Transient ST elevation due to coronary spasm in a young woman

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A 43-year-old woman was admitted to the coronary care unit at Nordland Hospital (Bodø, Norway) because of a two-week history of transient chest pain. Her total cholesterol level was 8.0 mmol/L and she was a heavy cigarette smoker. On arrival, her vital signs were stable and she was pain-free. An electrocardiogram (ECG) showed inverted and biphasic T waves in the precordial leads (Figure 1A), but after 30 min, she developed intense pressing chest

pain and an ECG indicated severe anterior transmural ischemia (Figure 1B). She was immediately transferred to the coronary care unit where the pain subsided in 5 min to 10 min. Echocardiography showed normal contractility in all coronary territories. A control ECG showed regression of the ST elevation (Figure 1C). She was transferred to the University Hospital of North Norway (Tromsø, Norway) for coronary angiography. Troponin T values were all below

Figure 1) A Electrocardiogram (ECG) on arrival (precordial leads V1 to V6 only). There are widespread T inversions. **B** ECG demonstrating ST elevations in the anterior leads during chest pain. **C** ECG showing regression of ST elevation after the chest pain resolved

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Figure 2) Angiogram of the left coronary artery. The arrow depicts a plaque in the proximal left anterior descending artery, just before the diagonal branch

0.01 µg/L. Coronary angiography showed a small plaque in the proximal left anterior descending artery (Figure 2) but the flow was unobstructed Thrombolysis In Myocardial Infarction (TIMI) grade 3. Coronary spasm was suspected and she was started on a calcium blocker. She remained symptom-free after six months.

Coronary spasm (variant angina or Prinzmetal's angina) is believed to be caused by a transient increase in the coronary artery vasomotor tone (1). Spasm may also occur in proximity to atherosclerotic lesions including small lesions (2). Patients are often heavy cigarette smokers (3). Treatment includes nitrates and calcium antagonists.

REFERENCES

1. Prinzmetal M, Kennamer R, Merliss R, Wada T, Bor N. Angina Pectoris. I. A variant form of angina pectoris, preliminary report. *Am J Med* 1959;27:375-88.
2. Yamagishi M, Miyake K, Tamai J, Nakatani S, Koyama J, Nissen SE. Intravascular ultrasound detection of atherosclerosis at the site of focal vasospasm in angiographically normal or minimally narrowed coronary segments. *J Am Coll Cardiol* 1994;23:352-7.
3. Kim HS, Lee MM, Oh BH, et al. Variant angina is not associated with angiotensin I converting enzyme gene polymorphism but rather with smoking. *Coron Artery Dis* 1999;10:227-33.