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CASE REPORT

Anton's Syndrome Due to Bilateral Occipito-Parietal Haemorrhage: A Case Report

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Abstract

Background: Anton's syndrome is a rare syndrome characterised by denial of blindness by a patient who obviously cannot see. Visual anosognosia and usually caused by bilateral occipital infarct. Rarely caused by demyelination or haemorrhage.

Objective: The aim was to report a case of Anton's syndrome due to bilateral occipito-parietal lobar haemorrhage following percutaneous coronary intervention due to myocardial infarction.

Methods: The case was thoroughly evaluated clinically then diagnosis was confirmed by CT scan of head showing bilateral occipito-parietal haemorrhage.

Result: The possible cause of bilateral lobar haemorrhage was due to use of Heparin during procedure and duel antiplatelet after percutaneous coronary intervention.

Conclusion: A suspicion of cortical blindness and Anton's syndrome should be raised in patients with atypical visual loss and evidence of bilateral occipital lobe injury. Though infarction is the common cause but any other cause that leads to bilateral occipital damage like haemorrhage in this patient may cause this syndrome. Drug induced extensive intracerabral haemorrhage is difficult to manage in the setting of myocardial infarction.

Keywords: Anton's syndrome, Occipito-parietal haemorrhage, Duel antiplatelet drugs, Visual anosognosia

Introduction

Anton's syndrome is bilateral cortical blindness with visual anosognosia and visual confabulation.[1] This syndrome describes the condition in which patients deny their blindness despite objective evidence of visual loss, and moreover confabulate to support their stance. This is a rare extension of cortical blindness in which, in addition to the injury to the occipital cortex visual association areas also involved.We describe a case of a patient with Anton's syndrome and its associated features due to bilateral occipito-parietal lobar haemorrhage following percutaneous coronary intervention (PCI) for NSTEMI. PCI is recommended in patients with both ST elevated & non ST elevated myocardial infarction.[2] PCI with stenting required duel antiplatelet therapy to prevent

thrombosis one of the devastating complications after PCI. Use of duel antiplatelet therapy may cause major haemorrhage like intracranial haemorrhage (ICH). The risk of ICH associated with duel antiplatelet drug is related to the individual and summative potency of the agents. In the Stent Anticoagulation Restenosis Study, the risk of majorhemorrhagic complications was 1.8%, 5.5%, and 6.2%in patients on aspirin, aspirin–ticlopidine, and aspirin–warfarin, respectively.[3]Here we described a case of Anton's Syndrome due to bilateral lobar haemorrhage after PCI with duel antiplatelet therapy for NSEMI.

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The case

A 55 year old known ischaemic heart disease patient presented with severe headache with altered level of consciousness following percutaneous coronary intervention. He was initially diagnosed as non ST elevated myocardial infarction in a private hospital. Then percutaneous coronary intervention was done with combination of antiplatelet drugs. He developed sudden severe headache with altered level of consciousness following the day of PCI. Then he was shifted to intensive care unit. Initial CT scan of head (figure 1) showed bilateral occipito-parietal lobar haemorrhage with subarachnoid extension. Then patient was managed with conservative approach with fresh frozen plasma, dexamethasone and mannitol.

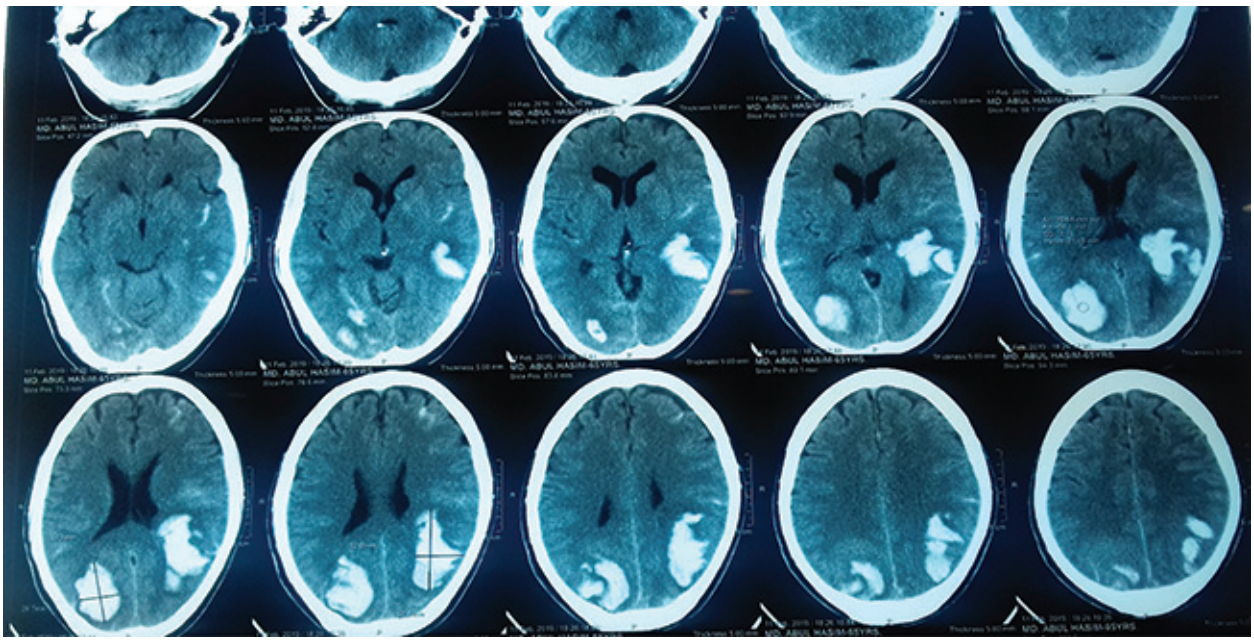

Figure 1: showing multi lober intracerebral haemorrhage with subarachnoid extension (both parieto occipital lober haemorrhage predominantly on the left side

All antiplatelet drugs were stopped after consultation with cardiologist. After a week patient's general condition was improved, he regained consciousness and was shifted to stroke center. On arrival to the stroke center his GCS was 15, he has no objective motor and sensory loss. He developed severe visual loss despite of normal pupillary reflex and normal fundoscopy. But after regaining consciousness he became restless, agitated and developed visual anosognosia. Despite of blindness he denied of it. Patient was unaware of visual loss and maintained he was able to see things around him. This syndrome is called Anton's syndrome. Repeat CT after a week showed slight resolution of haemorrhage. Subsequently patient was died due to recurrent myocardial infarction with cardiac arrest.

