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Acute Myocardial Infarction in a Patient with Buerger's disease A Case Report and A Review of the Literature

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A twenty-nine year old male smoker with a three year history of Buerger's disease was admitted with excruciating precordial chest pain. The electrocardiogram indicated an anterior transmural infarction, and he also exhibited hypereosinophilia.

A coronary angiogram disclosed a partial segmental occlusion of the left anterior descending artery at the proximal portion. He was discharged without any complications after conservative managment.

Key Words: *Buerger's disease, Myocardial infarction*

Buerger's disease is an inflammatory and thrombo-occlusive vascular disease which usually involves the small and medium-sized arteries and veins of the proximal and distal extremities of young male smokers. Occasionally cerebral[1], mesenteric[2], coronary and other arteries[3] may be involved. In Korea, Buerger's disease is the most common occlusive disease of the peripheral vascular system, especially in the lower extremities of young men[4].

Although there have been a number of studies the etiology of Buerger's disease is unclear, but there is a correlation between it and smoking. When smoking is discontinued, progression of the disease is halted. Recently, some authors have suggested that cellular hypersensitivity to human collagen[3] and hypereosinophilia[6] may be a cause of Buerger's disease. Although coronary arterial involvement in Buerger's disease is very rare event, a few reports have alluded to it, but most of them were found at autopsy with no previous clinical indication of a myocardial infarction[7]. Recently, we experienced a patient with Buerger's disease and acute myocardial infarction with hypereosinophilia. Herein is a report of the case and a review of the literature.

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REPORT OF A CASE

A 29 year old male was admitted to Keimyung University Hospital in 1987 with a continous substernal chest pain of three days duration. He had been previously admitted in because of pain, chills, a tingling sensation and Raynaud's phenomenon of the left hand and both feet. At that time peripheral arterial mapping indicated that the left brachial and radial arterial pulsations were weaker than the right, and the popliteal and dorsalis pedis arterial pulsations were absent in both.

The laboratory findings included WBC, 12,800/cu mm with 25% eosinophils (3,000/cu mm), hematocrit, 45%; AST, 17 IU/1; ASO titer, 125 U; C-reactive protein, negative; cryoglobulin and cryofibrinogen, negative; serum IgE level, zero; HBS antigen, negative. The electrocardiogram taken on admission, revealed a normal sinus rhythm with no abnormal Q waves and ST segments. An arteriogram of the upper left and both lower extremities (Fig. 1) showed a segmental luminal narrowing of the profunda brachii and brachial arteries with thin, tree root shaped collaterals. The femoral angiogram revealed luminal narrowing and a corrugated appearance of the left popliteal

artery and prominent sural artery, which resembled a spider leg or vine tendrill. Buerger's disease was easily confirmed on the basis of the clinical features and angiographic findings. The patient was advised to stop smoking.

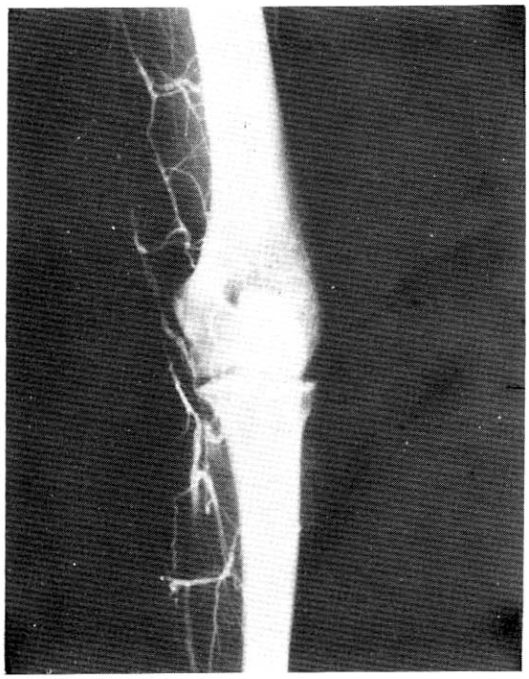

Fig. 1. *Arteriogram of the left upper extremity shows segmental occulsion of the left branchial artery at the left elbow joint, but it is recanalized through tourous collateral vessels from the superior ulnar collateral artery and radial collateral artery.*

Six months prior to readmission, he began smoking again, and three months later, anterior chest discomfort following heavy exercise or consumption of alcohol was noted. This symptom continued for five to ten minitues and subsided with rest.

