# A case of Balint syndrome: the importance of a specific neuropsychological appraisal in the clinical diagnosis of visuospatial disorders

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## Abstract

*Balint syndrome is characterized by a severe disturbance of visual spatial analysis including impaired oculomotor behaviour, optic ataxia, and simultanagnosia. The complete syndrome is relatively rare, and partial syndromes have been reported more frequently. The present study aims to describe a case of Balint syndrome who displayed all the three main neuropsychological features as a consequence of infarction in the watershed between the anterior and posterior cerebral artery territories. In this case report three days post stroke the clinical assessment showed a severe impairment in several visual spatial tasks (e.g., reading, writing, description of a visual scene, voluntary gaze-shift). Twelve weeks post-stroke the clinical assessment showed a significant improvement in reading, writing, as well as in verbal delayed recall processes, but only a mild improvement in visual spatial tasks like the description of a complex visual scene was registered. Balint's syndrome is rare and is not easy to assess with standard clinical tools. The classical neurological examination evaluates in detail the senses, motility, balance, and to some extent language, but, sometimes, it is much less concerned with*

*cognitive functions. The case discussed here is a good example of the need to emphasize that an acutely ill patient should also be accurately evaluated for the presence of cognitive and behavioural disturbances.*

*Keywords:* Balint; Ocular apraxia; Optic ataxia; Simultanagnosia; Visuospatial abilities; Stroke

## 1. Introduction

Balint syndrome is characterized by three main clinical signs: simultanagnosia (inability to perceive multiple items of a visual display), ocular apraxia (inability to shift gaze voluntarily to objects of interest despite unrestricted eye rotation), and optic ataxia (difficulty to reaching visual goals under visual guidance despite normal limb strength) (Balint, 1909). Progressive Balint syndrome may be found in Alzheimer's disease (Hof, Vogt, Bouras & Morrison, 1997) but more often the lesions are the result of infarctions in the border area between the anterior and the posterior cerebral artery territories (sudden and severe hypotension is the main cause) or is the consequence of multiple bilateral strokes (Ferro, 2001; Damasio, Tranel & Rizzo, 2000). Balint's syndrome can also be caused by bilateral metastases in the occipito-parietal region, trauma, prion disorder, and HIV infections. It derives from bilateral alteration of the parieto-occipital cortex junction (i.e., Brodmann areas 5, 7, 19), as well as the cingulum, the superior longitudinal and arcuate fasciculus.

Balint's syndrome represents the paradigm of the disorders of complex visual processing. It is not easy to be assessed only on the basis of history and standard clinical tools. In fact the classical neurological examination evaluates in detail the senses, motility, balance, and, to some extent, language, but, sometimes, it is much less concerned with cognitive functions, emotion and adaptive behaviour, especially in the context of acutely ill patients. Accordingly, the complete appraisal of these disorders should take into consideration neuropsychological, neuro-ophtalmological and neuroimaging tools.

The complete syndrome is relatively rare, and partial syndromes have been reported more frequently. In general, it should be distinguished from: (i) other complex visual deficits; (ii) disorders of spatially-directed attention; (iii) disorders of motility.

The disorders of complex visual processing are associated to the damage of the inferior parietal lobule and the posterior parietal gyrus, along to the U-shaped fibres that connect to visual areas of the temporal and the occipi-

tal lobes. In general, damage to these cross-modal zones produces defects in the ability to integrate memory, vision and proprioception. Patients are usually unable to resemble an object, event, or scene (i.e. disorders of mental imagery), and may experience a feeling of derealisation or out-of-body sensation (Ffytche, Blom & Catani, 2010).

The disorders of spatially-directed attention are related to lesions at the level of the right inferior parietal lobule. The clinical picture is characterized by a reduced awareness of parts of their body, peripersonal space or visual stimuli, mostly from the left side (Husain, Mattingley, Rorden, Kennard & Driver, 2000; Doricchi, Thiebaut de Schotten, Tomaiuolo & Bartolomeo, 2008; Olk, Hildebrandt & Kingstone, 2010; van Kessel, van Nes, Brouwer, Geurts & Fasotti, 2010).

