Large Coronary Thrombus As a Possible Cause of Periprocedural Transient Ischemic Attack During Percutaneous Coronary Intervention: Case Report

Perkütan Koroner Girişim Sırasında Periprosedürel Geçici İskemik Atağa Neden Olan Büyük Koroner Trombüs

ABSTRACT Cerebrovascular events during or after cardiac catheterization/percutaneous coronary intervention are infrequent but devastating complications. The low incidence of such events has precluded the emergence of a standard treatment. Emergent cardiac procedures, the techniques of catheter manipulation, interventions for vein grafts, and unplanned insertion of an intra-aortic balloon pump are predictors of these complications. Many of the risk factors associated with these events are not modifiable. We present an 56-year-old man who had suffered from transient ischemic attack during percutaneous coronary intervention in the catheterization laboratory due to large coronary thrombus. The possibility of combined cerebral embolism as a rare etiology should be kept in mind when a patient with acute coronary syndrome presents, especially when there is evidence of transient ischemic attack.

Key Words: Angioplasty, balloon, coronary; ischemic attack, transient; coronary thrombosis

ÖZET Serebrovasküler olaylar; kardiyak kateterizasyon/perkütan koroner girişim sırasında ya da sonrasında gelişen nadir ama yıkıcı komplikasyonlardır. Bu olayların insidansının düşük olması standart bir tedavi geliştirilmesini engellemiştir. Acil kardiyak girişimler, kateter manipülasyon teknikleri, ven greftine işlem yapmak ve plansız intraaortik balon pompası yerleştirilmesi bunların prediktörleridir. Bu komplikasyonla ilişkili olan risk faktörlerinin birçoğu modifiye edilemeyen risk faktörleridir. Biz bu olgu raporunda; perkütan koroner girişim sırasında, kateter laboratuarında, büyük ve hareketli koroner trombüsün neden olduğu geçici iskemik atak gelişen 56 yaşındaki bir erkek hastayı sunduk. Akut koroner sendrom hastalarında geçici iskemik atak geliştiği zaman, koroner trombüs embolizasyonuna bağlı nadir olarak görülen eş zamanlı serebral emboli olasılığı akılda tutulmalıdır.

Anahtar Kelimeler: Anjiyoplasti, balon, koroner; iskemik atak, geçici; koroner tromboz

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Stroke is an infrequent but potentially devastating complication of acute coronary syndromes (ACS) treated with percutaneous coronary intervention (PCI).¹ Patients who experience a stroke during or after diagnostic cardiac catheterization or PCI have an increased length of hospital stay by approximately four days, and experience moderate to severe disability post-discharge. The in-hospital mortality rate ranges from 25-44%.² Patients at higher periprocedural risk for stroke or transient ischemic attack are those with: advanced age, female gender, history of stroke, renal failure, diabetes mellitus, arterial hypertension, peripheral vascular disease,

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dyslipidemia, tobacco use, atrial fibrillation, previous myocardial infarction, PCI done under emergent conditions and the use of an intra-aortic balloon pump.^{3,4} Management of such events is challenging to all interventional cardiologists, because there is no standard treatment for this complication. We report a case of cerebral embolization that occurred during percutaneous coronary intervention because of dislodging of preexisting coronary thrombi, as confirmed by coronary angiography.

