

**#17 ACUTE ARTERIAL THROMBOSIS ASSOCIATED WITH COCAINE ABUSE**

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**Purpose:** Cocaine-induced arterial thrombosis is uncommon, and most of reported cases involved small-diameter vessels such as the cerebral and coronary arteries. This study was undertaken to review our experience of peripheral arterial thrombosis presumably caused by cocaine abuse.

**Methods:** Hospital records were reviewed for all patients who were admitted with acute arterial occlusion involving the abdominal or lower extremity arterial system over a ten-year period. Patients with confirmation of cocaine or its derivative crack usage within 24 hours of the hospital admission formed the basis of this study. Presenting symptoms, management, and outcome of these patients were reviewed.

**Results:** A total of 382 patients with acute peripheral arterial occlusion were identified during the study period. The presumptive diagnosis of cocaine-induced arterial occlusion was made in five patients, which included four men and one woman (mean age  $38 \pm 12$  years, range 27-53 years). Cocaine usage via intranasal inhalation occurred in two patients (40%) while the remaining three patients smoked crack cocaine (60%). All patients had history of polysubstance abuse, but without risk factors such as diabetes or hypertension. One patient (25%) was receiving antiviral therapy for acquired immunodeficiency syndrome (AIDS). The mean duration between cocaine usage and onset of arterial thrombosis was 10.4 hours (range 3–20 hours). Presenting symptoms included acute limb ischemia without pedal Doppler signals (n=3, 60%) and abdominal pain without femoral pulses (n=2, 40%). Arterial occlusion was confirmed by angiography in all patients, which revealed aortic thrombosis in one patient (20%), aortoiliac thrombosis in two patients (40%), and femoropopliteal artery thrombosis in two patients (40%). Surgical thromboembolectomy was successfully performed in four patients (80%) while one patient (20%) underwent successful thrombolytic therapy of a femoropopliteal artery occlusion. There was no perioperative mortality. All five patients who were discharged were available for follow-up (mean 36 months; range 6 to 75 months). There was one late death due to myocardial infarction. One patient developed recurrent lower extremity arterial thrombosis 28 months later which was successfully treated with thrombolytic therapy.

**Conclusions:** Our study underscores cocaine abuse as a potential etiology of acute arterial thrombosis. Cocaine-induced arterial thrombosis should be suspected in patients with recent history of cocaine or crack usage who present with acute limb ischemia without overt cardiovascular risk factors. Prompt angiography with operative or endovascular intervention is safe and effective in achieving durable outcome.