The disorders of motility are related to the damage of the posterior parietal cortex (i.e. Brodmann areas 5, 7, 39, 40, 19) that is connected to the occipital lobe through U-shaped fibers, as well as to the frontal lobe through the arcuate and superior longitudinal fasciculus (Catani et al., 2012; Thiebaut de Schotten, Dell'Acqua, Valabregue & Catani, 2012; Yeterian, Pandya, Tomaiuolo & Petrides, 2012). At a first glance, patients seem to be clumsy and their voluntary movements are excessively slow and inaccurate (i.e. ataxia). The optic ataxia is solely the impairment in reaching for object (Shallice, Mussoni, D'Agostino & Skrap, 2010) and the lesion is located in the precuneus and posterior parietal gyrus (Karnath & Perenin, 2005). The extensive damage of parietal lobe is characterized by the inability to initiate and perform previously learned skilled movements involving the use of tools (i.e. limb kinetic apraxia). Apraxias are mostly related to left-sided lesions of the inferior parietal lobule (Glickstein & Berlucchi, 2008; Heilman & Watson, 2008). Oculomotor apraxia is the inability to disengage fixation to move gaze from one visual target to another and occur with dorsal parieto-occipital lesions.

In the present paper, we describe a case that displayed the three main features of the Balint's syndrome as a consequence of infarction in the watershed between the anterior and posterior cerebral artery territories. We followed the operational definition according with that given by Damasio, Tranel and Rizzo (2000): an acquired disturbance of the ability to perceive the visual field as a whole, resulting in the unpredictable perception and recognition of only parts of it (simultanagnosia). Moreover, there is an impairment of target pointing under visual guidance (optic ataxia) and an inability to shift gaze at will toward new visual stimuli (ocular apraxia).

## 2. Case report

An eighty-two year old right handed woman (F.G.) (five years of formal education) was admitted for acute onset visual difficulties. She had trouble to insert the key into the keyhole, to stick a fork into a potato or to open a door without slamming it in her face. Also, she complained severe difficulties in reading and writing. The activities of daily life, instrumental activities of daily life, mood/behaviour and visual capacities (driving a car, reading) were preserved till 24 hours before the admission. She suffered of mild hypertension. The MRI performed 7 days post-stroke revealed damage in the occipito-parietal junction; lesions were symmetrical with a slight left prevalence, and a mild involvement of the corresponding posterior white matter (*Figure 1*). An electroencephalogram showed sporadic train of low voltage theta at the posterior regions. Color flow doppler sonogram performed at the level of great vessels, echocardiogram, and EKG, were reported as normal.

