The hyperparathyroid heart mimicking acute myocardial infarction

JAMES KER

Summary

Left ventricular hypertrophy is a common complication of primary hyperparathyroidism.

Numerous disturbances of myocardial physiology have been described as a result of excess parathyroid hormone action. In this brief communication, another phenotype of the hyperparathyroid heart is proposed, the ‘pseudo-myocardial infarction hyperparathyroid heart’.

Patients with primary hyperparathyroidism have an increased risk of death due to cardiovascular disease, and hypertension and/or left ventricular hypertrophy are frequently present in these patients.

It has been shown that parathyroid hormone-induced calcium overload in cardiomyocytes has adverse effects on myocardial energy metabolism, structure and function. Furthermore, cardiac calcification is common, and parathyroid hormone itself is able to induce left ventricular hypertrophy. The fourth Tromso study included 27,159 patients and it was clearly shown that in patients over 60 years old, parathyroid hormone was a significant predictor of left ventricular hypertrophy.

Other observed adverse cardiovascular effects include left ventricular diastolic dysfunction, valvular calcifications and bright echoes in the myocardium, indicative of calcified deposits.

The hypertrophy in primary hyperparathyroidism can be symmetrical or asymmetrical, and it can even assume the phenotype of hypertrophic cardiomyopathy. Demers et al described a case of primary hyperparathyroidism with diffuse calcium deposits in the myocardium, coronary arteries, kidneys and lungs, with concentric left ventricular hypertrophy and endocardial fibrosis. They coined the term ‘hypercalcaemic cardiomyopathy’.

Fig. 1 shows the electrocardiogram of an asymptomatic 60-year-old male with previous hyperparathyroidism due to an adenoma, treated with surgical excision. Further important background information is that he had well-controlled hypertension on 10 mg amlodipine per day. Note the striking ST-elevation, typical of acute anterior myocardial infarction. However, no infarction was present and the electrocardiographic picture was persistent. Fig. 2 is a transthoracic echocardiographic image, demonstrating left ventricular hypertrophy with a sigmoid septum.
Discussion
Stefenelli et al. demonstrated that in normotensive patients, left ventricular hypertrophy was reversible after parathyroidectomy. However, regression of hypertrophy did not occur in the hypertensive patient after parathyroidectomy.

Another phenotype of the hyperparathyroid heart is proposed, the ‘pseudo-myocardial infarction hyperparathyroid heart’. I suggest that this electrocardiographic picture mimicking acute anterior myocardial infarction was due to the striking left ventricular hypertrophy causing the sigmoid septum. It is also proposed that this conspicuous degree of hypertrophy was due to parathyroid hormone action on the left ventricular myocardium. As discussed above, current data suggest that the hypertensive patient with treated hyperparathyroidism does not experience regression of left ventricular hypertrophy, even with adequate blood pressure control, as seen in this case.

It is hoped that this brief communication will focus attention on the profound physiological effects that parathyroid hormone has on the human heart, and influence any possible future explanations of pathophysiological alterations induced by increases in parathyroid hormone.

References