches, A., (1975). Associative Visual Agnosia with Alexia. *Neurology* 25(4), 322-326
- McCarthy, R. A., & Warrington, E. K. (1986). Visual associative agnosia: a clinico-anatomical study of a single case. *Journal of neurology, neurosurgery, and psychiatry*, 49(11), 1233–1240. <https://doi.org/10.1136/jnnp.49.11.1233>
- Oliver, M., Dorothee, P., Emmanuel, G., Meud, L., David, H., Didier, H. (2012). Visual Agnosia and Posterior Cerebral Artery Infarcts: An Anatomical-Clinical Study. *PLOS ONE*, 7(1), e30433.
- Perez, L. (2021). Visual Agnosia, seeing without recognition. *NeuronUP*. <https://neuronup.us>visual-agnosia>
- Righi, G., Tarr, M. (2018). Visual Agnosia. *Encyclopedia of Clinical Neuropsychology*, 1-4

Rogers, K., Young, G. (2021). Associative Visual Agnosia. Britannica. <https://www.britannica.com/science>
Accessed 09/06/2023.

Rubens, A. B., & Benson, D. F. (1971). Associative visual agnosia. *Archives of neurology*, 24(4), 305–316.
<https://doi.org/10.1001/archneur.1971.00480340037003>

Shelton, P. A., Bowers, D., Duara, R., Heilman, K. M. (1994). Apperceptive visual agnosia: A case study.
Brain and Cognition. 25(1), 1-23

Sydney Cognitive Development Centre. Agnosia. RESOURCES. <https://sdccenter.con/agnosia> Accessed
06/06/2023.

Tranel, D., Damasio, A. R. (2001). Visual Agnosia. *International Encyclopedia of the Social and Behavioral Sciences*.

Tanaka, M. (2021). Occupational Therapy in Integrative Visual Agnosia and Memory Disorder: A case report.
Asian Journal of Occupational Therapy, 17, 27-3