Discussion

Anton's syndrome is the denial of loss of vision (visual anosognosia) associated with confabulation in the setting of obvious visual loss and cortical blindness. Frequently, patients with bilateral damage to the occipital lobes also have damage to their visual association cortex which may account for their lack of awareness.[4]

Anton's syndrome should be considered in all cases of visual impairment, with bilateral occipital lobe lesions. Underlying pathology is injury to the visual association cortex in the occipital lobes. The anterior visual pathways remain intact. The most common cause of cortical blindness is PCA occlusion.[5] When the terminal bifurcation of basilar artery is involved in a thrombo-embolic event, resulting stroke can be bilateral.[6]Although any cause of cortical blindness may potentially lead to Anton's syndrome, cerebrovascular disease is themost common.[8] In addition to the more commoncauses of Anton's syndrome, it has also been reported in hypertensive encephalopathy with pre-eclampsia, obstetric haemorrhage with hypoperfusion, andtrauma, amongst others.[8-10] Our patient developed this rare syndrome due to bilateral lober haemorrhage (right occipital & left parieto-occipital lober haemorrhage) following percutaneous coronary intervention due to non ST elevated myocardial infarction (NSTEMI). Possible mechanism may be due to use of heparin during the procedure associated with the use of multiple antiplatelet drugs after the procedure. Though the patient initially presented with headache and altered level of consciousness but after improvement of consciousness symptoms of Anton's syndrome were apparent. After initial improvement the patient subsequently died of recurrent Myocardial Infarction. Drug induced intracerebral haemorrhage in a patient of myocardial infarction is difficult to manage. Antiplatelet drugs should be cautiously used. We stopped all the antiplatelet drugs after consulting with

the cardiologist because our patient had not only intracerebral haemorrhage but also subarachnoid haemorrhage.

Conclusion

A suspicion of Anton's syndrome should be raised in patients with atypical visual loss and evidence of bilateral occipital lobe injury. Though infarction is the common cause but any other cause that leads to bilateral occipital damage like haemorrhage in our patient may cause this syndrome. Drug induced extensive intracerabral haemorrhage is difficult to manage in the setting of myocardial infarction. Though the initial improvement our patient was finally died due to subsequent massive myocardial Infarction.

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References

1. Chen JJ, Chang HF, HsuYC, Chen DL. Anton Babinski syndrome in an old patient: A case report and literature review. Psychogeriatrics. 2015; 15:58-61.DOI: 10.1111/psyg.12064
2. Amsterdam EA, Wenger NK, Brindis RG, et al. 2014 AHA/ACC guideline for the management of patients with non-ST-elevation acute coronary syndromes: executive summary: a report of the ACC/AHA Task Force on Practice Guidelines [published correction appears in Circulation. 2014;130(25):e431–e432].Circulation.2014;130(25):2354–2394. DOI: 10.1161/CIR.0000000000000133
3. Leon MB, Baim DS, Popma JJ, Gordon PC, Cutlip DE, Ho KK, Giambartolomei A, Diver DJ, Lasorda DM, Williams DO, Pocock SJ, Kuntz RE: A clinical trial comparing three antithrombotic-drug regimens after coronary-artery stenting. Stent Anticoagulation Restenosis Study Investigators. N Engl J Med. 1998; 339:1665–71. DOI: 10.1056/NEJM199812033392303
4. Heilman KM, Anosognosia: Possible neuropsychological mechanisms. In awareness of deficit after Brain injuri: clinical and theoretical issues. Edited by Prigatano FP, Schacter DL. New York: Oxford University Press;1991: 53-62.
5. Symonds C, Mackenzie IAN. Bilateral loss of vision from cerebral infarction. Brain. 1957; 80:415–55. DOI: 10.1093/brain/80.4.415
6. Satija L, Singh AP, Khanna SK, et al. Rare cause of complete blindness : Bilateraloccipital cortical infarction: A Report on Three Cases. Med J Armed Forces India.1999;55:149–50. DOI: 10.1016/S0377-1237(17)30273-3
7. Aldrich MS, Alessi AG, Beck RW, Gilman S: Cortical blindness:etiology, diagnosis, and prognosis. Ann Neurol. 1987, 21:149-158. DOI: 10.1002/ana.410210207.
8. Misra M, Rath S, Mohanty AB: Anton syndrome and corticalblindness due to bilateral occipital infarction. Indian J Ophthalmol. 1989, 37:196.
9. Argenta PA, Morgan MA: Cortical blindness and Antonsyndrome in a patient with obstetric haemorrhage. Obstet Gynecol. 1998, 91:810-812. DOI: 10.1016/S0029-7844(97)00718-7
10. McDaniel KD, McDaniel LD: Anton's syndrome in a patient withposttraumatic optic neuropathy and bifrontal contusions. Arch Neurol. 1991, 48:101-105. DOI: 10.1001/archneur.1991.00530130113028