Central Nervous System Complications of Percutaneous Transluminal Coronary Angioplasty

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SUMMARY During 1968 consecutive percutaneous transluminal coronary angioplasty (PTCA) procedures, 4 patients (0.2%) suffered focal central nervous system complication. Two patients had a hemisphere infarct, one a brainstem infarct and one a hemisphere transient ischemic attack. Embolism was the likely mechanism in 3 cases; in 1 air was injected through the guiding catheter, and in 2 post-coronary bypass cases the ascending aorta was "scraped" with the guiding catheter while searching for a graft ostium. In 1 case the event occurred after a successful PTCA during a period of hypotension. Neurologic complications are rare during PTCA but will occasionally occur as the procedure is performed more frequently.

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PERCUTANEOUS TRANSLUMINAL ANGIOPLASTY was developed by Dotter and Judkins in 1964.1 Its purpose was to dilate atherosclerotic obstructions in peripheral arteries utilizing co-axial catheters of increasing diameter. Innovations in catheter design and modifications in technique led to the performance of angioplasty for proximal stenosis of coronary arteries.2 3

The number of percutaneous transluminal coronary angioplasties (PTCA) performed in the United States has increased markedly in the past five years. Rare complications will become more visible as the number of patients treated increases, yet analysis of neurologic events during PTCA has been virtually ignored.

In this report we discuss the frequency and types of central nervous system complications seen in a large consecutive series of patients undergoing PTCA at the Cleveland Clinic Foundation.

Methods

A. PTCA Registry

Data on all patients undergoing PTCA at the Cleveland Clinic is logged into a prospective registry. All patients undergo an initial history and physical exami-
nation under the supervision of the staff cardiologist who will perform the PTCA. Patients are then followed through the PTCA procedure by the attending cardiologist and registry data is recorded by a research fellow in cardiology. Complications, therefore, are initially observed by the cardiologist performing the procedure. About 75% of the patients in the registry were personally managed by one of the authors (JH.).

Between 1981 and April 1985, 1968 PTCA procedures were done in 1829 patients. Computer assistance was obtained to identify patients who had sustained neurologic complications during PTCA. Registry data was cross-checked by reviewing the neurology department consult records.

B. PTCA Technique

PTCA is performed in the cardiac catheterization laboratory using a Gruentzig-style double-lumen balloon catheter. PTCA catheters are 4.3 French, and are passed through 8 or 9 French guiding catheters. Pressure is delivered to the balloon by a hand-held syringe or various mechanical pumps. Pressure monitoring devices are attached to prevent over-inflation and subsequent balloon rupture. One lumen in the catheter is utilized for inflation of the balloon, while the other can be used for hemodynamic monitoring or contrast injection during the angioplasty procedure.

Under fluoroscopic guidance, a pre-shaped guiding catheter is introduced percutaneously at either a brachial or femoral site. Test contrast injections are made in the descending aorta although occasionally this is done in the ascending aorta. The desired coronary ostium is located, and the dilating catheter is directed to the coronary artery through the guiding catheter. Localization of the dilating catheter over the stenosed segment is achieved with small injections of contrast material through the dilating catheter. Hemodynamic monitoring through the dilating catheter records an intra-coronary pressure drop when crossing the stenotic segment. The balloon is inflated within the atherosclerotic stenosis by the injection of water-soluble contrast material. Following inflation for 3 to 10 seconds, the balloon is rapidly deflated. Success of the procedure is determined by a diminished intra-coronary pressure gradient and by angiographic evidence of a widened lumen. The dilation is usually repeated multiple times, interspersed with rest periods to allow distal flow. Electrocardiographic and hemodynamic monitoring are maintained throughout the procedure. Aspirin 5 gr p.o. tid and dipyridamole 75 mg p.o. tid are begun 24—36 hours prior to PTCA. Low molecular weight dextran may or may not be added for further antiplatelet action. Anticoagulation with heparin (10,000 U) is employed during the procedure and may be continued 24 hours post-angioplasty. Coronary artery vasospasm is treated prophylactically or as needed with nitroglycerin or calcium-entry blockers.

