Magnesium in Coronary Artery Disease

Lloyd T. Iseri

Department of Medicine, University of California, Irvine, California College of Medicine, Orange

Summary

Magnesium in coronary artery disease is reviewed with regard to its role in the pathogenesis of arteriosclerosis, coronary spasm, myocardial function, acute myocardial infarction and ventricular arrhythmias. Experimentally, magnesium depletion potentiates and supplementation retards the effect of atherogenic diets. Evidence from human studies is circumstantial. Reactivity of arterial smooth muscle is enhanced by low and suppressed by high magnesium media. Evidence that magnesium depletion may initiate coronary spasm is provided by dog and retrospective human studies.

Although experimental magnesium deficiency disrupts myocardial mitochondria, there are no studies which show that magnesium deficiency will lead to cardiac failure or that replacement will improve cardiac function. It is known that an infarcted or ischaemic myocardium loses magnesium and this may be the basis for ventricular arrhythmias. Coronary occlusion in a previously magnesium-depleted heart will result in a larger area of necrosis and ischaemia. The fall in serum magnesium in acute myocardial infarction is probably due to the formation of soap in fat cells undergoing catecholamine lipolysis.

Ventricular fibrillation in coronary artery disease will respond to parenteral magnesium, even in the presence of normal serum concentrations.

This article highlights some of the points brought out by Seelig and Heggtveit (1974) in their review on magnesium interrelationships in ischaemic heart disease, and discusses some of the more recent data regarding this relationship, in particular the role of magnesium in: (a) the pathogenesis of arteriosclerotic coronary artery disease; (b) coronary artery spasm; (c) myocardial function; (d) acute myocardial infarction; and (e) stabilisation of tachyarrhythmia.

1. Role of Magnesium in Pathogenesis of Arteriosclerotic Heart Disease

1.1 Experimental Atherogenesis

Ever since Kruse et al. (1933) showed that young dogs fed low magnesium (0.08%) high fat (butter 8%) diets developed esterified cholesterol plaques in their arterial walls, numerous studies have been reported confirming this observation. On the other hand, Vitale et al. (1961) showed that unsaturated
corn oil was not atherogenic even when magnesium was deficient, and that high magnesium (0.2%) in the atherogenic diet (casein and saturated cottonseed oil) protected the animals against development of atherosclerosis. When calcium levels were kept low, serum β-lipoproteins were also prevented from rising and a further protection against atherosclerosis was observed (Vitale et al., 1959) [fig. 1]. The role of magnesium in these atherogenic diets may be additive and provocative since it has been found that diets rich in fat interfere with magnesium absorption (Seelig and Heggtveit, 1980).

Other methods which deplete magnesium appear to increase blood lipids and the tendency to arteriosclerosis; for example, excessive intake of vitamin D which causes renal retention of calcium and renal loss of magnesium, accelerates the development of atherosclerosis (Seelig, 1975).

The role of magnesium in experimental atherogenesis can be summarised as follows:

1. Magnesium deficiency potentiates atherogenesis
2. High magnesium supplements suppress atherogenesis
3. Magnesium deficiency causes little change in total lipids or free cholesterol but raises esterified cholesterol
4. High magnesium and low calcium supplements lower β-lipoproteins and further protect against atherogenesis.

1.2 Human Studies

Evidence from human studies implicating magnesium deficiency as a factor in development of atherogenic lesions is circumstantial. For example, infants given toxic amounts of vitamin D frequently have diffuse coronary atherosclerosis associated with their tendency to lose magnesium and retain calcium (Linden, 1974). Also, infants born to eclamptic and hyperparathyroid mothers who are presumably depleted of magnesium, frequently show arteriosclerosis of the small coronary arteries (Seelig and Heggtveit, 1980).

As might be expected, patients manifesting arteriosclerotic coronary artery disease and hyperlipidaemia do not consistently show hypomagnesaemia. Possibly the only positive correlation reported was by Bersohn who found lower serum cholesterol, higher serum magnesium and a lower incidence of ischaemic heart disease in Bantus compared with higher serum cholesterol, lower serum magnesium and a higher incidence of coronary artery disease in the white South African population (Bersohn and Oelofse, 1957).

A recent study attempting to correlate serum magnesium with coronary artery disease has shown that significantly low magnesium levels are found in angiographically proven severe coronary artery disease. Possibly the only positive correlation reported was by Bersohn who found lower serum cholesterol, higher serum magnesium and a lower incidence of ischaemic heart disease in Bantus compared with higher serum cholesterol, lower serum magnesium and a higher incidence of coronary artery disease in the white South African population (Bersohn and Oelofse, 1957).

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