ACUTE ISCHEMIC STROKE AFTER PERCUTANEOUS CARDIAC INTERVENTION IN AN ELDERLY PATIENT

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SUMMARY

Because of tremendous progress in the field of interventional cardiology and maturation in operator techniques, cardiac catheterization and percutaneous cardiac intervention have steadily become safer with improved outcomes. Acute ischemic stroke during or after cardiac catheterization/percutaneous cardiac intervention is a rare but devastating complication. The low incidence of such events has precluded the emergence of a standard treatment. We present an elderly person who had suffered from acute ischemic stroke after percutaneous cardiac intervention in the catheterization laboratory. Relevant literature is also reviewed.


Key Words: cardiac catheterization, ischemic stroke, percutaneous cardiac intervention

Introduction

Ischemic stroke during or after cardiac catheterization/percutaneous cardiac intervention (PCI) is a rare but catastrophic complication. Risk factors include diabetes mellitus, hypertension, increased age, prior stroke, and renal insufficiency. Moreover, the technique of catheter manipulation, emergent cardiac procedures, interventions for vein grafts, and unplanned insertion of an intra-aortic balloon pump are predictors of this complication. Management of such events is challenging to all interventional cardiologists, because there is no standard treatment for this complication. Immediate cranial computed tomography or magnetic resonance image (MRI) with magnetic resonance angiography (MRA) is essential to evaluate new neurologic deficits noted after PCI. Prompt cerebral angiography can be considered in selective situations for diagnosis and treatment. We present an elderly person who had suffered from sudden neurologic deficits on-table after PCI in our catheterization laboratory. Ischemic stroke was proven by emergent cranial MRI with MRA. In our report, we discuss the characteristics of acute ischemic stroke after PCI and how to manage this serious complication in clinical practice.

Case Report

An 83-year-old man with a medical history of hypertension and renal insufficiency was admitted to our hospital because of angina pectoris. He could not accomplish the treadmill exercise test because of osteoarthritis of bilateral knee joints. Besides, he refused a myocardial perfusion image study. Echocardiogram revealed a significant hypokinesis of the inferior wall of the left ventricle. Cardiac catheterization was suggested by the interventional cardiologist and was performed the day after admission. Before the cardiac procedure went ahead, the patient’s chest X-ray revealed a tortuous aorta, and the electrocardiogram showed a regular sinus rhythm with ST depression in leads II, III and aVF. In the catheterization laboratory, coronary angiography...
showed significant stenosis (>80%) of the proximal to the middle segment of the right coronary artery. One bare-metal stent was deployed on the diseased segment of right coronary artery without hemodynamic instability or uncomfortable symptoms. Diagnostic and guiding catheters were advanced through the transradial sheath. In spite of widening and tortuosity of the aortic root, ostia of coronary arteries were engaged by catheters without much manipulation. All procedures were performed following standard practice with carefully checking back-bleeding whenever catheters were advanced. An air bubble was also expelled by meticulous flushing with heparinized saline. Unfractionated heparin was used at a total amount of 7,000 U. The fluoroscopic time during our procedure was within 30 minutes. The total amount of contrast used for diagnostic angiography and PCI was less than 100 mL. After PCI, we noticed that the patient presented with a lethargic appearance with sudden right limb weakness in our catheterization laboratory. Cranial MRI with MRA was performed emergently within 30 minutes. Cranial MRI showed a high signal intensity at the left upper temporal cortex and posterior frontal lobe in diffusion-weighted imaging (Figure 1). MRA revealed severe stenosis of the left vertebral artery and branches of left middle cerebral artery (Figure 2). All images were reviewed by an experienced radiologist and neurologist immediately, and the diagnosis of acute ischemic stroke was confirmed. We explained this situation to the family. They refused thrombolytic therapy and cerebral angiography. Adequate rehabilitation was planned and performed during hospitalization. This patient was discharged from our hospital after 14 days with partial recovery of motor and speech function.

Discussion

Although the overall rate of ischemic strokes during or after cardiac catheterization/PCI is low, ranging from 0.18% to 0.40%1–3, it is the most debilitating complication associated with a high rate of morbidity and mortality. However, low incidence and inexperience with this complication in a catheterization laboratory preclude operators from choosing an immediate and adequate management. The incidence of this complication is perhaps underestimated because of the increasingly high volume of cardiac procedures in the current era. The occurrence of mild transient ischemic attacks is another possible reason for the underestimation, because transient and mild symptoms or signs can be easily ignored by clinicians.

Ischemic strokes are usually embolic and could have many origins. For the purpose of left ventriculography, dislodged calcified particles from a stenotic aortic valve by retrograde catheterization would cause embolization to the brain4. Catheter tips traversing the aortic arch could dislodge atheromatous plaques, which embolize cerebral circulation. Keeley and Grines5 observed plaque dislodgment off the aortic arch with catheter advancement in more than 50% of 1,000 PCI procedures studied. Thus, inadequate and frequent catheter manipulation during cardiac procedures would increase risks of cerebral embolization. During urgent procedures, unplanned insertion of an intra-aortic balloon pump and less meticulous care in advancing the catheter through the aorta increase the chance of scraping aortic plaques with subsequent embolization.

