Plasma atrial natriuretic peptide in patients with acute myocardial infarction: effects of streptokinase

Sir,

Phillips et al studied plasma concentrations of atrial natriuretic peptide after acute myocardial infarction (1989;61:139–43). They did not report a significant correlation between plasma concentrations of atrial natriuretic peptide and peak concentrations of creatine kinase, which is an index of the severity of infarction. In their study, blood was sampled for the determination of atrial natriuretic peptide on the morning after admission, which was, as we understand, at least 0–32 hours after the onset of symptoms.

The exact timing of blood sampling for atrial natriuretic peptide is of great importance. We measured atrial natriuretic peptide concentrations at fixed times during 48 hours in 38 patients who were admitted to the coronary care unit within 4 hours 25 minutes after the onset of symptoms. Three hours after admission, the mean atrial natriuretic peptide concentration was significantly lower than it was on admission. Thereafter, atrial natriuretic peptide concentrations rose till 15 hours after admission. Both the atrial natriuretic peptide value on admission and the individual mean atrial natriuretic peptide value during the study period of 48 hours were significantly correlated with the maximum creatine kinase value.

We agree with the hypothesis of Phillips et al that acute myocardial dysfunction after myocardial infarction, by raising atrial pressures, causes a release of atrial natriuretic peptide from atrial storage granules. The ensuing decrease in atrial natriuretic peptide concentrations found in our study may possibly be attributed to depletion of these storage granules. At this stage, circulating atrial natriuretic peptide concentrations will decrease despite increased intracardiac pressures. Subsequently atrial natriuretic peptide is synthesised at a greater rate causing its concentrations in the blood to rise again. Our results imply that the correct interpretation of atrial natriuretic peptide values after acute myocardial infarction depends on the timing of blood sampling.

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Reference


This letter was shown to the authors, who reply as follows:

Sir,

We found that plasma concentrations of atrial natriuretic peptide were higher in patients with acute myocardial infarction who were not treated with thrombolysis than in similar patients admitted with non-ischaemic chest pain and patients with myocardial infarction treated with streptokinase. Patients with ischaemic chest pain had intermediate concentrations of atrial natriuretic peptide. The finding of raised plasma atrial natriuretic peptide in the acute stages of myocardial infarction was confirmed by Tan et al and others.1

References

Both Tan et al and Svanegaard et al found that initially raised plasma atrial natriuretic peptide concentrations in patients with myocardial infarction fell soon after admission. The extent of the fall found by Tan et al is unclear. Despite this fall plasma atrial natriuretic peptide concentrations remained raised in the patients reported by Svanegaard et al especially when cardiac failure was present. This fall in plasma atrial natriuretic peptide may reflect the beneficial effect of treatment (for example, bed rest, diuretics, glyceryl trinitrate, analgesia, thrombolysis) on left ventricular function as well as the initial depletion of atrial storage granules. Not enough detail is given by these other workers for us to comment further on the cause of the fall. Depletion of storage granules, however, seems to be an unlikely mechanism, because plasma atrial natriuretic peptide concentrations fell in all patients irrespective of the initial atrial natriuretic peptide concentration.

We agree with Tan et al that both the timing of the blood sample and the degree of infarction are important variables in interpreting plasma atrial natriuretic peptide concentrations. Indeed we found that in an experimental model of myocardial infarction the concentration of plasma atrial natriuretic peptide correlated well not only with the haemodynamic changes but also with the degree of infarction measured histologically. In this experimental model it was also possible to show an inverse relation between plasma atrial natriuretic peptide and atrial natriuretic peptide. That we did not find a relation between peak concentrations of cardiac enzymes and plasma atrial natriuretic peptide in our human study was probably the result of the confounding effects of streptokinase treatment, which increased peak enzyme concentrations but lowered plasma atrial natriuretic peptide, and the inclusion of patients with ischaemic chest pain who had normal concentrations of cardiac enzyme but intermediate concentrations of plasma atrial natriuretic peptide, as well as the variable times of blood sampling (7–32 hours after admission). This underscores the difficulty of studying and interpreting the dynamics and mechanism of atrial natriuretic peptide release in patients with heart disease.

The finding of raised concentrations of plasma atrial natriuretic peptide during the acute stage of myocardial infarction before appreciable volume expansion could occur suggests that acute myocardial dysfunction alone may raise plasma atrial natriuretic peptide concentrations, probably by reducing ventricular compliance and increasing atrial pressures. This may be a beneficial homeostatic response that acts to reduce cardiac preload and limit salt and water retention during the acute stages of myocardial ischaemia.

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References

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**Pulmonary capillary haemangiomatosis**

Sir,

A registry is being formed to document and study cases of pulmonary capillary haemangiomatosis, an exceptionally rare disease (only eight cases have been described) that leads to proliferation of capillaries in the lung and pulmonary hypertension. Anyone knowing of patients with this disorder is asked to contact either (1) CANADA and EUROPE: Dr David Langleben, Jewish General Hospital, 3755 Côte St Catherine, Montreal, Quebec H3T 1E2, Canada; or (2) USA, MIDDLE EAST, and ORIENT: Dr Carl W White, Division of Pulmonary Disease, The Children’s Hospital, 1956 East 19th Avenue, Denver, Colorado 80218-1088, USA. Strict confidentiality will be maintained and a newsletter will be circulated to all contributors.

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