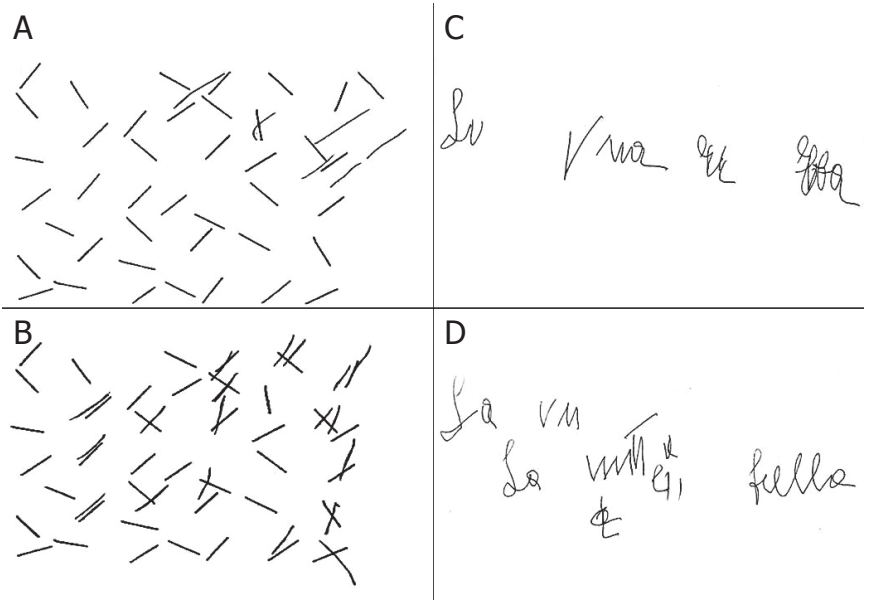


*Figure 1. Line cancellation task (A and B) and writing sentence (C and D)*

### 2.1. Phase 1 assessment

During the examination, she was collaborative, and aware of and upset by her difficulties. She was oriented to season, day of week, town, place, and person, but not to the year and to the month. On the cranial nerves examination she showed severe limitation of gaze in all directions. No grossly visual field defects were detected by confrontation method (by two neurologist, separately); also, somatosensory perception, muscle strength, and tone were normal. Tendon stretch reflexes were normal. Babinski's sign was absent. The gait was excessively slow and cautious. An ophtalmoscopic evaluation did not show pathological conditions of the retinae, vessels, and optic disc.

In the visual domain, F.G. had severe difficulties on all tests involving visuo-spatial, ocular motor and visuomotor abilities; performances concerning visual scanning, constructional praxis, gestural praxis, and visual guided reaching were particularly compromised. She could identify several geometric, two-dimensional shapes only when they were presented each at a time. In the case of geometric three-dimension or uncommon complex shapes (especially large size ones), they were systematically not identified. Counting of visual dots was impossible, even for few numbers of items.

Performance on Line Cancellation task was very poor showing lines misalignment and marked avoidance of the left and the bottom side of the page (*Figure 2A*). She was unable to perform more complex cancellation tasks (i.e. condition containing letters and shapes like in bells test or in single letter cancellation test). Single letters reading was preserved, but bi-sillabic/three-sillabic words reading or text reading was impossible. Writing revealed very severe spatial dysgraphia (with numbers and letters markedly incomplete and misaligned during spontaneous writing) (*Figure 2B*), and spatial calculation abilities were grossly impaired as well. She was unable to draw a clock (*Figure 3*). Face recognition was normal.

Perception of colours was preserved. Oral speech and comprehension were normal. Arithmetic skills were moderately defective. Verbal learning functions were within normal range as delayed recall. No significant difficulties in recognizing and naming in Sartori naming test. The neuropsychological tests scores summary was shown in *Table 1*.

She was unable to accurately point and reach visual targets located in the space near her body (she was asked to touch three small boxes located on a desk, either aligned or deviated 60° to the right or left of the midline). She deviated several centimetres away from the target, especially on the left side. Also, pointing with left hand was markedly worse than with the right hand.