CASE REPORT

An 56-year-old man was referred to our hospital for the management of chest pain that had rapid onset and had persisted for several days. The pain was described as non-pleuritic, and not worse with palpation or movement. There was no associated shortness of breath or diaphoresis. He denied tobacco, alcohol, or drug use, and does not have a family history of cardiovascular disease. He has no known drug allergies. He reported no history of cardiovascular or cerebrovascular disease. On admission, the pulse oximetry saturation was 96%; pulse rate, 90 beat/min; and blood pressure, 120/70 mmHg. His heart and respiratory sounds were normal, and pretibial edema was not observed. Blood analysis showed a normal hemogram and increased levels of cardiac enzymes. A point-of-care troponin T test yielded a positive result. The electrocardiogram (ECG) presented diffuse ST-segment depression to D1 and aVL leads and from V4 to V6 precordial leads with supraventricular ectopic beats and QS wave in leads D2, D3 and aVF. A chest roentgenogram showed mild pulmonary congestion and a high cardiothoracic ratio (62%). An echocardiogram showed hypokinesis of the inferior and apical wall of the left ventricle. The left ventricular ejection fraction as assessed using the modified Simpson method was 40%. The septal and posterior wall thicknesses at the end-diastole were 14 mm each, and the left atrial dimension at endsystole was 40 mm. A color Doppler echocardiogram did not show significant valvular heart disease. He immediately underwent PCI after pretreatment with 300 mg of aspirin and 600 mg of clopidogrel and 8.000 IU of intravenous unfractionated heparin. Diagnostic coronary angiography showed the chronic totally occluded left anterior descending artery (LAD) and total occlusion of the proximal right coronary artery. PCI was performed using a 6Fr guiding catheter through the femoral approach. 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. The angiogram taken immediately after ballooning revealed that the vessel was occluded at a more proximal portion of RCA, most likely because of migration of the huge, fresh and floating thrombus (Figures 1-4). We first administered tirofiban via intracoronary route at a dose of 10 µg/kg followed by peripheral intravenous infusion at 0.15 µg/kg/min and then implanted a coronary 3.5 ×18 mm bare metal stent into RCA. After PCI, we noticed that the patient presented with a lethargic appearance with sudden right limb weakness in our catheterization laboratory. Directly after the coronary angiography the patient underwent neurological consultation. Cranial MRI with MRA was performed emergently within 30 minutes. Urgently performed MRI with MRA of the head did not reveal any pathology. On follow-up after several hours, his power of right upper and lower limb was normal. In neurologic examination as well as in the cerebral computed tomography scan no pathologies were found. The patient was discharged home on the third day after the coronary angiography in a good general condition and without any symptoms.

DISCUSSION

The overall rate of ischemic strokes during or after cardiac catheterization ranges from 0.18% to 0.40%, with higher morbidity and mortality rates.⁵ Not surprisingly, the rate was higher for those receiving PCI for ACS compared with those undergoing elective PCI (0.6% vs. 0.3%).⁶ Strokes among patients with acute coronary syndromes enrolled in the Organization to Assess Strategies for Ischemic Syndromes (OASIS) I and II studies



FIGURES 1-4: Migration of the huge and fresh thrombus.

were associated with six-month mortality rates of 27%.⁶

Infarcts during catheterization and intervention arise from various embolic sources. The composition of the emboli also varies, from air to soft clot to calcified atheroma, or multiple compositions such as atheroma with a fibrin clot around it. Thrombus formed within the catheter or catheter tip during the procedure can also become a source of emboli.^{7,8} 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. It is estimated that asymptomatic cerebral infarction after cardiac catheterization has an incidence of approximately 15%.9

While the risk factors for cardiovascular disease and cerebrovascular disease are similar, invasive cardiac procedures carry additional inherent risks, which means the catheter laboratory team team must be aware of hospital protocol should a PCI patient suffer a stroke during or after their procedure. Rapid recognition of a stroke and immediate intervention can significantly improve the long-term outcomes. Therefore, identifying patients at high risk, and understanding the symptoms and treatment possibilities for stroke is vital.

In the case presented, the patient suffered a transient ischemic attack during percutaneous coronary intervention for non-ST elevation myocardial infarction. Hoffman et al. found at univariate analysis that intracoronary thrombus was more frequent in the post PCI cerebrovascular events (CVEs), however this remained only as a trend (P=0.0734) when introduced into the multivariate model.¹⁰ In distinction to previously published analyses we thought that the presence of an intra-coronary thrombus is independently associated with a periprocedural CVE. Guptill et al. showed significantly higher rates (1.3%) of intervention-related CVEs in a primary PCI population that by definition must have an elevated thrombus burden supports our findings of the role of thrombus in intervention-related CVEs.¹¹ It would be extremely interesting to attempt to determine the mechanism of action of this association, particularly if the thrombus is a marker for a prothrom-

botic state or whether itself is the source of the subsequent embolus as our case.

In conclusion, acute ischemic stroke is one of the most catastrophic complications of PCI. Expeditious management including a multidisciplinary response team (cardiologist, neurologist, radiologist, and potentially a neurointerventionalist) is required to facilitate immediate imaging and institution of therapy akin to standard acute stroke management. Coronary interventional procedures must be performed with meticulous attention to technical detail. Careful procedural planning should thus be undertaken in an attempt to reduce the risk of PCIstroke, particularly in patients at increased risk.

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