Report of Cases

Case 1

A 50-year-old white male with angina pectoris was admitted for PTCA of a 99% stenosis of the left anterior descending artery. There was no history of hypertension, diabetes mellitus, stroke, claudication or tobacco use. The pre-PTCA physical examination was normal. The patient was premedicated according to protocol. Following the introduction of the femoral guiding catheter and during the injection of contrast material into the ascending aorta, the patient experienced acute left-sided weakness and numbness. It was the operator's impression that air had been injected into the ascending aorta. Three control injections had been performed using the 9F guiding catheter.

The patient claimed that his left side did not belong to him. A right gaze preference was noted. The procedure was aborted and the patient was transferred to the cardiac intensive care unit. Within 30 minutes of the event, the patient was evaluated by the neurology service. By that time there had been considerable resolution of his neurologic deficits. There was only mild weakness of the distal left arm and a left Babinski response, which subsequently cleared. The following day PTCA was performed successfully. Carotid angiograms and CT of the head were normal. A follow up thallium stress test was negative at 6 months. The patient experienced no residual neurologic deficit and returned to his full time practice of law.

Case 2

A 37-year-old white male with severe angina was admitted to the Cleveland Clinic for PTCA of the proximal native left anterior descending artery, PTCA of the distal insertion site of a vein graft to the diagonal artery and PTCA of the distal insertion site of the LAD graft. The patient had undergone coronary bypass surgery two years previously for triple-vessel disease. After successful PTCA of the proximal LAD and during a search of the ascending aorta for a proximal vein graft anastomosis the patient complained of double vision with blurring. The procedure was completed with all three attempted sites dilated. The patient was returned to the cardiac floor and was evaluated by the neurology service within an hour of the onset of the episode. Examination revealed a left internuclear ophthalmoplegia, binocular rotary nystagmus, and a left ptosis. All other cranial nerves were intact, and there were no other neurological deficits. CT performed the following day revealed a poorly defined area of decreased attenuation coefficient at the junction of the midbrain and pons. Magnetic resonance imaging revealed several areas of increased signal intensity in the pons. Alternate patching of the eyes was recommended as well as 5 gr of aspirin daily. Seven days post-PTCA the patient's diplopia and adduction palsy had resolved but rotary nystagmus on horizontal gaze persisted. At 3 months post PTCA, the patient was free of angina and any neurologic disability.

Case 3

This 49-year-old, right-handed female was admitted for PTCA of a saphenous-vein graft to the right coronary artery. The patient had undergone bypass surgery 14 years earlier, with excellent results. The admission
neurologic examination was normal except for a right supraclavicular bruit. The patient was pre-medicated according to the standard protocol.

During the second injection of contrast to localize coronary vessels she became acutely aphasic. The cardiologists in attendance noted no weakness. The patient was evaluated by the neurology service within the hour, following discontinuation of the PTCA procedure. The aphasia was beginning to improve by that time, and the patient was able to answer questions and make simple statements. In addition to a non-fluent dysphasia, she had mild right facial weakness and deviation of the tongue to the right. The patient denied any previous history of stroke but reported a history of migraine headaches.

A CT of the head was normal. Over the following 48 hours the patient's speech difficulty worsened. Repeat CT remained normal and the patient was placed on aspirin and dipyridamole. Carotid IA-DSA was performed three days post PTCA and revealed a 40% stenosis of the left internal carotid artery. A speech pathology evaluation suggested a strong functional element to her dysfluent speech, but could not entirely rule out an organic component.

During the next week the patient's speech difficulty waxed and waned. The patient was discharged approximately 10 days post-PTCA. She ultimately underwent a successful left carotid endarterectomy.

Case 4

This 69-year-old, right-handed white female with triple vessel coronary disease and a one-month history of angina was admitted for PTCA of a proximal 90% stenosis of the left anterior descending artery. The patient's past medical history was significant for hypertension of approximately thirty years. The patient developed her neurologic deficit following PTCA and revealed a right hemisphere transient ischemic attack and an anoxic encephalopathy. Further details were not provided.