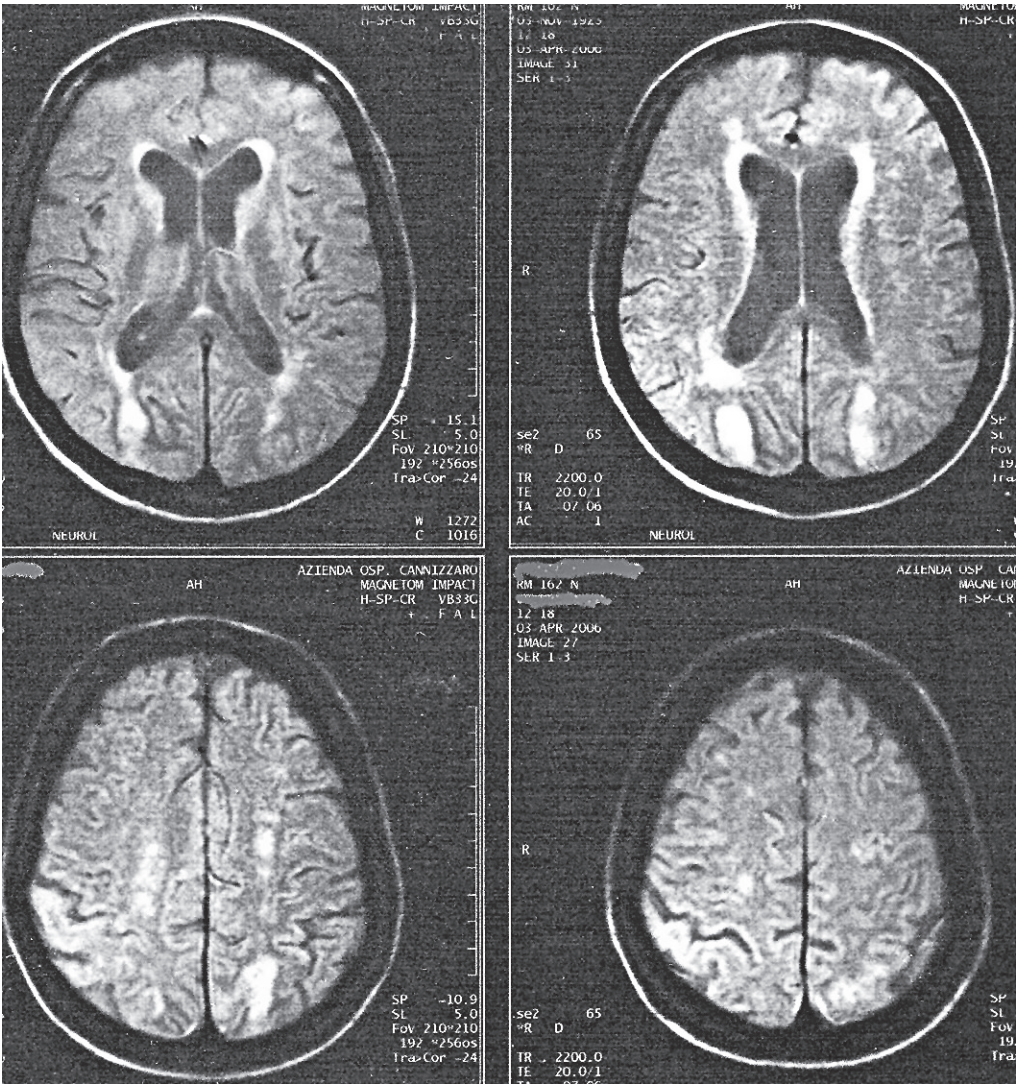


*Figure 2. Axial T1-weighted MR scan showed cerebral infarcts in both parieto-occipital areas along the middle and posterior cerebral arteries watershed zone seen as hyperintensity foci*

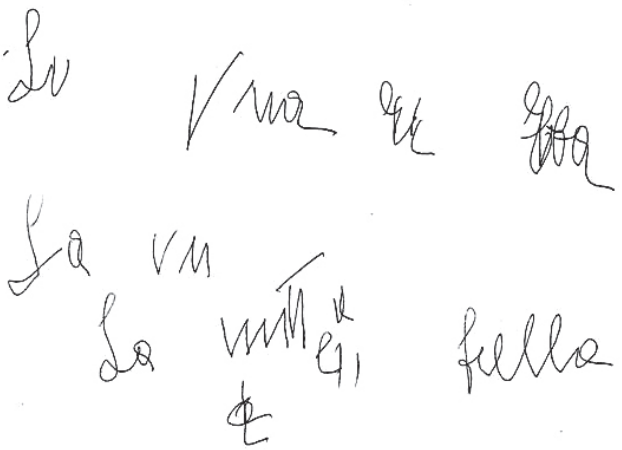

*Figure 3. Spontaneous writing on the third day after stroke (A) and 12 weeks later (B), respectively. She wrote "La vita è bella" (Life is beautiful)*

*Table 1*

| | 1 WEEK AFTER STROKE | 12 WEEKS AFTER STROKE | |
|---|---|---|---|
| | *Corrected score* | *Corrected score* | *Cut-off* |
| MMSE | 18.85 | 19.85 | 24 |
| Trail-Making Test A | N.A. | N.A. | |
| | Digit span | | |
| Forward | 3.75 | 4.75 | 3.75 |
| Backward | 2 | 3 | |
| Geriatric Depression Scale | 6 | 6 | |
| *Verbal Memory, 15 Words (Rey, 1958)* | | | |
| Immediate Recall | 31.6 | 33.6 | 28.53 |
| Delayed Recall | 5.97 | 6.97 | 4.69 |
| *Babcock Story Recall Test (Carlesimo, 2002)* | | | |
| Immediate | 3.6 | 6.6 | 3.10 |
| Delayed | 2.50 | 4.50 | 2.39 |
| *Verbal Fluency* | | | |
| A + F + S | 11.3 | 16.3 | 17.3 |
| Fruits + Animals + Colors + Towns | 8 | 10 | 7.25 |
| Clock Drawing Test | 0/5 | 0/5 | 3 |
| Sartori Naming Test | 45 | 53 | Normal Range (50-56) |
| Efron Test | 13 | 16 | 16.5 |
| Line Cancellation Task | 1 | 11 | 38 |
| Bells Test | N.A. | N.A. | |

On the other hand, the patient did not show any primary motor deficit: elementary movements were executed correctly, as well as reaching movements under proprioceptive control. In fact, she had no difficulty pointing with precision to targets in her own body or garments using somatosensory information as well as no difficulty pointing to the source of sounds.

