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Kasuistika | Case report

Seeing double: Transient third nerve palsy after cardiac catheterization

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SOUHRN

Bylo popsáno široké spektrum neurologických komplikací vzniklých po srdeční katetrizaci, přičemž nejčastějšími z nich jsou cévní mozkové příhody a neurooftalmologické projevy. V této kasuistice popisujeme přechodnou parézu třetího mozkového nervu (nervus oculomotorius) v návaznosti na diagnostickou srdeční katetrizaci. Muž ve věku 59 let vyhledal lékařské ošetření v nemocnici pro dvojité vidění při pohledech všemi směry, které se vyvinulo po diagnostické levostranné srdeční katetrizaci. Fyzikálním vyšetřením u něj bylo zjištěno omezení addukce, pohledu vzhůru a pohledu dolů na pravém oku, jakož i pravostranná hypermetropie suspektní z parézy n. oculomotorius. Pacient při korekci vykazoval normální zrakovou ostrost, měl normální nález na očním pozadí a ani v dalším neurologickém nálezu nebyly zjištěny žádné abnormality.
Výsledky zobrazení mozku byly negativní. Po částečném ústupu příznaků byl nemocný propuštěn do domácího ošetření s doporučením strategie modifikující jeho rizikové faktory, zahrnující užívání antiagregačního léčiva, statinu a beta-blokátoru.

Keywords:
Cardiac catheterization
Neurologic complications
Third nerve palsy

ABSTRACT

A wide variety of neurologic complications have been described after cardiac catheterization, with cerebrovascular accidents and neuro-ophthalmologic manifestations being the most common. Here we report a case of transient third nerve palsy following diagnostic cardiac catheterization. A 59-year-old male presented to the hospital with complaints of diplopia at all gazes after a diagnostic left heart catheterization. Physical examination revealed limitation on adduction, supra- and infraduction in the right eye, and right hypertropia on all gazes suggestive of third nerve palsy. He had normal visual acuity with correction, a normal retinal exam, and an otherwise benign neurologic exam.
Neuroimaging was negative. After the patient's symptoms improved, he was discharged home with risk factor modification strategy including anti-platelet therapy, statin and beta-blocker.

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Introduction

Neurologic complications of cardiac catheterization and coronary angioplasty are uncommon, but potentially devastating. A wide variety of manifestations have been described, with cerebrovascular accidents and neuro-ophthalmologic manifestations being the most common. Though embolism was believed to be the mechanism for development of neurological events, the exact pathophysiology still remains unclear. Previous literature demonstrated that old age, female sex, classic vascular risk factors increases the likelihood of complications [1]. Furthermore, coronary angioplasty appears to have more risk for adverse effects than diagnostic catheterization [1]. We describe a case of transient third nerve palsy following diagnostic cardiac catheterization.

Case review

A 59-year-old male presented to the hospital with complaints of diplopia at all gazes after a diagnostic cardiac catheterization. He underwent coronary angiography via femoral approach sixteen hours prior to presentation, which showed non-obstructive coronary artery disease that did not require any intervention. Immediately following the procedure, the patient noticed some visual changes that transiently improved. Subsequently, he was discharged home. However, he awoke in the middle of the night with significant double vision, which prompted him to immediately visit the emergency department. The patient denied any dizziness, dysphagia, or difficulties with speech. He also denied any motor or sensory symptoms in all four of his extremities. By way of brief background, he is known to have an abdominal aortic aneurysm, hyperlipidemia, and is an active smoker but not a diabetic. He did not have history of head trauma. Physical examination revealed limitation on adduction, supra- and infra-duction in the right eye, and right hypertropia on all gazes suggestive of third nerve palsy. Pupillary reflexes were intact. He had normal visual acuity with correction, and retinal exam. There were no other focal motor or sensory deficits on exam, and the rest of the cranial nerve exam was unremarkable. He was in sinus rhythm, and the access site was intact without any evidence of bleeding or hematoma. Magnetic Resonance Imaging (MRI) of the brain showed few scattered nonspecific T2/FLAIR changes in the brain without evidence of acute ischemia with unremarkable MR Angiography. Carotid duplex demonstrated bilateral minimal atherosclerotic disease. Transthoracic echocardiography with a bubble study, that was performed to evaluate for a cardiac source of embolism, did not demonstrate any evidence of mural thrombus or patent foramen ovale. There was gradual improvement in his symptoms without requiring any intervention. The patient was discharged home with risk factor modification strategy, including anti-platelet therapy, statin and beta-blocker.

