Isolated Septal Perforator Block In A Patient With Unstable Angina: A Case Report *

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SUMMARY:

Isolated septal perforator narrowing as a cause of significant subjective and objective myocardial ischemia is rare. We are reporting one such case of isolated II septal perforator narrowing resulting in rest angina, and positive stress ECG and stress echocardiography.

The ventricular septum receives more than two-thirds of its blood supply from septal perforators originating from the left anterior descending artery. In most cases the major septal branch is the second septal perforator which may reach a diameter of more than 2.5mm. Obstruction of the large septal perforators in patients with diffuse coronary artery disease is common, but obstruction of septal perforators in the absence of significant narrowing in the other branches, is rare, being reported only twice [1, 2]. We are reporting one such case who was significantly symptomatic because of isolated septal ischemia.

SNL, a 55-year old non-diabetic, non-hypertensive, smoker and a patient of effort angina (NYHA-Class II) presented with chest pain at rest of 30 minutes duration, not being relieved by sublingual nitrates. He had a blood pressure of 130/90mmhg, heart rate of 114/mt. and a loud S^4 gallop with grade II/VI systolic murmur at apex. His serum cholesterol was 278mg%, HDL was 35mg% and S. Triglyceride was 300mg%. ECG showed RBBB with 1mm ST depression in V_5-V_6 during pain, he was kept on morphine, nitroglycerine intravenously and Inderal 40mg qid. He was pain free after 2 hours with normal cardiac enzyme pattern during this episode. Two dimensional echocardiogram, done next day, had no wall motion abnormality, or mitral regurgitation. He was discharged without any remaining clinical signs. Stress test done after 2 weeks showed 1.0mm upsloping ST depression, 0.08 seconds after J point with angina at maximum achieved heart rate of 154/mt. S-T segment changes lasted till 2 minutes in post exercise phase. Figure-1. S_4 gallop and a grade II/VI systolic murmur accompanied chest pain. Immediate post exercise two dimensional echocardiogram showed hypokinesia of septal segment in long and short axis views, with mild MR on colour flow imaging.

Coronary angiography done by Judkins technique showed 99% narrowing of a prominent 2nd septal perforator without any disease in other vessels. Left ventriculogram in the lateral view showed hypokinetic mid and apical septum with post ectopic normokinesia.

Generally, barring a small zone of the septum near the posterior interventricular sulcus and the region of the antioventricular node, which is supplied by the posterior descending artery, most of the septal blood supply comes from the left anterior descending via the septal perforators[3]. In some cases the first septal branch is unusually large and runs parallel to the LAD

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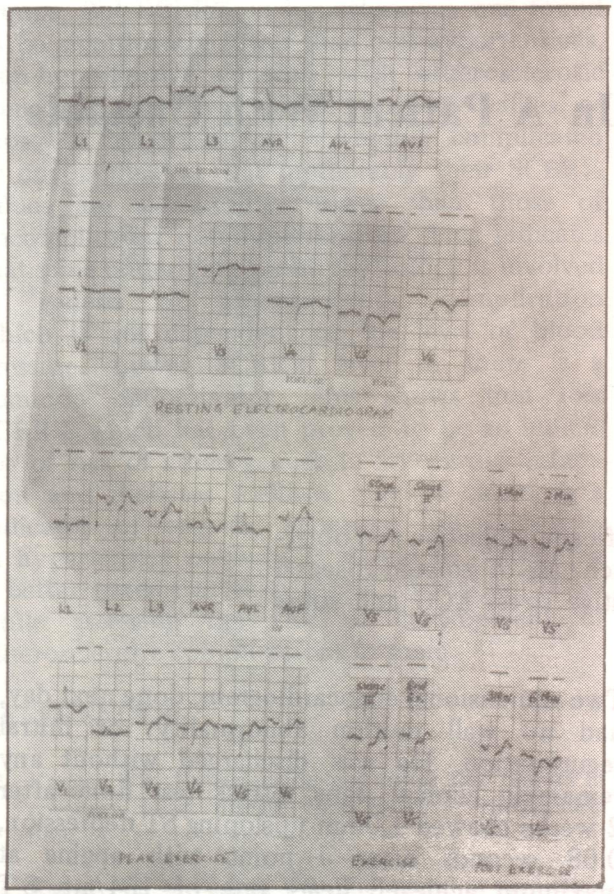

Fig. 1 Treadmill Stress Test showing the results of exercise ECG.

(dominant first septal artery) whereas in others there may be a bifurcation of the left anterior descending artery, one branch solely supplying the septum (Dual LAD) [3]. Obstruction of these major septal arteries may cause considerable myocardial ischemia.

In our case there was isolated severe obstruction of a "true" septal perforator, instead of a dominant first or a dual LAD. The patient had significant angina with objective evidence of coronary artery disease in the form of 1) Resting RBBB with primary ST-T changes 2) positive exercise ECG 3) positive exercise echocardiogram. Since there were no lesions in the other coronary arteries and since we routinely use nitroglycerine before angiography to rule out spasm, it may be inferred that septal artery obstruction was the sole cause of the manifestations of myocardial ischemia, ECG changes occurring in

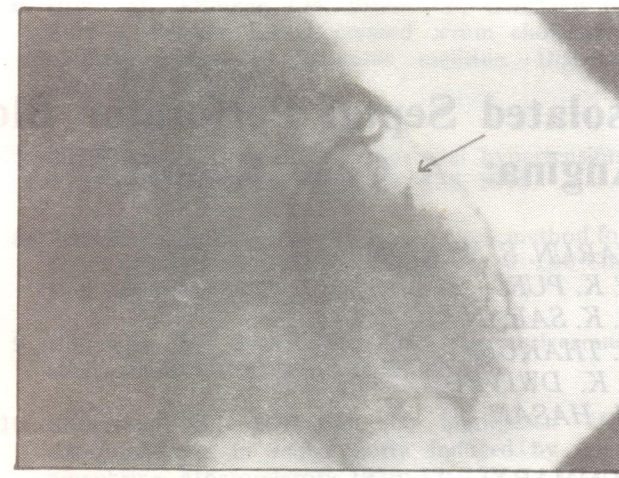

Fig. 2. Left Coronary Angiogram (30^{o} RAO) showing 99% narrowing of II perforator (arrow).

the lateral wall could not help localize the area of transient ischemia on treadmill test and this was better achieved by exercise echocardiogram, stressing the superiority of this test over exercise ECG.

Such isolated septal obstruction as the cause of patients symptoms has been reported only twice: Once by Nishimura et al[1] who in 1984 reported the 2-dimensional echocardiographic appearance of septal infarct, secondary to isolated occlusion of the first septal perforator. In the second report of 4 cases by Plokker et al (1988)[2], all patients had isolated septal perforator obstruction with abnormal septal motion and symptoms of coronary artery disease. One had a positive stress ECG and thallium scintigraphy revealing a perfusion defect in the proximal septum. One had a negative stress ECG but a positive stress thallium. One had a ventricular septal defect oblong to the course of the septal perforator and the tissue surrounding the defect was found to be an old myocardial infarction on biopsy. No evidence of infarction in other areas of the left ventricular free wall was seen.

Hence it appears that isolated obstruction of the septal arteries may produce considerable symptoms of myocardial ischemia and patients are at risk of myocardial infarction. Our case, therefore, adds to the rationale of including septal artery revascularization in an attempt to achieve complete revascularization [5 6]

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