Due to ocular apraxia, her ability to direct gaze voluntarily toward a new stimulus in the periphery of the visual fields was heavily impaired, even when alerted verbally that the examinator's finger had indeed entered her visual field (she promptly reported the appearance of the stimulus, but was not able to produce the saccade toward it).

In summary, F.G. showed a severe impairment in several visual-spatial tasks depending on right or left parietal lobe functions, whereas her perceptual visual recognition abilities were relatively spared.

### *2.2. Phase 2 assessment*

A new neuropsychological testing was performed at 12 weeks after stroke. It revealed a significant improvement in reading and writing performances (*Figure 2D*), as well as in verbal recall functions (especially in delayed recall); whilst, only a mild improvement in several tasks assessing visual-spatial abilities was registered.

A relative improvement in line-crossing test was registered: on one hand, only lines on the extreme left and bottom were left uncrossed; on the other hand, several lines misalignment were still present, reflecting the impairment for reaching objects under visual guidance (i.e., optic ataxia) (*Figure 2C*). She failed when the examiner asked to point in the direction of any object within the range of vision and to estimate the distance from her by sight alone.

She was able to perform a letter cancellation task, but she was very slow and inaccurate; moreover, she was unable to detect the target letter "A" if its size was significantly larger than the others (*Figure 4*). In addition, the description of the Cookie Theft picture confirmed a severe impairment in the analysis of a visual scene (i.e., simultanagnosia); in particular, after a prolonged exposition, she was able to detect only few objects, and was absolutely unable to describe the picture meaning. She complained that an object that was clearly seen at a given moment might suddenly vanish from view in a few seconds.

Finally, the difficulties to direct gaze voluntarily toward a new stimulus in the periphery of the visual fields were unchanged, indicating a severe ocular apraxia.

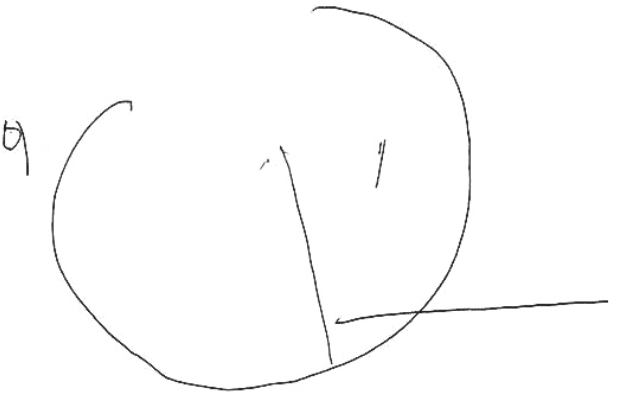

*Figure 4. Clock drawing test. The drawing was largely incomplete, and also showed a marked misalignement of lines and numbers (one week after stroke)*

## 3. Discussion

The main relevance of the report is that all core diagnosis criteria of the syndrome are fulfilled, without any grossly ocular and visual field defects.

Furthermore, our study underlines that the classical neurological examination is scarcely concerned with cognitive functions; therefore, a comprehensive neurological examination should also include a cognitive assessment, even in the context of acutely ill patients.

Our case clearly had a Balint syndrome caused by infarction in the border area between the anterior and the posterior cerebral artery territories (occipito-parietal region). Probably, the physiopathologic mechanism was a sudden and severe episodic hypotension during the nocturnal sleep.

Below we discuss the case, taking into consideration each symptom (i.e., simultanagnosia, optica ataxia, ocular apraxia), according to the data of the literature.