Among our series of 1892 patients, four (0.2%) suffered central nervous system complications. This rate compares favorably with the rate in the NHLBI study. Two patients suffered a left hemisphere infarct, one a right hemisphere transient ischemic attack and one a rostral brainstem infarct.

The events described in this paper are unusual complications of PTCA. Case 1 was likely due to injection of air through the guiding catheter. Fortunately the patient had no permanent neurologic deficit. Cases 2 and 3 were post-coronary bypass patients who experienced their events when searching the ascending aorta for aorto-saphenous vein graft anastomotic sites. During this phase of the procedure the ascending aorta is "scraped" with the relatively stiff guiding catheter. The potential for embolism of atherosclerotic material is obvious and should alert the operator to use extra care in these patients, particularly when manipulating the stiff guiding catheter on the ascending aorta. Case 4 developed her neurologic deficit following PTCA during a period of relative hypotension. Hypotension is relatively common after PTCA and may be due to the diuretic effect of the contrast, or vagal reaction to groin sheath manipulation or removal.

Coronary arteriography, which is the diagnostic forerunner of PTCA, has been performed since the 1960's, and analysis of its complications is extensive.7-15 The mechanisms of central nervous system complications, whatever they may be, must be similar between the two procedures. The rate of central nervous system damage during coronary arteriography is less than 1% in numerous studies.12,13 Systemic heparinization,10,17 aspirin pre-treatment,10 avoidance of entry into cerebral vessels,10,15 modifications in catheter design16 and original techniques,17 as well as acquisition of skill and experience17 all contributed to the fall in complications during catheterization. These modifications have been incorporated into PTCA and probably affect the neurologic complication rate.

References


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Calcific Cerebral Emboli and Aortic Stenosis: Detection of Computed Tomography

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SUMMARY Embolism with brain infarction rarely complicates calcific aortic stenosis (CAS). We report a case with severe CAS where the patient experienced multiple embolic strokes immediately following retrograde heart catheterization. Calcific emboli in the cerebral arteries were demonstrated by computed tomography (CT).

IN THE ABSENCE OF coexistent mitral stenosis or infective endocarditis, clinical brain embolism is uncommonly associated with CAS.1-3 In a single previous report, occlusion of a femoral artery by a large calcium embolus followed left heart catheterization in a patient with severe CAS.4 We report a patient who suffered several embolic strokes following retrograde heart catheterization for CAS, with intra-arterial calcific emboli and cerebral infarction demonstrated by CT.

Case Report
A 66 year-old black male underwent aortography and left heart catheterization for evaluation of aortic valvular stenosis and suspected coronary artery disease. Prior to angiography, M-mode echocardiography had revealed a heavily calcified, stenotic aortic valve (fig. 1). The mitral valve annulus was minimally calcified; there was no mitral stenosis. Retrograde heart catheterization was done via a transfemoral approach severely stenotic with a 0.6 cm² valve orifice.

We report a patient who suffered several embolic strokes following retrograde heart catheterization for CAS, with intra-arterial calcific emboli and cerebral infarction demonstrated by CT.

Two days following cardiac catheterization, the patient developed a left hemiparesis, ataxia and confusion. The following day, the weakness progressed to a left hemiplegia, and a left hemianopsia was detected. A CT scan of the head done without contrast enhancement showed a focal calcific density along the course of the right middle cerebral artery (MCA) just proximal to the usual location of the MCA trifurcation and another in the proximal perimesencephalic segment of the right posterior cerebral artery (fig. 2). A low attenuation area was seen in the right basal ganglionic area and the adjacent corona radiata, that evolved in density on subsequent scans and was consistent with an acute cerebral infarction (fig. 3). Careful fundoscopic examination failed to reveal calcific retinal emboli.

Heparin therapy was briefly instituted, but was discontinued when hemiplegia persisted for 48 hours. No neurologic worsening occurred after the fifth day postcardiac catheterization. A follow up CT scan eight months later showed no change in the position of the calcific emboli and no extension of the area of infarction.

Discussion
While the underlying pathology of CAS is usually a congenitally bicuspid aortic valve or an inflammatory fibrocalcific process (including rheumatic valvular disease), the continuing disease process is one of organizing microthrombi on disrupted valvular endothelium.

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