Risk stratification of patients with right ventricular infarction: is there a need for a specific risk score?

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This issue features a report by Gumina et al. of 580 consecutive patients hospitalized with acute inferior or lateral wall myocardial infarction, 102 (18%) of whom were retrospectively identified as having had right ventricular infarction. These patients had worse in-hospital outcomes than those without right ventricular infarction, and as their Thrombolysis in Myocardial Infarction (TIMI) ST-elevation risk scores[1] increased, so did their in-hospital and long-term mortality rates. However, unlike the cohort of patients in whom the TIMI risk score was originally devised, there was no further increase in mortality with risk scores beyond 4–5[2] (Fig. 1).

The TIMI risk score[1] is one of several that have been developed to risk-stratify patients with acute ST-elevation infarction[3]. These scores have been devised and validated in cohorts of patients with left ventricular infarction, but have not been separately validated in patients with right ventricular infarction. Given the higher morbidity and mortality[4] and the unique pathophysiology of right ventricular infarction, it is important that these risk scores are also evaluated in these patients. The TIMI risk score is a simple bedside scoring system[1]. Eight variables are dichotomously classified as positive or negative, and a weighted score is assigned to positive variables (Fig. 1). The score was originally devised in 14 114 patients from the Intravenous NPA for the Treatment of Infarcting Myocardium Early (InTIME)-II trial, which compared lanoteplase with alteplase, and was validated in patients from the TIMI-9 trial, which compared hirudin with unfractionated heparin[1]. The 30-day mortality rate was <1% in patients with a score of 0, rising gradually and almost uniformly to a rate of 36% in patients with a score of >8, i.e. more than a 40-fold increase (Fig. 1). The two major
constituents of the TIMI risk score are age (≥ 75 scores 3 and 65–74 scores 2) and the haemodynamic state (a systolic blood pressure of <100 mmHg scores 3, a heart rate of >100 beats \( \text{min}^{-1} \) scores 2, and a Killip class of ≥ 2 scores 2).

Right ventricular infarction is recognized clinically by a history of myocardial ischaemia, together with findings of a raised jugular venous pressure, hypotension and clear lung fields, usually in association with inferior infarction and characteristic eletrocardiographic findings. Anterior infarction can also be associated with infarction of the right ventricle\(^5\), as its anterior wall may receive some of its blood supply from the left anterior descending coronary artery. Isolated right ventricular infarction is rare\(^5\).

No electrocardiographic criteria have been defined to identify anterior wall necrosis in the right ventricle\(^9\). On the other hand, there are well defined electrocardiographic criteria for the diagnosis of posterior right ventricular transmural ischaemia or infarction, occurring usually with inferior infarction. ST elevation in \( V_{2,3}R \) has been shown to have greater sensitivity and specificity for the diagnosis of right ventricular dysfunction than echocardiographic or radionuclide detection, and also identifies patients at risk of adverse early outcomes\(^7\). Right-sided ST elevation recorded at the time of hospital admission can, however, be relatively transient\(^8\), and the ST segment normalizes within 10 h in half of these cases. The other criteria used to diagnose right ventricular infarction are a right ventricular wall motion abnormality on echocardiography or on contrast or radionuclide ventriculography, and the haemodynamic features of a high right atrial pressure and abnormal waveform\(^7,9\).

Although right ventricular infarction was thought to be relatively benign in the past\(^10\), a recent meta-analysis of almost 4000 patients found that right ventricular infarction (predominantly defined by ST elevation in the right precordial leads) occurred in nearly half of the patients with inferior wall infarction\(^4\), and was associated with a threefold increase in the combined incidence of early mortality, cardiogenic shock, ventricular tachyarrhythmia and advanced heart block as compared with inferior infarction alone\(^4\). The association between right ventricular infarction and early mortality has been shown to be independent of the left ventricular ejection fraction in several studies\(^4,11,12\). It is important to note that right ventricular infarction is associated with substantial increases in mortality in elderly patients\(^11,12\).

The pathophysiology of right ventricular infarction may explain these findings. In left ventricular infarction, low blood pressure is associated with impairment of left ventricular function, whereas in right ventricular ischaemia or infarction, acute right ventricular distension within the restraining pericardium may flatten the interventricular septum towards the left ventricle during diastole, restricting left ventricular diastolic filling and increasing the left ventricular end-diastolic pressure\(^13\). Thus right ventricular infarction may compromise cardiac output, culminating in cardiogenic shock. The higher incidence of advanced heart block may be explained by the fact that the right ventricle and atrioventricular node share a common origin of blood supply. The reason for the increased incidence of ventricular arrhythmia is unclear. Sympathetic activation secondary to the low-output state is proarrhythmogenic, and it is possible that infarction of the right ventricle may itself be more arrhythmogenic than infarction that is confined to the left ventricle.

The Gumina study included patients with either ST elevation in the right-sided chest leads, proximal occlusion or thrombus of the right coronary artery on angiography, or echocardiographic right ventricular dysfunction\(^2\). Proximal occlusion of the right coronary artery is likely to be a non-specific criterion of right ventricular infarction due to the fact that the right ventricle is a low-pressure, thin-walled chamber with low oxygen requirements and the potential to rapidly develop collateral circulation\(^14–16\). In addition, this study was retrospective, and it is possible that biased selection of sicker patients for investigation may partly explain why patients with right ventricular infarction in this study had worse outcomes than those in other studies, as evidenced by the in-hospital mortality rule (21·6\%\(^2\) vs 15\% in the meta-analysis)\(^4\). While there were no differences in the baseline characteristics or left ventricular ejection fractions of patients with and patients without right ventricular infarction, the heart rates and blood pressures were not reported\(^2\).