After 3 days of episodes of severe substernal chest pain which continued for several hours and did not subside even with rest, he presented at Keimyung University Hospital to an evaluation of the chest pain. Upon admission the patient's vital sign were blood pressure: 130/80 mmHg, body temperature: 36.8C, and pulse rate 78/minitue. He was alert and heart and lung auscultations seemed to be normal. The peripheral arterial pulsations on both sides of the upper and lower extremities were symmetric, but the pulsations of the popliteal and dorsalis pedis arteries, which were absent on the first admission, were present but weak, The laboratory results included WBC, 6,300/cu mm with eosinophils 14% (910/cu mm); hematocrit, 39.3%; ESR, 20 mm/hour; creatinine kinase, 84.0 U/L; asparate transaminase, 15.4 U/L; lactic dehydrogenase, 158.4 U/L; total cholesterol, 109 mg/dl; total protein, 7.2 mg/dl and uric acid 5.9 mg/dl.

The electrocardiogram (Fig. 2) displayed a sinus rhythm with abnormal Q wavese and an elevation of more than 1 mm in the ST segments in lead I, AVL and V2-5. Two weeks after the substernal chest pain episode, left ventriculography and

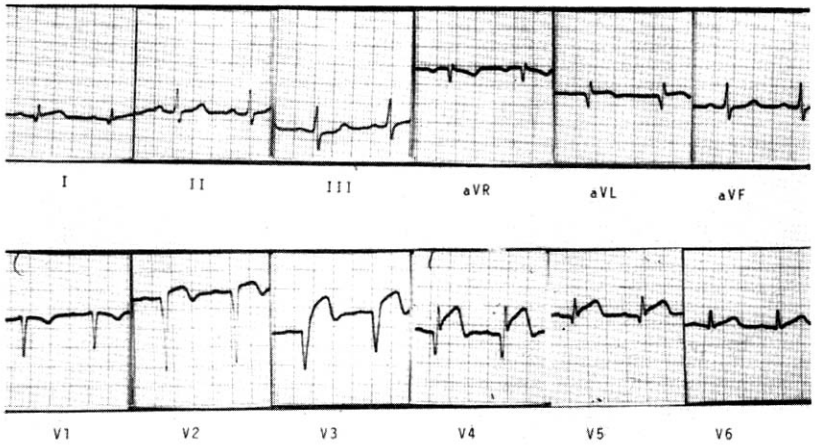

Fig. 2. *Electrocardiogram, taken on admission, shows elevation of ST segments and abdominal Q waves in lead, I, a VL and V2–5 which represents anterior myocardial infarction.*

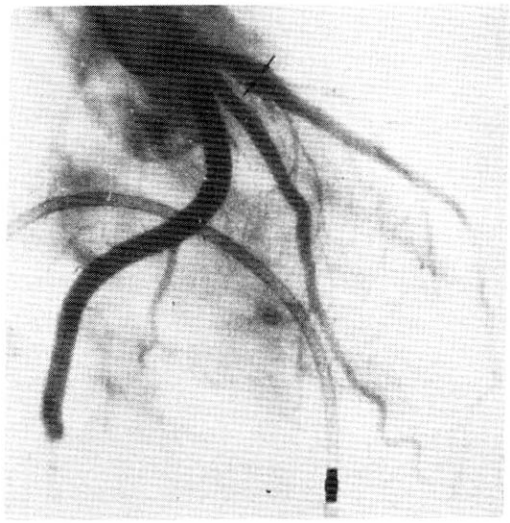

Fig. 3. *Left coronary arteriogram in the right anterior obilque projection shows a partial segmental narrowing of the proximal left anterior descending artery (arrow).*

coronary angiography were performed, and the left ventriculogram revealed hypokinetic or akinetic movement in the anterior region.

