Letters to the Editor

Non-invasive prediction of reperfusion and coronary artery patency by continuous ST segment monitoring in the GUSTO-I trial

In their paper, P. Klootwijk et al.[1] determine the sensitivity, specificity, and predictive value of continuous ST segment monitoring for patency and non-patency in infarct-related coronary arteries at 90 and 180 min following thrombolytic treatment. Taking into account the sensitivity and specificity data presented in Fig. 4 and using the equations given in the legend of Table 2, the calculated predictive values for patency and occlusion do not correspond with those published in Table 2. For example, in patients with TIMI grade 0 and 1 flow (indicating an occluded coronary artery) at 90 min following thrombolysis (Fig. 4) the ECG assessment correctly predicted the occluded vessel status in 44% of patients (true negative) but in the remaining 56% of patients the ECG evaluation led to a false prediction (false positive). In patients with TIMI grade 2 and 3 flow (indicating an open coronary artery) at 90 min after thrombolytic treatment (Fig. 4) the ECG assessment correctly predicted the patent vessel status in 75% and 85% of patients (true positive) but not in the remaining 25 and 15% of patients, respectively (false negative). The calculated predictive values from the ECG for reperfusion is 58 (vs 70 presented in Table 2) and that for occlusion is 68 (vs 58 given in Table 2). If the data of Fig. 4 are correct, the predictive value of the ECG as regards occlusion has to be superior than that for patency, which is in sharp contrast with the authors’ conclusion: ‘the technique appears better for prediction of patency than for occlusion’.

From a theoretical point of view, the predictive power of continuous ST segment monitoring in establishing reperfusion should be limited. It is well accepted that the achievement of reperfusion in the occluded infarct-related coronary artery is desirable for two reasons: on the one hand the reperfusion could result in salvage of the acutely ischaemic myocardium, on the other the reperfusion of the infarcted myocardium, which occurs within 12 h after necrosis, is supposed to prevent or diminish the remodelling of the left ventricle. Continuous ST segment monitoring is a useful method for evaluating ongoing ischaemia, but not for detection of reperfusion in infarcted myocardium (post-necrotic reperfusion). The accelerated Q wave development[2] and the evolution of transient post-ischaemic inverted T waves[3] were suggested as markers of reperfused infarcted myocardium. Therefore continuous ST segment monitoring per se, without taking into account the ECG signs of post-necrotic reperfusion will inevitably underestimate the true reperfusion rate during thrombolysis in patients with myocardial infarction.

K. SIMON
A. SZÉPVÖLGYI
A. BADICS
T. BÖHM
St. George’s Hospital.
II. Dept. of Internal Medicine.
Hungary

References


Diastolic filling in idiopathic dilated cardiomyopathy shows a good correlation to heart rate and ejection fraction but not to blood pressure as in healthy subjects

Recently Kangro and coworkers[1] published their Doppler investigation of healthy volunteers aged 50 years. The authors report a good correlation between heart rate and diastolic blood pressure with E/A ratios both in men and women, while left ventricular diameter correlated only in men with E/A ratios. Other investigators report that E-wave deceleration time correlates with left ventricular end-diastolic pressure in unselected patients of varying age[2]. Doppler indices of diastolic filling are therefore helpful in estimating ventricular loading conditions. In order to investigate diastolic Doppler indices in patients with impaired ventricular filling we undertook the following study.

We investigated 22 patients (all male) with invasively diagnosed idiopathic dilated cardiomyopathy (IDC) in sinus rhythm. Echocardiographic investigation was done as described recently[3] using a Vingmed CFM 800. The mean age of our population was 58 ± 11 years. The heart rate was 79 ± 15 beats.min⁻¹, systolic blood pressure 136 ± 19 mmHg and diastolic blood pressure 77 ± 9 mmHg. Echocardiographically measured left ventricular end-diastolic diameter (LVEDD) was 68 ± 6 mm, while left ventricular ejection fraction was 35% ± 6%. The peak velocity of early diastolic filling (E wave) was 0.82 ± 0.24 m.s⁻¹ while late diastolic filling (A wave) showed a mean peak of 0.76 ± 0.23 m.s⁻¹. The E/A ratio was 1.22 ± 0.61. Acceleration time of the E wave was 0.07 ± 0.02 s while the deceleration time was 0.15 ± 0.04 s.

To investigate whether similar correlations, as described in healthy volunteers[2], can be seen in patients with severely impaired left ventricular performance, we correlated the parameters mentioned above with diastolic Doppler findings. We found a good correlation between duration of E wave acceleration time and left ventricular ejection fraction (r=0.57, P<0.01). Furthermore, there was good correlation between heart rate and E wave acceleration and deceleration times (r=-0.53 and r=-0.61, P<0.02 and P<0.003, respectively). None of the other parameters showed a correlation to diastolic filling patterns.

Patients with dilated cardiomyopathy often have impaired left ventricular filling. This differs from observations in healthy volunteers whose blood pressure and heart rate show no correlation to the E/A ratio. There is, however, a good correlation between left ventricular ejection fraction and E wave acceleration time, and the higher resting heart rate is the shorter of the acceleration and deceleration times of the E wave in IDC patients.