Simultanagnosia is a disorder characterized by an inability to see more than one object at a time. Farah, Brunn, Wong, Wallace and Carpenter (1990) distinguished between "dorsal" and "ventral" simultanagnosia. It has been suggested that patients with Balint syndrome have "dorsal" simultanagnosia with spatial-temporal analysis disturbances due to an impairment of attentional mechanisms. It is hypothesised that visuospatial and temporal attention depend on the integrity of a circuit including occipital areas, con-

nections with temporal and parietal lobes, frontal eyes fields, and prefrontal cortices. Differently, patients with "ventral" simultanagnosia have left occipito-temporal lesions with slowed visual processing speed, causing a difficulty in simultaneously recognising the single item of a scene. These latter patients perform usually better than those affected by dorsal simultanagnosia in describing all the elements of a visual scene, but, similarly to the formers, they are not able to report the meaning of the scene.

Our patient suffered from dorsal simultanagnosia. In the Cookie Theft picture she correctly reported only few items, failed to describe the meaning of the figure, even after a prolonged exposition. In the same line, in the *target letter test*, she was unable to detect the target letter "A" if its size was significantly larger than the others.

She was unable to judge object location, distance, orientation, size or motion. Aphasia and agnosia were excluded: she was able to recognize several common objects that were presented one each time.

On neuroimage, the frontal and temporal lobes, the cerebellum and the basal ganglia did not show significant focal lesions. So, the bilateral occipito-parietal damage seems to be responsible of a "dorsal" simultanagnosia.

Optic ataxia is characterized by an impaired visual control of the direction of arm reaching to a visual target, accompanied by defective hand orientation and grip formation. In humans, optic ataxia is associated with lesions of the Superior Parietal Lobule (SPL), which also affect visually guided saccades and other forms of eye-hand coordination. In today's literature, optic ataxia is regarded as an independent disorder, since it can occur in the absence of the other signs of the parietal syndrome (Damasio & Benton, 1979). A constant feature is the site of the lesion that mainly involves the SPL, at the parieto-occipital junction (Vallar & Perani, 1986). This is an important distinction from hemispatial neglect, which usually has been associated with lesions of the inferior parietal lobule (Vallar & Perani, 1986) and of the temporo-parietal-occipital junction (Leibovitch et al., 1998). Noteworthy, there are reports that open new avenues concerning the anatomical substrates of optic ataxia; it has been showed after a left thalamic haemorrhage affecting the most posterior fibres of the caudal limb of the internal capsule, and associated by a metabolic depression involving the SPL (Classen et al., 1995). The author hypothesis pointed to a potential role for parieto-ponto-cerebellar circuits as critical for the visual control of the limb, thus questioning the view of optic ataxia as an exclusive consequence of cortico-cortical disconnection. Nowadays it is hypothesized that in patients with optic ataxia the directional errors that characterize reaching are a consequence of the breakdown of the combination of directional eye and hand information within the global tuning fields of parietal neurones. This

breakdown could be dependent on the failure of a re-entrant fronto-parietal signalling as a consequence of the lesion of the cortico-cortical systems linking parietal and frontal lobes (Battaglia-Mayer & Caminiti, 2002).

In our case, optic ataxia was demonstrated by the patient's failure pointing and reaching boxes placed on the desk, whilst she had no difficulty in naming the same objects and pointing to targets in her own body or garments as well as no difficulty pointing to the source of sounds. The site of ischaemic lesion affecting the pariet-occipital junction was congruous with most of the literature reports.

Bilateral posterior hemispheric lesions, affecting the posterior part of the parietal lobe, result in eye movement disorders consisting of the absence or severe impairment of foveal smooth pursuit and opto-kineticus nistagmus, and severe disturbance of visually guided saccades, with preservation of some intentional saccades, such as saccades made on verbal commands (Pierrot-Deseilligny, Gray & Brunet, 1986). These disorders could result from damage both the parietal eyes field, at the superior part of the angular gyrus, and to medial superior temporal area and in the adjacent temporal cortex (Pierrot-Deseilligny, 1994). With bilateral lesions affecting both this posterior hemispheric cerebral region and the posterior part of the frontal lobe, a severe syndrome is observed (Shallice et al., 2010); such a patient has great fixity of gaze, spontaneous saccades are very rare, and intentional saccades made on verbal commands or visually guided saccades are either absent or performed with great difficulty. Foveal smooth pursuit and optokinetic nystagmus are also absent. These eye movements' abnormalities have been termed "acquired ocular motor apraxia" (Pierrot-Deseilligny, Gautier & Loron, 1988).

