Case Report

DOI: 10.7860/JCDR/2021/50886.15596

Diplopia following Percutaneous Coronary Intervention- A Rare Case

Others Section

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ABSTRACT

Acute neurologic complications are rare following cardiac catheterisation and lead to substantial morbidity and mortality. The most common abnormalities reported are seizures, visual disturbances, facial palsy, and hemiparesis, with a preponderance of anterior circulatory events among the localising deficits and intracranial bleeding. A 51-year-old male patient presented with symptom of precordial chest heaviness radiating to left upper arm associated with sweating since >24 hours. Electrocardiogram (ECG) showed QS complexes with ST-segment elevation for which the patient underwent coronary angioplasty. Later, the patient develops diplopia following successful coronary angioplasty and examination revealed isolated left partial oculomotor nerve palsy. Magnetic resonance imaging of brain showed acute infarct in the left superior pons in paramedian location and other vascular territories. The patient was managed conservatively, and symptoms improved at the time of discharge. On review of literature, there are very few case reports on the involvement of oculomotor nerve post cardiac catheterisation. Early diagnosis and management are vital for recovery.

Keywords: Coronary angioplasty, Neurological complications, Oculomotor nerve palsy

CASE REPORT

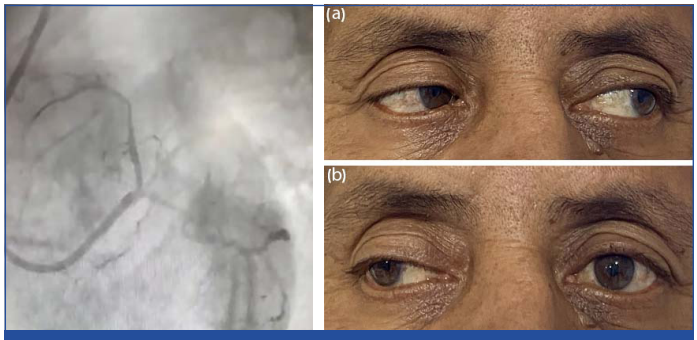
A 51-year-old male patient presented to the Emergency Department of a hospital with symptom of precordial chest heaviness radiating to left upper arm associated with sweating since >24 hours. The patient was hypertensive and had type-II diabetes mellitus for last five years. There was no other significant past medical or surgical history.

On physical examination, he was afebrile with pulse rate of 107 beats/minute, respiratory rate of 14 breaths/minute, blood pressure of systolic 100 mmHg and diastolic 70 mmHg. Systemic examination was unremarkable. Electrocardiography (ECG) showed QS complexes with ST-segment elevation from leads V1 to V4 and echocardiogram revealed regional wall motion abnormality in Left Anterior Descending artery (LAD) territory with moderate left ventricular systolic dysfunction. Haemogram and renal function parameters were within normal limits. Based on the clinical history, ECG and echocardiography findings diagnosis of acute anterior wall ST-segment elevation myocardial infarction (out of window period) was made.

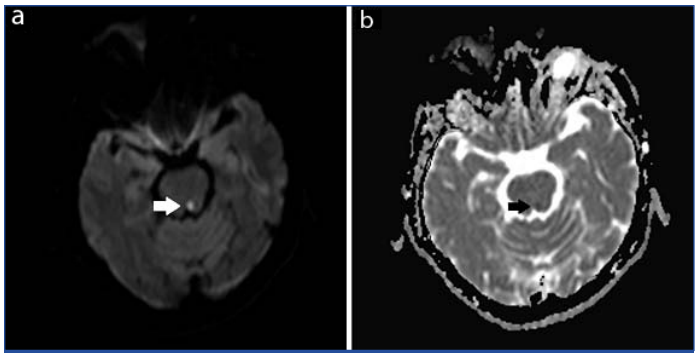
He was treated with aspirin 150 mg once daily, clopidogrel 75 mg once daily, atorvastatin 40 mg once daily and inj. enoxaparin 60 mg subcutaneous twice daily, inj. furosemide 40 mg intravenous twice daily. He underwent coronary angiography through the right radial approach which showed mid LAD subtotal occlusion with thrombus [Table/Fig-1], proximal and mid Right Coronary Artery (RCA) diffuse disease (small caliber vessel). Adhoc Percutaneous Transluminal Coronary Angioplasty (PTCA) was done to LAD. Peri-procedural and immediate postprocedure period was uneventful.

The next day (28 hours after procedure) he complained of double vision on right gaze. On clinical examination, there was absent abduction movement of left eye with preserved supraduction and infraduction movements and pupillary reflexes were preserved with no other abnormal neurological signs. Hence, a diagnosis of isolated partial left oculomotor nerve palsy with pupillary sparing was made [Table/Fig-2].