Therefore we conclude that patients with dilated cardiomyopathy show a transmitral Doppler filling pattern that is different from that of...
prostacyclin analogue with similar effects. Although antagonism of platelet aggregation is regarded as a basic mechanism of the drug's action, observations have been made about the hyperreactivity of platelets during and after infusions of prostacyclin or iloprost in both animals[4] and patients[5]. Moreover, iloprost is able to attenuate the beneficial effect of rt-PA in patients with acute myocardial infarction, that reduce the rate of reperfusion and increase the rate of reocclusion[6]. Recently, Kovacs et al. observed platelet hyperreactivity in some patients with markedly enhanced coagulation during and after infusion of iloprost[5]. These findings challenge the view that antagonism of platelet function is an important factor of iloprost therapy and suggest that patients receiving iloprost are at risk of thromboembolic events, especially as patients are already in a prethrombotic condition[7]. These arguments support the use of inhaled nitric oxide when screening for acute vasodilator responsiveness in primary pulmonary hypertension[8].

References

Iloprost is a chemically stable prostacyclin analogue with similar effects. Although antagonism of platelet aggregation is regarded as a basic mechanism of the drug's action, observations have been made about the hyperreactivity of platelets during and after infusions of prostacyclin or iloprost in both animals[4] and patients[5]. Moreover, iloprost is able to attenuate the beneficial effect of rt-PA in patients with acute myocardial infarction, that reduce the rate of reperfusion and increase the rate of reocclusion[6]. Recently, Kovacs et al. observed platelet hyperreactivity in some patients with markedly enhanced coagulation during and after infusion of iloprost[5]. These findings challenge the view that antagonism of platelet function is an important factor of iloprost therapy and suggest that patients receiving iloprost are at risk of thromboembolic events, especially as patients are already in a prethrombotic condition[7]. These arguments support the use of inhaled nitric oxide when screening for acute vasodilator responsiveness in primary pulmonary hypertension[8].

References

Iloprost is a chemically stable prostacyclin analogue with similar effects. Although antagonism of platelet aggregation is regarded as a basic mechanism of the drug's action, observations have been made about the hyperreactivity of platelets during and after infusions of prostacyclin or iloprost in both animals[4] and patients[5]. Moreover, iloprost is able to attenuate the beneficial effect of rt-PA in patients with acute myocardial infarction, that reduce the rate of reperfusion and increase the rate of reocclusion[6]. Recently, Kovacs et al. observed platelet hyperreactivity in some patients with markedly enhanced coagulation during and after infusion of iloprost[5]. These findings challenge the view that antagonism of platelet function is an important factor of iloprost therapy and suggest that patients receiving iloprost are at risk of thromboembolic events, especially as patients are already in a prethrombotic condition[7]. These arguments support the use of inhaled nitric oxide when screening for acute vasodilator responsiveness in primary pulmonary hypertension[8].

References

Iloprost is a chemically stable prostacyclin analogue with similar effects. Although antagonism of platelet aggregation is regarded as a basic mechanism of the drug's action, observations have been made about the hyperreactivity of platelets during and after infusions of prostacyclin or iloprost in both animals[4] and patients[5]. Moreover, iloprost is able to attenuate the beneficial effect of rt-PA in patients with acute myocardial infarction, that reduce the rate of reperfusion and increase the rate of reocclusion[6]. Recently, Kovacs et al. observed platelet hyperreactivity in some patients with markedly enhanced coagulation during and after infusion of iloprost[5]. These findings challenge the view that antagonism of platelet function is an important factor of iloprost therapy and suggest that patients receiving iloprost are at risk of thromboembolic events, especially as patients are already in a prethrombotic condition[7]. These arguments support the use of inhaled nitric oxide when screening for acute vasodilator responsiveness in primary pulmonary hypertension[8].

References

Iloprost is a chemically stable prostacyclin analogue with similar effects. Although antagonism of platelet aggregation is regarded as a basic mechanism of the drug's action, observations have been made about the hyperreactivity of platelets during and after infusions of prostacyclin or iloprost in both animals[4] and patients[5]. Moreover, iloprost is able to attenuate the beneficial effect of rt-PA in patients with acute myocardial infarction, that reduce the rate of reperfusion and increase the rate of reocclusion[6]. Recently, Kovacs et al. observed platelet hyperreactivity in some patients with markedly enhanced coagulation during and after infusion of iloprost[5]. These findings challenge the view that antagonism of platelet function is an important factor of iloprost therapy and suggest that patients receiving iloprost are at risk of thromboembolic events, especially as patients are already in a prethrombotic condition[7]. These arguments support the use of inhaled nitric oxide when screening for acute vasodilator responsiveness in primary pulmonary hypertension[8].

References

Iloprost is a chemically stable prostacyclin analogue with similar effects. Although antagonism of platelet aggregation is regarded as a basic mechanism of the drug's action, observations have been made about the hyperreactivity of platelets during and after infusions of prostacyclin or iloprost in both animals[4] and patients[5]. Moreover, iloprost is able to attenuate the beneficial effect of rt-PA in patients with acute myocardial infarction, that reduce the rate of reperfusion and increase the rate of reocclusion[6]. Recently, Kovacs et al. observed platelet hyperreactivity in some patients with markedly enhanced coagulation during and after infusion of iloprost[5]. These findings challenge the view that antagonism of platelet function is an important factor of iloprost therapy and suggest that patients receiving iloprost are at risk of thromboembolic events, especially as patients are already in a prethrombotic condition[7]. These arguments support the use of inhaled nitric oxide when screening for acute vasodilator responsiveness in primary pulmonary hypertension[8].

References