Ocular apraxia was evinced in several conditions: spontaneous saccades were extremely rare. Reflexive saccades were absent; she was unable to direct gaze toward a new stimulus (i.e. examinator's finger) that has appeared in the periphery of the visual field. Intentional visually guided saccades were severely impaired; she was unable to produce the saccade toward the stimulus even when verbally alerted that it has indeed entered her visual field. Also, foveal smooth pursuit and optokinetic nystagmus were absent. She failed tests of visual scanning, such as counting dots, and searching letters or shapes.

In our patient the MRI showed bilateral infarcts in the occipito-parietal junction without frontal eyes field and medial superior temporal area involvement. These latter areas are also usually reported to be damaged in ocular motor apraxia (Pierrot-Deseilligny, Gaymard, Müri & Rivaud, 1997). So, it seems that bilateral damage to the posterior parietal region might be sufficient in itself in the determining of this ocular movement disturbance.

Furthermore, also according to the ischaemic nature of the brain damage, it is noteworthy the discrete recovery after time, especially at the

level of reading, writing, verbal memory recall, and spatial orientation of attention abilities; whilst, the distinctive clinical profile of the syndrome is unchanged.

## 4. Conclusions

To conclude, the main features in this patient are acute onset of difficulties with visual spatial analysis and reaching objects under visual guidance. This picture is broadly compatible with Balint's syndrome due to a bilateral infarction in the border zone between the anterior and the posterior cerebral artery territories, as demonstrated by magnetic resonance images.

Balint's syndrome is rare and is not easy to assess with standard clinical tools. In fact, the classical neurological examination evaluates in detail the senses, motility, balance, and to some extent language, but, sometimes, it is much less concerned with cognitive functions, emotion and adaptive behaviour. Accordingly, the complete appraisal of this disorder should take into consideration neuropsychological, neuro-ophtalmological and neuroimaging tools. In particular, the neuropsychological approach was crucial in defining the clinical picture. For instance, the simultanagnosia was specifically detected after administering both the Cookie Theft picture and the target letter test.

The case discussed here is a good example of the need to emphasize that acutely ill patients should also be accurately evaluated for the presence of cognitive and behavioural disturbances. From the functional point of view, this very peculiar cognitive syndrome, is responsible of a remarkable disability, in spite of the fact that primary sensory-motor functions and several "metacognitive" abilities are discretely spared. Finally, a thorough neuropsychological assessment may be useful to program possible rehabilitation treatments in order to optimize the rehabilitation of visual-spatial skills.

## References

Balint, R. (1909). Seelenlahmung des "Schauens", optische Ataxie, raumliche Storung der AufmerKsamkeit. *Monatschrift fur Psychiatrie und Neurologie*, 25, 51-181.

Battaglia-Mayer, A., & Caminiti, R. (2002). Optic ataxia as a result of the breakdown of the global tuning fields of parietal neurones. *Brain*, 125 (Pt 2), 225-237.

Catani, M., Dell'Acqua, F., Vergani, F., Malik, F., Hodge, H., Roy, P., Thiebaut de Schotten, M. (2012). Short frontal lobe connections of the human brain. *Cortex*, 48 (2), 273-291.

Classen, J., Kunesch, E., Binkofski, F., Hilperath, F., Schlaug, G., Seitz, R.J., Freund, H.J. (1995). Subcortical origin of visuomotor apraxia. *Brain*, 118 (Pt 6), 1365-1374.

Damasio, A.R., & Benton, A.L. (1979). Impairment of hand movements under visual guidance. *Neurology*, 29 (2), 170-174.

Damasio, A.R., Tranel, D., & Rizzo, M. (2000). Disorders of complex visual processing. In: Mesulam, M.M. (ed.), *Principles of behavioral and cognitive neurology*, 2nd ed. New York: Oxford University Press, pp. 332-372.

