Acute myocardial infarction with normal coronary arteries — viral myopericarditis and possible coronary vasospasm

A case report

T. H. DIAMOND, P. LOTZOF, F. ZIADY

Summary

A young man with normal coronary arteries presented with a transmural apical myocardial infarction. This diagnosis was based on elevated serial cardiac enzyme values, ECG changes, exercise scarring with thallium-201, left ventricular angiography and selective coronary arteriography. Some of the pathophysiological mechanisms implicated in myocardial infarction in patients with normal coronary arteries are discussed and the probable diagnosis of a virus-induced myopericarditis together with coronary artery vasospasm is favoured.


Department of Internal Medicine, 1 Military Hospital, Voortrekkerhoogte

T. H. DIAMOND, M.B., B.CH., M.R.C.P.

P. LOTZOF, M.B. B.CH.

F. ZIADY, M.D., F.R.C.P., F.C.P. (SA.)
Discussion

Myocardial infarction in young people has received much attention over the past decade. Many authors have documented numerous factors implicated in the pathogenesis of this condition, some in patients with angiographically normal coronary arteries. The incidence of normal coronary arteries in all patients with acute myocardial infarction has been estimated to be approximately 2%. This figure is markedly elevated to 22% if only patients under the age of 35 years are considered.

Myocardial ischaemia and its extreme consequence, acute myocardial infarction, can result from a transient or permanent imbalance between myocardial oxygen demand and coronary artery blood flow. By far the most common and important pathological condition underlying myocardial infarction is coronary atherosclerosis. Cheitlin et al., however, have clearly indicated the numerous causes of myocardial infarction without coronary atherosclerosis. These include: non-atherogenic disease of the coronary artery wall (coronary arteritis, trauma, metabolic disorders, aortic dissection and spasm), intraluminal coronary artery occlusion (septic or bland emboli), congenital coronary artery anomalies (anomalous left coronary artery originating from the pulmonary artery), myocardial oxygen demand-supply disproportions and various other disorders.

Miklozek and Abelman described 9 patients who were shown to have viral myocarditis, transmural myocardial infarction and normal coronary arteries. The mechanisms responsible for coronary occlusion may have been coronary arteritis or vasospasm, but neither was clearly elucidated. Increasing attention has recently been focused on the syndrome of coronary artery vasospasm in normal or diseased coronary arteries; today it is considered to be one of the most frequent
mechanisms involved in the pathogenesis of myocardial infarction with normal coronary arteries. 1-3,7 The case described demonstrated the classic ECG manifestations of a transmural apical myocardial infarction in a young man with angiographically proven normal coronary arteries. The ECG at first showed features of an acute apical myocardial infarction, i.e. loss of ensuing R wave, evidence of Q waves and concave-upward elevation of the ST segments in standard leads III, AVF and frontal leads V2 - V5. Left anterior hemiblock and transient right bundle-branch block were also present. This transient bifascicular block, together with the pericardial friction rub, were thought to be complications of the acute myocardial infarction. The persistence of the elevated ST segments in the frontal leads 6 weeks after the initial injury suggested the presence of an apical left ventricular aneurysm. 8

Several systemic viral infections (Coxsackie virus infection, mumps and influenza) may cause a myocarditis, which can occasionally be fatal. 4,9,10 ECG abnormalities in myocarditis include T-wave and ST-segment deviations, conduction and rhythm disturbances and rarely, signs of transmural myocardial infarction. 10 This case illustrates these features and the elevated influenza virus titre suggests an underlying viral myopericarditis.

Pathological changes in interstitial viral myocarditis include an inflammatory cellular infiltrate, cellular swelling and coagulative necrosis. 3,9 These changes may manifest clinically as cardiomegaly, congestive cardiac failure, or a severe cardiomyopathy with diffuse ECG changes and low-voltage T waves. Our patient had a normal cardiac radiographic silhouette and no clinical evidence of congestive cardiac failure. The ECG and 201Tl scan showed a focal apical infarction rather than a diffuse myopathic pattern. Various pathophysiological mechanisms have been described in patients with myocardial infarction and normal coronary arteries, 1-3,4,9,10 but have been difficult to demonstrate. We consider that in our patient coronary artery vasospasm occurred as a result of coronary arteritis or a viral myopericarditis, the triggering mechanism for the spasm being chemical or virus-related substances (e.g. thromboxane A2) released during the inflammation. Unfortunately, an ergometrine maleate provocation test was not performed at coronary angiography to verify this. Emboli formed in small coronary arteries adjacent to the inflammatory process cannot be totally excluded as another mechanism for transient coronary occlusion. This is nevertheless unlikely since on the coronary arteriogram 3 weeks after infarction no coronary occlusion or embolus was visible. Coronary arteritis and focal coronary artery vasospasm would involve a localized area or affect a single coronary artery and result in a focal area of infarction, as occurred in this patient.

Our patient presented with clinical, enzymatic and ECG changes of an acute apical myocardial infarction. In view of the absence of risk factors for atherogenesis and the circumstantial evidence of an existing viral infection, it was felt that he had viral myopericarditis and that this, together with coronary arteritis or vasospasm, caused the myocardial necrosis. In view of the numerous factors incriminated in the occurrence of myocardial infarction in young patients, coronary angiography was mandatory. The finding of a normal coronary angiogram, together with evidence of a possible viral infection, led to the diagnosis of viral myopericarditis. The mechanism of myocardial infarction is difficult to prove, but myocardial inflammation and coronary arteritis with focal coronary artery vasospasm resulting in an apical myocardial infarction remains a strong possibility.

REFERENCES

Nuus en Kommentaar/News and Comment

Diplomas vir mediese verteenwoordigers

Die meeste praktisyns vind besoke van goed ingeligte mediese verteenwoordigers van farmaseutiese firmas aanvaarbaar. Die Switsers wou verskeie dat hierdie verteenwoordigers inderdaad goed ingelig is deur die stigting van ’n vereniging van mediese verteenwoordigers in Switzerland, wat ook ’n eksamen van hierdie mense afneem. In 1984 het hierdie eksamen regeringsverteenwoordigers in Switserland, wat ook ’n eksamen van verteenwoordigers van farmaseutiese firmas aanvaarbaar. Die Switsers wou verseker dat hierdie verteenwoordigers inderdaad die meeste praktisyns vind besoeke van goed ingeligte mediese verteenwoordigers.