Discussion

Neurological complications after cardiac catheterization or percutaneous coronary intervention (PCI) is rare, ranging from 0.2% to 0.4% [1,2]. However, the complications can be highly debilitating, leading to a high rate of morbidity and mortality [1,2]. Several neurological manifestations have been described, including cerebral vascular accidents (CVA), transient ischemic attack (TIA), confusion/stupor, seizures, and migraines [2]. Cardiac catheterization-associated neurologic events involve the vertebrobasilar circulation in more than 50% of cases, and the preponderance of posterior circulatory events is independent of the route of cardiac catheterization [2–4]. This leads to a wide range of visual symptoms. Patients may present with visual loss, diplopia, nystagmus, cortical blindness, and internuclear ophthalmoplegia. However, isolated third nerve palsy, as seen in our patient, has not been described previously. The fact that our patient had neurologic complication despite undergoing catheterization via femoral approach goes against the traditional belief that the antecubital approach is more commonly associated with these events [5].

Cerebral embolism originating from large atherosclerotic vessels has been described as the main pathophysiologic process. This finding is supported by transcranial Doppler studies performed during cardiac catheterization, which show the systematic occurrence of cerebral microemboli [1,6]. However, there are some suggestions that these events are not solely due to embolic phenomenon [3]. In one study by Lockwood et al., emboli could be documented in only one sixth of patients with neurological sequelae of cardiac catheterization [7]. Occurrence of these transient events, similar to our patient, can occur even in the absence of atherosclerotic disease, suggesting alternative mechanisms such as transient vasospasm [8]. Other complications, like cortical blindness, may be related to direct neurotoxic effect of the contrast agent on the blood-brain barrier in the occipital lobe [9]. Recent studies have shown a correlation between neurologic complications and a larger atherosclerotic burden, including advanced coronary artery disease, peripheral vascular disease, diabetes mellitus, and prior stroke [2]. Keeley et al. showed that scraping of aortic plaques happens in half of PCI cases and can occur more frequently with larger, rather than smaller, catheters [5,10]. Our patient appeared to have a large atherosclerotic burden, but no other risk factors, such as left ventricular hypertrophy and poor left ventricle function. Small vessel strokes accounted for almost half of the neurologic events in one study (48%), with a subset of these in the distribution of small penetrator vessels (23%) [3]. This is highly unusual for embolic mechanism because such strokes are more commonly associated with a lacunar type mechanism, rather than embolism [3]. Negative neuroimaging in our case effectively ruled out intracranial processes, which can have similar presentation such as demyelinating process and space occupying lesion. Likely pathophysiologic process in our case appeared to be either transient vasospasm or cerebral microinfarction considering transient nature of symptoms with negative neuroimaging.

When patients present with new neurological deficits after cardiac catheterization, it is reasonable to evaluate them with neuroimaging, such as diffusion weighted MRI or CT perfusion study [4,6]. Hemorrhagic stroke is very important to rule out before initiating any therapy, especially with the increasing use of aggressive antithrombotic therapy [4]. However, neuroimaging can be negative,

as in our case, especially when an alternative mechanism described above might be the culprit. Our patient did not undergo neurointervention because of delayed presentation, low NIH stroke scale, and absence of objective signs of ischemia on neuroimaging. Our patient had spontaneous gradual resolution of symptoms, as described in previous studies [1,4]. In the case of intra-procedural stroke, cerebral angiography and reperfusion by mechanical means or local thrombolysis is preferred, as it gives greater chance of recanalization over systemic thrombolysis [1,4]. Because the majority of these patients have recently received anti-platelet agents and full dose anti-coagulation, there is a low high of hemorrhagic complications [4].

In conclusion, neurological complications of cardiac catheterization are rare, but can be devastating. Cerebral microembolism is the main mechanism and preponderant involvement of posterior circulation has been described in several studies. Though spontaneous resolution of these symptoms has been reported, early identification and appropriate intervention can lead to better recovery.

Conflict of interest
No conflict of interest.

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Ethical statement
The case report was written according to ethical standards.

Informed consent
The patient provided the informed verbal consent to participate in the case report.

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