Editorial

PRINZMETAL'S VARIANT ANGINA

Variant angina was first described by Prinzmetal et al. in 1959. The clinical features of this syndrome being markedly different from angina pectoris made coronary artery spasm to come up as a probable cause. Animal experiments demonstrated that although coronary blood flow is regulated by the metabolic requirements of the myocardium, it is greatly influenced by neural mechanism also. Coronary arteriography further gave evidence of coronary artery spasm.

The larger segments of the coronary arteries also called the conductance vessels are found on the epicardial surface. The smaller pre-capillary vessels or resistance vessels lie within the myocardium. In normal states the larger vessels have no contribution to coronary vascular resistance. The smaller vessels show changes in their lumen with fluctuations in resistance (Braunwald et al., 1976). Neurogenic stimuli cause the large conductance vessels to constrict giving an increased coronary vascular resistance. The basic regulatory mechanism of the coronary blood flow is the metabolic requirement of the myocardium. Experiments showed that metabolic products accumulated when the coronary blood flow was reduced or when the myocardial activity was increased. These products had a local control on the coronary vascular tone and relaxed the smooth musculature in the pre-capillary arterioles which in turn reduced the resistance and increased the coronary blood flow. The substances suggested are oxygen (Daniell and Bagwell, 1968; Gellai et al., 1973), Carbon-dioxide, Hydrogen-ion, Potassium-ion (Driscol and Berne, 1957), Lactic acid, Prostaglandins (Nakano, 1968; Higgins et al., 1971; Afonso et al., 1974) and adenosine.

The neural control of the coronary artery flow has also been studied in experimental animals. The alpha adrenergic receptors which richly supply the large coronary arteries have a profound influence on their blood flow. Alpha adrenergic blockade prevents coronary artery vaso-constriction whereas stimulation of the parasympathetic nervous system may indirectly stimulate the sympathetic nerves leading to vaso-constraction of the large coronary arteries.

The clinical features of variant angina present themselves as chest pain at rest rather than the typical pre-cordial pain on exertion or emotional stimulation of angina pectoris, where myocardial ischaemia results as the increased oxygen demand is not met with. The pain recurs at the same time of the day usually in the early hours of the morning awakening the patient from his sleep. The ECG recorded during pain shows ST elevation in the inferior leads, various degrees of A-V block and occasionally ventricular arrythmias. This is due to a sudden reduction of myocardial oxygen supply due to the coronary artery spasm giving a transmural ischaemia and S-T elevation. A coronary arteriogram performed on a group of such patients by Prinzmetal et al showed severe stenosis of the proximal end of one or more of the large coronary vessels. It was therefore proposed that coronary artery spasm at the site of the stenosis gave transmural myocardial ischaemia which lead to chest pain and S-T elevation in the ECG. Myocardial scintigrams demonstrated a deficit of tracer up-take in the transmural region during an episode of pain with normal results in the same patient when there was no pain. In addition the coronary blood flow when measured by the thermodilution technique, showed a considerable fall during chest pain and was normal after the pain was over (Ricci et al., 1978).

A sub-group of these patients also showed atheromatous changes alongwith the coronary artery spasm. A variation in the symptoms was encountered in this group. Chest pain on exertion was a feature of the early stage of the clinical course which was later followed by pain at rest. Usually a history of myocardial infarction was present and the S-T elevation was seen in the antero-lateral leads.

Nitroglycerin given sub-lingually or intravenously produces immediate relief in all types of angina. Beta-adrenergic blockers are highly effective in typical angina pectoris. In variant angina they may be extremely harmful (King et al., 1973) as the Alpha receptors remain unopposed giving coronary artery vaso-constriction. On the contrary Beta-adrenergic stimulation produces coronary artery vaso-dilatation in variant angina. Verapamil and

Nifedipine both being potent coronary vasodilators, prevent coronary spasm of variant angina (Sollberg et al., 1978; Muller and Gunther, 1978). Verapamil, a papaverine derivative and Nifedipine, a dehydropyridine derivative, inhibit the contraction of smooth muscles by decreasing the cellular up-take of calcium (Fleckenstein et al., 1975).

Coronary artery bypass relieves the pain of typical angina pectoris but is of doubtful value in variant angina. Spontaneous graft closure is commonly found in these patients and spasm at the site of insertion of anastomosis may reduce the flow to an extent of coronary thrombosis.

The more sophisticated techniques of diagnosis now developed can help in identifying the different groups of patients with chest pain and further selection of an appropriate therapy according to the factor causing the symptoms.

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