Right and left coronary cineangiography (Fig. 3) in the right and left anterior oblique projections demonstrated partial segmental occlusion of the proximal left anterior descending artery (42% of the luminal area), complete occlusion of the first diagonal brach of the left anterior descending artery in the distal portion, and irregular and tortous contour of the right coronary artery without obvious luminal narrowing. Conservative management included the administration of nitrate, beta-blocker and calcium channel blocker. The patient was discharged on the 17th day with no recurrence of chest pain.

COMMENTS

Since Dr. Leo Buerger first described thromboangitis obliterans (Buerger's disease) as a progressive peripheral insufficiency occuring primarily in young male smokers, many authors have reported on this unique clinicopathologic disease entity. This disease has a geographic and ethnic prevalence, especially among Indians, Jews and Orientals. In Korea, Buerger's disease is a much more prevalent peripheral vascular occlusive disease than arteriosclerotic obliterans[4]. Although there have been occasional reports of involvement of the mesenteric and cerebral arteries and rarely of the coronary arteries[3,8], myocardial infarction, as a clinical feature, is an extremely rare event in Buerger's disease[9]. However, Shionoya et al[7,10], reported that on autosy a patient Buerger's disease showed atheromatous changes in the coronary and abdominal aorta, and also that patients with Buerger's disease were susceptible to the arteriosclerotic process.

Our patient's electrocardiomgram showed an extensive anterior wall infarction, and the coronary angiogram revealed a partially occluded proximal portion of the left anterior descending artery, further proof of an anterior myocardial infarction. It is widely accepted that a myocardial infarction is the result of either a totally or critically occluded (less than 25% of the luminal area) coronary artery[11].

Recently, several mechanisms have been proposed as possible cause of the myocardial infarction in patients with normal or near normal coronary arteries. In 1983, Rosenblatt and Selzer[12] suggested three possible mechanisms to explain such a discrepancy; 1) a myocardial infarction is produced by a coronary arterial spasm; 2) coronary lesions may cause myocardial infarctions, but their presence is not noted on a subsequent coronary angiogram; and 3) an occlusive lesion, present at the time of infarction, has disappeared due to recanalization or lysis of the thrombus. Coronary arteriographic studies in our patient revealed a 42% luminal narrowing of the left anterior descending artery, which was greater than the critical level of acute myocardial infarction. Thus we suspected that other mechanisms, such as coronary vasospasm or recanalization, might be the cause of a transmural infarction with arteriosclerosis.

Another finding which supports the suggestion of a coronary vasospasm, as a cause of an acute myocardial infarction, is Raynaud's phenomenon which was observed in the patient prior to the myocardial infarction. Ciraulo et al[13], reported that migrain or Raynaud's phenomenon was more frequently associated with coronary vasospasm and resulted in myocardial infarction in patients with normal coronary arteries. Based on these reports, we sugges that the coronary vasospastic phenomenon was involved in this case.

The etiology of Buerger's disease, though still obscure, is correlated with cigarette smoking. Most of the patients cited in the literature were

cigarette smokers who experienced a slowdown in the progression of disease inwhen they stopped smoking.

In 1983, Adar et al[5], reported that patients with Buerger's disease exhibited a cellular sensitivity to human type I or type III antigens (or both) and developed antibodies to anticoagulants, suggesting that immunologic factors were involved in the etiology of the disease. Other aspects of the disease are the genetic predisposition and the noted prevalence of HLA-A9 and HLA-B5 in many affected persons[14] and of HLA-A9 and HLA-W10 in Japanese patients.

In 1985, Ferguson et al[6], reported a case of Buerger's disease associated with idiopathic hypereosinophilia and suggested that eosinophilia might be involved in the pathogenesis of Buerger' s disease. They proposed a wherein long term smoking incited an allergic reaction with hypereosinophilia in some patients. It is interesting to note that on both admission, our patient presented hypereosinophilia with a normal IgE level and other immunologic data, but these results might not be consistent with Ferguson's report. The treatment of Buerger's disease is rather simple & specific; the patients should stop smoking. When our patient stopped smoking and followed conservative management for the myocardial infarction, his chest pain subsided. In conclusion, a myocardial infarction is a possibility in Buerger's disease due to coronary vasospasm and coronary arteriosclerosis.

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