Figure 1. Immediate cranial magnetic resonance imaging showing high signal intensity at the left upper temporal cortex and posterior frontal lobe in diffusion-weighted imaging.

Figure 2. Magnetic resonance angiography revealing severe stenosis of the left vertebral artery and branches of left middle cerebral artery.
Besides, hemodynamic instability in emergent situations and low cardiac output would further compromise cerebral perfusion and worsen ischemic events. Thrombus formation in situ on catheter tips is another possible source of embolism. Other embolic origins include air emboli and fragmented interventional devices.

In 20,679 consecutive patients who underwent PCI in a large-volume center reported by Dukkipati et al., cerebral infarction after angioplasty could be attributed to the following artery distributions: middle cerebral in 56%, anterior cerebral in 2%, posterior cerebral in 37%, superior cerebellar in 5%, posterior inferior cerebellar in 5%, and basilar in 7%. In the report by Dukkipati et al., infarcts to the anterior circulation represented 58%, and those to the posterior circulation represented 54%. This finding is of interest, because only about 20% of blood flow to the brain traverses the posterior circulation. One possible explanation is that the anatomy of the branches from the aortic arch probably favors small emboli entering the vertebral arteries.

Ischemic strokes after angioplasty mostly presented with motor and speech deficits. The interventional cardiologists should become aware of this point and observe their patients closely after procedures. Risk factors of periprocedural ischemic stroke include diabetes mellitus, hypertension, increased age, prior stroke, and renal insufficiency. Patients with chronic renal insufficiency often have concomitant cardiovascular and cerebrovascular risk factors, such as diabetes mellitus and hypertension. Diabetes and hypertension predispose to occlusive disease of intracranial and extracranial arteries. The preexisting neurovascular and systemic vascular compromise greatly reduces cerebrovascular reserve and hampers the ability of the brain to tolerate embolization. The immediate and longer-term consequences of this complication are devastating in terms of both morbidity and mortality, with a risk of in-hospital death of greater than 35% and a tenfold increase in 1-year mortality.

The first aim of those sudden neurologic deficits in the catheterization laboratory is to confirm that the acute event is due to a stroke or other diseases (e.g., seizure, migraine, encephalopathy, and hypoglycemia). Currently, no standard treatment for periprocedural ischemic stroke exists. Intravenous thrombolytic therapy with recombinant tissue plasminogen activator within 3 hours of ischemic stroke symptom onset has been shown to be efficacious in the general stroke population. However, the benefits and risks of this therapy for periprocedural ischemic strokes are unclear. Hemorrhagic risk is the most critical concern if there is concomitant administration of heparin or glycoprotein IIb/IIIa inhibitor, which are usually used in PCI. The use of intra-arterial thrombolysis in heparinized patients is not recommended, and the PROACT-I trial showed significantly higher rates of hemorrhagic transformation in intra-arterial thrombolytic-treated patients given high-dose heparin (approximately 10,000 U) compared with the lower doses (approximately 3,000 U). The risks of recombinant tissue plasminogen activator for stroke in the presence of glycoprotein IIb/IIIa inhibitors is not known, although preliminary data based on some small series of patients with stroke treated with the combination of these agents suggest potential safety. De Marco et al. reported six cases of periprocedural ischemic stroke complicating cardiac catheterizations in which immediate cerebral angiography was the key factor in their successful management. Cerebral angiography allows identification of the occluded vessels and offers reperfusion by mechanical means (e.g., thrombus aspiration, retrieval of a fractured device) or selective thrombolysis with a greater chance of recanalization than with intravenous thrombolysis. Although thrombolytic therapy is deemed to be safe and efficacious in some small studies, its utility remains to be determined for various origins of embolization. For example, if dislodged aortic arch plaque is the predominant origin, this calcified clot may not be amenable to lytic therapy. Conversely, a fresh thrombus on the tip of catheters or guidewire may be easier to destabilize.

In our report, an 83-year-old male suffered from acute ischemic stroke attributed to left middle cerebral artery territories after PCI. Although this elective procedure was accomplished following the standard process by an experienced operator and team, acute ischemic stroke was still the unfortunate occurrence. An embolic origin may be produced from multiple atheromatous plaques scraped by catheter tips or from tiny thrombus formation on catheters, which cannot be easily seen during procedures. A moderate amount of heparin used in PCI precluded the family’s and clinician’s decision for thrombolytic therapy. This unexpected event taught us that every procedure should be performed carefully.

In conclusion, acute ischemic stroke is one of the most catastrophic complications of PCI. Coronary interventional procedures must be performed with meticulous attention to technical detail. Immediate cranial
MRI with MRA or computed tomography is critical to assess any neurologic deficits of patients during or after procedures. Cerebral angiography can be considered for selective thrombolysis or endovascular interventions such as thrombus aspiration and retrieval of fractured devices. Whether thrombolysis is safe and efficacious remains to be determined, but the existing evidence from some small studies seems favorable. Thrombolytic therapy should be used under prompt evaluation for bleeding risks. Expeditious management including a multidisciplinary response team, including an interventional cardiologist, neurologist, radiologist and, potentially, a neurointerventionalist, is required to facilitate immediate imaging and treatment.

References