Magnetic Resonance Imaging (MRI) brain showed acute infarct in the left superior pons in paramedian location and acute infarcts in the cortex of right parafalcine frontal lobe, cortex of left parafalcine parietal lobe and cortex of right temporal lobe and cortex of bilateral



[Table/Fig-1]: Coronary angiogram showing mid LAD subtotal occlusion with thrombus. **[Table/Fig-2]:** Ocular movements: a) Preserved abduction of left eye indicating preserved lateral rectus muscle which is supplied by abduscens nerve; b) Absent adduction of left eye indicating medial rectus palsy which is supplied by oculomotor nerve. (Images from left to right)



[Table/Fig-3]: Magnetic Resonance Imaging of Brain: a) MRI brain axial diffusion weighted image showing restricted diffusion (high signal intensity) in the left side of superior pons in paramedian location (arrow) suggestive of acute infarct; b) MRI brain axial apparent diffusion coefficient map showing restricted diffusion (low signal intensity) in the left side of superior pons in paramedian location (arrow) suggestive of acute infarct.

cerebellar hemisphere [Table/Fig-3a,b]. Carotid artery Doppler showed non significant atherosclerotic disease. Due to evidence of infarct he was evaluated again with ECG and transthoracic echocardiography, which showed sinus rhythm and no evidence of thrombus, respectively.

The patient was treated with aspirin 150 mg once daily Postoperatively (PO), clopidogrel 75 mg once daily PO, atorvastatin 40 mg once daily PO and inj. enoxaparin 60 mg subcutaneous twice daily, inj.

furosemide 40 mg intravenous twice daily, nitroglycerin 2.5 mg orally twice daily PO, nicorandil 5 mg twice daily and was discharged after total hospital-stay of eight days. At follow-up, after two weeks, patient's symptoms resolved completely with no signs of diplopia. On ocular examination adduction, supra and infraduction in the left eye were present indicating recovery of muscle function.

DISCUSSION

The incidence of developing a neurologic event within 36 hours of cardiac catheterisation is 0.03-0.3 %. Approximately, half of these resolved by 24 hours, the incidence of neurologic deficits persisting >24 hours is only 0.2% [1,2]. The case fatality rate among middle-aged adults with out-of-hospital stroke is 10.3% at 30 days, nine and the one year mortality among one month stroke survivors is 9.8% [3]. The mechanism of acute neurologic complications after catheterisation include embolisation of atheromatous material originating in the ascending aorta, thrombus formation on catheters and guidewires, idiosyncratic reactions from contrast agents and spasm of cerebral arteries. Seizures are primarily due to a direct toxic effect of contrast to the central nervous system and is both dose-related and more common in patients with underlying central nervous system pathology [1].

Neuroimaging should be mandatory, if patients presents with neurological deficits after cardiac catheterisation, such as diffusion-weighted MRI or Computed Tomography (CT) perfusion study [4,5]. Haemorrhagic stroke should be ruled out before initiating any therapy, especially any aggressive antithrombotic therapy [4].

On review of literature, there were few case reports on the involvement of oculomotor nerve post cardiac catheterisation [6-9]. In a case report by Jilani MH et al., a 72-year-old diabetic male, who underwent elective cardiac catheterisation for stable angina which revealed multivessel CAD and no intervention was done. Two hours after procedure, patient developed left third nerve palsy. Rest of the neurological examination and CT angiogram of head and MRI brain were normal. Symptoms resolved eight weeks after discharge. They described embolic phenomenon as possible mechanism [6].

In the case report by Vasavada A et al., there was acute onset transient partial third cranial nerve palsy presenting as diplopia and ptosis after 24 hours of double vessel angioplasty which spontaneously improved after a day of its occurrence [8]. Their patient's neuroimaging with MRI of brain was normal and the explanation discussed was that the lesion could be lacunar infarct which was too small to be picked up on MRI involving the third nerve nuclei or an oculomotor involvement as seen in patients with diabetes or a transient inflammatory or immune response to contrast.

In another case report by Neupane S et al., there was transient third nerve palsy following diagnostic cardiac catheterisation [9]. MRI of the brain showed few scattered non specific transverse relaxation time (T2) and Fluid Attenuated Inversion Recovery (FLAIR) changes in the brain without evidence of acute ischaemia with unremarkable MR angiography. Carotid duplex demonstrated bilateral minimal atherosclerotic disease. Transient vasospasm or microinfarction was proposed to be the underlying mechanisms due to the transient symptoms and normal neuroimaging.

In present case, there was isolated unilateral oculomotor nerve palsy. MRI brain showed acute infarct in the left superior pons in the paramedian location and acute infarcts in the cortex of right parafalcine frontal lobe, cortex of left parafalcine parietal lobe, cortex of right temporal lobe and cortex of bilateral cerebellar hemisphere. The most likely mechanism could be the embolisation of atheromatous material from ascending aorta.

CONCLUSION(S)

Prompt diagnosis and timely management of acute neurological complications after cardiac catheterisation are keys for recovery and preservation of function. Multidisciplinary approach with cardiologist and neurologist should be implemented for patients' overall well-being. All interventionalist should be aware of this rare complication after successful PCI.

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PLAGIARISM CHECKING METHODS: [Jain H et al.]

- Plagiarism X-checker: Jun 16, 2021
- Manual Googling: Jun 23, 2021
- iThenticate Software: Oct 05, 2021 (20%)

ETYMOLOGY: Author Origin

AUTHOR DECLARATION:

- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Jun 15, 2021**
Date of Peer Review: **Aug 06, 2021**
Date of Acceptance: **Sep 26, 2021**
Date of Publishing: **Nov 01, 2021**