Letters to the Editor

Acute heart failure and transient low voltage in electrocardiogram after massive catecholamine release from a phaeochromocytoma

The association between acute heart failure and phaeochromocytoma is well established1-4. In earlier reports, a wide variety of ECG changes has been described. The ECG may remain normal or show elevation or depression of the ST segment, flattening or inversion of the T-wave, all suggesting acute ischaemia1-4.

We report a patient who developed generalized low voltage, as demonstrated on the ECG, parallel to the development of transient left ventricular failure. To the best of our knowledge, this has not been described before in phaeochromocytoma-associated catecholamine storm.

A 46-year-old female was admitted to hospital for an elective tympanoplasty. Her past history was uneventful. The operation, however, had to be interrupted because of persistent tachycardia and hypertension. Soon after she showed signs of severe respiratory distress and was admitted to the intensive care unit (Day 1). On admission, the patient had signs of alveolar pulmonary oedema. The heart rate was 145 beats min−1 and blood pressure 90/70 mmHg. The chest radiogram showed a normal sized heart and confirmed alveolar pulmonary oedema. Echocardiography revealed severe diffuse left ventricular hypokinesia with an ejection fraction (EF) of 20%.

Phaeochromocytoma was suspected soon after admission and computed tomography revealed a solid tumour on the left adrenal gland. The diagnosis of an adrenaline- and noradrenaline-secreting phaeochromocytoma was confirmed by analysis of 24-h urinary excretion of catecholamine metabolites. On questioning after recovery, the patient gave a 5-year history of several episodes of palpitation, sweating, pallor, and breathlessness lasting 10-15 min. Three months before the episode of acute heart failure she visited a physician following such an episode. However, on examination the patient had already recovered. Blood pressure and ECG (Fig. 1) were normal.

In contrast, the ECG taken on admission to the intensive care unit showed generalized low voltage but not pathological P-wave, Q-wave, ST-segment, or T-wave changes (Day 1 in Fig. 1). The sum of the QRS amplitudes measured from leads I, II, III, V1, and V6 revealed a decrease from the pre-operative value of 4-3 mV to 1-9 mV on Day 1. Serum creatine kinase activity was not increased, indicating that there was no myocardial necrosis to explain the low voltage on the ECG.

Pulmonary oedema and hypotension were treated with mechanical ventilation, dopamine, dobutamine and diuretics. The left ventricular failure improved rapidly and mechanical ventilation and inotropic support could be stopped on Day 4. The low voltage on the ECG showed parallel improvement with left ventricular function (Fig. 1). Ejection fraction was within normal limits (65%) on Day 12. The ECG voltage slowly corrected and was 77% of the control value on Day 12 (4-3 mV vs 3-3 mV). The patient underwent uncomplicated adrenalectomy on Day 25, and was discharged well a week later. Four weeks after adrenalectomy (Day 53), the ECG voltage was correct.

Our findings, together with the earlier reports, indicate that a massive but short-lasting release of catecholamines causes acute transient injury to the myocardium with a spontaneous tendency to complete recovery within 1-2 weeks without removal of the tumour.

The most interesting feature in our patient was the development of a transient, generalized low voltage on the ECG, parallel to the development of transient left ventricular failure. The mechanism of low voltage is very probably related to acute and massive catecholamine release, causing a direct toxic effect on myocytes. Low voltage is hard to explain by mechanisms other than an inability of the myocytes to build up the normal energy-requiring membrane potential. The mechanism of catecholamine-induced myocardial injury is probably multifactorial5. Increased calcium influx to the myocytes, damage by oxidation products, and coronary vasoconstriction have been proposed.

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References


Low total cholesterol and high total mortality in patients with coronary heart disease

The title (see above) and conclusion by S. Behar et al10 are extremely provocative!

Only patients who were excluded from the Bezafibrate Infarction
Prevention (BIP) study (n = 3122) and who had complete mortality data were included in the analysis. How many patients were excluded with incomplete mortality data?

My first impression is that the selection of patients excluded high risk patients in the high cholesterol group! Indeed, in my view, the 3122 excluded patients are at the highest risk.

In the 'control high cholesterol' group the mean cholesterol was 230 (SD 40), the mean HDL was 39.1 (SD 10.9). So, in this group a substantial number of subjects must have fulfilled the entry criteria (cholesterol >180 <250; HDL <45) for the BIP study. How many of these patients were not included in the study and for what reasons? Unless a sufficient explanation from the authors, the report does not convince me.

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Reference

A reply

Dr Hugo Ector suggests that the title and conclusion of our article are extremely provocative. However, this is not the first article linking low total cholesterol levels with higher risk for non-cardiac death. Davey Smith in the Editorial published in the same issue, has thoroughly discussed the major studies over the last 20 years on this topic.

The data of our study, the first one demonstrating higher non-cardiac mortality among coronary patients with low total cholesterol, is not in contradiction to a recent trial showing that reducing total cholesterol has a favourable effect on mortality. In the 4S study the decrease in mortality was achieved by reducing high cholesterol levels, while in our article we reported mortality in patients who had spontaneously low levels of cholesterol. Furthermore, we have emphasized that low total cholesterol, resulting from diet or medical treatment, was not associated with excess mortality.

Dr Ector suspects that high risk patients with high cholesterol have been excluded from our article.

The BIP Registry included more than 15 000 age-eligible (45–74 years) patients of whom 3122 were included in the ongoing BIP study. Sixty-two percent of patients were excluded from BIP on the basis of lipid profile (TC < 180 or >250 mg. dl⁻¹; HDL-C >45 mg. dl⁻¹; TG >300 mg. dl⁻¹), 45% for medical reasons and 4% refused to sign the informed consent form (some patients had more than one exclusion reason). From these data it is clear that patients in the low total cholesterol group (n = 595; TC < 160 mg. dl⁻¹) a priori excluded from the BIP study, were compared to patients with TC >160 mg. dl⁻¹, including those with hypercholesterolaemia. Figure 1 depicts the distribution of the total cholesterol among all BIP Registry patients. Table 1 represents the lipid profile and mean age of 11 563 screened patients excluded from BIP and of the 805 patients excluded from the analysis due to lack of complete mortality data. Figure 2 shows the distribution of total cholesterol in the above two groups. Thus, laboratory findings were similar among both groups and therefore it seems improbable that the fate of the patients excluded from analysis (n = 805), if known, could alter the results of the study.

One of the limitations of our study is its short follow-up time (mean 3.3 years). Since the preparation of the article, we have updated our data to 5.2 years follow-up (range: 4–6). Table 2 reports the rates of cardiac and non-cardiac death in the two

Figure 1 Total cholesterol among all Bezafibrate Infarction Prevention Registry patients.

Table 1 Lipid profile of screened patients excluded from and patients included in the analysis

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<th>Excluded (n=805)</th>
<th>Included (n=11 563)</th>
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<tbody>
<tr>
<td>Age (mean ± SD) years</td>
<td>61 ± 7</td>
<td>60 ± 7</td>
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<tr>
<td>Total cholesterol (mg. dl⁻¹)</td>
<td>229</td>
<td>226</td>
</tr>
<tr>
<td>LDL-C (mg. dl⁻¹)</td>
<td>157</td>
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<tr>
<td>HDL-C (mg. dl⁻¹)</td>
<td>38.8</td>
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<tr>
<td>Triglyceride (mg. dl⁻¹)</td>
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<td>165</td>
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