Doricchi, F., Thiebaut de Schotten, M., Tomaiuolo, F., & Bartolomeo, P. (2008). White matter (dis)connections and gray matter (dys)functions in visual neglect: gaining insights into the brain networks of spatial awareness. *Cortex*, 44 (8), 983-995.

Farah, M.J., Brunn, J.L., Wong, A.B., Wallace, M.A., & Carpenter, P.A. (1990). Frames of reference for allocating attention to space: evidence from the neglect syndrome. *Neuropsychologia*, 28 (4), 355-447.

Ferro, J.M. (2001). Hyperacute cognitive stroke syndromes. *Journal of Neurology*, 248 (10), 841-849.

Ffytche, D.H., Blom, J.D., & Catani, M. (2010). Disorders of visual perception. *Journal of Neurology, Neurosurgery and Psychiatry*, 81 (11), 1280-1287.

Glickstein, M., & Berlucchi, G. (2008). Classical disconnection studies of the corpus callosum. *Cortex*, 44 (8), 914-927.

Heilman, K.M., & Watson, R.T. (2008). The disconnection apraxias. *Cortex*, 44 (8), 975-982.

Hof, P.R., Vogt, B.A., Bouras, C., & Morrison, J.H. (1997). Atypical form of Alzheimer's disease with prominent posterior cortical atrophy: a review of lesion distribution and circuit disconnection in cortical visual pathways. *Vision Research*, 37 (24), 3609-3625.

Husain, M., Mattingley, J.B., Rorden, C., Kennard, C., & Driver, J. (2000). Distinguishing sensory and motor biases in parietal and frontal neglect. *Brain*, 123 (Pt 8), 1643-1659.

Karnath, H.O., & Perenin, M.T. (2005). Cortical control of visually guided reaching: evidence from patients with optic ataxia. *Cerebral Cortex*, 15 (10), 1561-1569.

Leibovitch, F.S., Black, S.E., Caldwell, C.B., Erbert, P.L., Ehrlich, L.E., & Szalai, J.P. (1998). Behavior correlations in hemispatial neglect using CT and Spect: the Sunnybrook Stroke Study. *Neurology*, 50 (4), 901-908.

Olk, B., Hildebrandt, H., & Kingstone, A. (2010). Involuntary but not voluntary orienting contributes to a disengage deficit in visual neglect. *Cortex*, 46 (9), 1149-1164.

Pierrot-Deseilligny, C. (1994). Saccade and smooth pursuit impairment after cerebral hemispheric lesions. *European Neurology*, 34 (3), 121-134.

Pierrot-Deseilligny, C., Gautier, J.C., & Loron, P. (1988). Acquired ocular motor apraxia due to bilateral fronto-parietal infarcts. *Annals of Neurology*, 23 (2), 99-202.

Pierrot-Deseilligny, C., Gaymard, B., Müri, R., & Rivaud, S. (1997). Cerebral ocular motor signs. *Journal of Neurology*, 244 (2), 65-70.

Pierrot-Deseilligny, C., Gray, F., & Brunet, P. (1986). Infarcts of both inferior parietal lobules with impairment of visually-guided eye movements, peripheral visual inattention and optic ataxia. *Brain*, 109 (Pt 1), 81-97.

Shallice, T., Mussoni, A., D'Agostino, S., & Skrap, M. (2010). Right posterior cortical functions in a tumour patient series. *Cortex*, 46 (9), 1178-1188.

Thiebaut de Schotten, M., Dell'Acqua, F., Valabregue, R., & Catani, M. (2012). Monkey to human comparative anatomy of the frontal lobe association tracts. *Cortex*, 48 (1), 82-96.

Vallar, G., & Perani, D. (1986). The anatomy of unilateral neglect after right-hemisphere stroke lesions: a clinical/CT-scan correlation study in man. *Neuropsychologia*, 24 (5), 609-622.

van Kessel, M.E., van Nes, I.J., Brouwer, W.H., Geurts, A.C., & Fasotti, L. (2010). Visuospatial asymmetry and non-spatial attention in subacute stroke patients with and without neglect. *Cortex*, 46 (5), 602-612.

Yeterian, E.H., Pandya, D.N., Tomaiuolo, F., & Petrides, M. (2012). The cortical connectivity of the prefrontal cortex in the monkey brain. *Cortex*, 48 (1), 58-81.