Gumina et al. found that there was an incremental increase in mortality with increasing TIMI risk scores in the low range (≤ 5 points). The in-hospital mortality rates were approximately 7\% in patients with a score of 0–1, 12·5\% in those with a score of 2–3, and 26\% in those with a score of 4–5. There was no further rise in mortality in those with scores above 5. The number of patients within each score group was not reported. However, given that the overall hospital mortality rate was 21·6\%, it is likely that the majority of patients had a score of ≥ 4 — whereas in the original TIMI risk score cohort\(^1\), only 34\% of the patients had a score of ≥ 4. Thus in the Gumina study\(^2\) the TIMI risk score helped to risk-stratify the minority with low scores, but failed to further risk-stratify a large group of patients with scores of ≥ 4.
The prognostic discriminatory capacity of a risk score may be evaluated by its C-statistic (i.e. the area under the receiver operating characteristic curve). A C-index of >0.8 is generally considered useful, while a C-index of <0.7 is considered limited. In the original TIMI risk score model[12], the C-index of the score was 0·78 in the derivation set and 0·75 in the external validation set. In the Gumina study[2], C-statistics were not reported.

In addition to the possibility of a selection bias, many patients with right ventricular infarction in the Gumina study may have had varying degrees of hypotension, and arbitrary dichotomous cut-off values — such as a systolic blood pressure of <100 mmHg, which is used in the TIMI risk score model — leave little room for further risk stratification. The Global Use of Strategies to Open Occluded Coronary Arteries (GUSTO) risk model[3] uses a graded scoring system for haemodynamic parameters, and may better classify patients at varying risk such as those with and those without right ventricular infarction. However, this hypothesis will need to be tested in patients with right ventricular infarction.

When the mortality rate of the patients with right ventricular infarction in the Gumina study is stratified by the TIMI risk score and compared with the mortality rate in unselected patients with ST-elevation infarction in the InTIME-II trial[4], large differences are noted (Fig. 1). The 30-day mortality rates were 7% vs 1·3% in those with scores of 0–1, 12·5% vs 3·3% in those with scores of 2–3, and 26% vs 9·3% in those with scores of 4–5. Thus although there was a mortality gradient up to a score of 5 in the Gumina study, the mortality rates were actually 2·8–5·4 times higher than those observed in the corresponding groups with the same TIMI scores in the original InTIME-II cohort. Although many factors are likely to be involved, these data highlight the fact that a low TIMI risk score may be misleading in patients with right ventricular infarction, leading to false reassurance about their prognosis.

The long-term prognosis of patients with right ventricular infarction has not been well defined[10]. Berger et al. identified 58 patients with right ventricular dysfunction out of 1110 patients undergoing predischARGE radionuclide ventriculography in the TIMI-2 trial[17]. Right ventricular function had returned to normal after 6 weeks in over 80% of patients, and the initial right ventricular dysfunction was not associated with increased mortality at 1 year. This supports the finding by Gumina et al. that patients with right ventricular infarction did not have a worse long-term outcome provided they survived the early phase. However, other studies have shown that right ventricular dysfunction may persist[17–19], and if it does, it predicts an adverse long-term outcome[20,21]. The differences in these studies may depend upon whether the patients studied had true right ventricular infarction or ischaemia with resultant right ventricular stunning that subsequently recovered completely. The prognosis may also be different if patients receive reperfusion therapies. Prompt and complete reperfusion of the right ventricle by primary angioplasty has been shown to dramatically improve right ventricular function and hence the clinical outcome[22].

Right-sided electrocardiographic measurements must be obtained in all patients with acute inferior infarction at the time of admission in order to diagnose those with right ventricular infarction. Further clinical studies are required to define the prognostic importance of right-sided ST elevation in the reperfusion era, and to establish whether risk scores should incorporate points for right-sided electrocardiographic changes.

C.-K. WONG
H. D. WHITE
Cardiovascular Research Unit,
Green Lane Hospital,
Auckland, New Zealand

References

Catheter ablation: is it good for all postinfarction ventricular tachycardias?

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The growing number of patients surviving an episode of myocardial infarction and the prevention of sudden death in high risk patients, due to the more widespread application of these rules, however, has several important implications. The implantation of an ICD is not a curative approach; the risk of arrhythmia recurrence remains unaltered by the intervention, and frequently repeated shocks are a cause of significant discomfort, which reflects on decreased patient acceptance of the device and a lower quality of life[1].

The occurrence of arrhythmic storms, furthermore, may pose a significant threat to the patient's cardiac status and, sometimes, even survival[4]. For all the abovementioned reasons the need for a curative approach, such as that potentially offered by catheter ablation, is felt as an important goal by those electrophysiologists who are frequently involved in the management of patients with VT. As recently published in this Journal, catheter ablation performed using a single catheter approach based on conventional activation mapping and pacing techniques, frequently associated with concomitant administration of antiarrhythmic drugs can offer a reasonable rate of success[16].

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