Depression and heart failure — not yet a target for therapy?

See page 1579 for the article to which this Editorial refers.

Cardiac output is not the only thing that is depressed in heart failure. Heart failure is the most common malignant disease and one of the most malignant common diseases in Europe. As with most other malignant diseases, heart failure is also associated with a high morbidity in terms of symptoms, reduced exercise capacity and recurrent hospitalization not only for acute exacerbations of heart failure but also for infection, for arrhythmias and for thromboembolic events. Therefore it is not surprising that patients with heart failure report a very poor quality of life[1–4]. Perhaps it is surprising how cheerful most patients with heart failure appear to be considering what lies ahead of them and perhaps it is the lack of education given to them by their physicians that is responsible. As with other malignant disease, there is probably an element of denial and collusion between

References

the patient and doctor to avoid discussing their prognosis, and anxiety and depression are probably commonly concealed and/or ignored.

The paper by Majani et al.\(^7\) in this issue focuses on the mental health scores of 114 men, aged 41–60 years, with heart failure being assessed for heart transplantation in comparison with 895 healthy volunteers. Some of the results were predictable, others were not. Patients were more depressed and had more psychosomatic complaints. However, patients were not more anxious and actually had lower fear and phobia scores, lower social anxiety ratings and trends to less medical-related anxiety. It is impossible to tell from this report whether the lack of anxiety expressed by these patients reflects denial of, adaptation to or acceptance of their fate or some aspect of the referral process that selected patients with low anxiety levels. Patients perhaps also select themselves; anxious patients may have a worse outcome and do not survive. It seems strange that patients facing either death or a major life event such as heart transplantation should not exhibit anxiety. The findings of the present study contrast with some previous reports\(^3\)–\(^4\).

The paper by Majani et al.\(^7\) also highlights the relationships between disease severity, psychological well-being and perception of health (health related quality of life). They show, not unexpectedly, that all three are interrelated but correlation does not prove cause and effect. It is not surprising that patients with more severe symptoms of heart failure and with greater haemodynamic impairment had worse scores for both anxiety and depression. It is natural to assume that quality of life depends not only on physical but also psychological health as the authors suggest. However, it is also possible that mental health impacts on the progression of disease and the physical incapacity of patients, as has been shown for other cardiovascular problems.\(^8\)

The paper must be welcomed as an attempt to bring psychological issues in patients with heart failure to a wider cardiology audience, but it has many limitations. There are a number of methodological issues regarding the provenance and validation of the psychological tools used and the use and interpretation of the terms ‘subjective’ and ‘objective’ domain. Only 38% of patients completed their questionnaires. Patients were being evaluated for heart transplantation with some patients being accepted and others being refused for reasons that could engender optimism or pessimism. The population under study was highly selected by the referral process and only included men. Gender may have a powerful influence on the relationship between mental and physical health. Despite an excess of sexual dysfunction interpersonal relationships with partners were reported to be good and this may have been a powerful counterweight to the anxiety and depression. However, most patients with heart failure are women, many of whom have outlived their partner and who live alone. Anxiety and depression appears much more common in such patients.\(^5\)–\(^6\). Men may not be as effective as women in supporting a sick partner.

If mental health is impaired in heart failure is it then a target for therapy? The intuitive answer is ‘yes’, but before we invest time and resources in any new intervention in medicine it is first necessary to show that treatment is effective and then that it is cost-effective. The evidence in favour of either for depression in heart failure is lacking. It is not clear that counselling and education improves the mental state of patients with heart failure. Well-intentioned carers lacking training in counselling or psychological medicine could easily do more harm than good. A complete physical and psychological rehabilitation programme may fare better but there is no proof that this is a good use of resources. Even with expert psychological interventions it is not clear that patients would benefit. Neither is the safety or efficacy of pharmacological therapy established in this population (although 24% were taking such treatment in the present study). Given that many health care systems fail even to provide adequate echocardiographic services for the diagnosis of heart failure in the community it is not clear that we should currently be expending resources on routine mental health care for patients with heart failure. A carer who is concerned for the patient’s welfare and who has up-to-date knowledge on recent advances in heart failure is all that can currently be recommended. Although all doctors should want their patients to feel better, specific treatment, pharmaceutical or otherwise, aimed at the psychological well-being of the patient with heart failure is firmly in the domain of research or educated guessing.

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References

Different benefits, different risks, equal cost

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An 88-year-old man who is undergoing coronary angioplasty for a type A lesion of the obtuse marginal branch should be considered a less ideal candidate for IIb/IIIa antiplatelet therapy than a 50-year-old man with unstable angina and a bifurcated lesion of the left anterior descending coronary artery and the diagonal branch. A severe haemorrhagic complication would be less likely to be justified if it occurred in the first patient rather than in the second.

The CAPTURE trial[1] is the first, if not the only, abciximab study in which an attempt has been made to investigate, according to the clinical characteristics of the patients and the morphological characteristics of the lesions, the presence of a gradient in the benefits obtained following intravenous antiplatelet therapy.

In the CAPTURE trial, death or non-fatal myocardial infarction occurred at 6 months in 7.5% of the patients treated with placebo and in 9.5% of the patients treated with abciximab when the troponin T levels were negative. In contrast these events occurred in 23.9% of the patients treated with placebo and in 9.5% of the patients treated with abciximab (relative risk = 0.32, 95% confidence interval, 0.14–0.62; \( P = 0.002 \)) when the baseline troponin T levels were elevated[2]. These findings raise the issue of different benefits according to the baseline risk level, with minimal or no demonstrable benefit in the low risk group.

In the angiographic analysis of the CAPTURE trial presented in this issue, van den Brand et al. analyse the morphological characteristics of the culprit lesions before and following abciximab treatment[3]. Changes following active therapy are compared to the ones following placebo administration. More importantly, the authors look for the presence of a relationship between lesion characteristics or location and clinical benefit following active treatment.

In this study, 1233 patients with refractory unstable angina (Brawnvald class III), were randomized to standard therapy (nitrates, heparin oral antiplatelets) and placebo vs standard therapy and abciximab bolus and infusion. Prior to enrolment in this trial, patients underwent diagnostic angiography and were found to have a culprit lesion suitable for percutaneous intervention. Treatment was started following the diagnostic angiogram and the intervention was scheduled 18–24 h later. The major end-points of this study were the occurrence of death, myocardial infarction or urgent revascularization by 30 days.

The first items the authors addressed in the angiographic analysis within this trial were the differences, if any, between the lesion and the flow appearance at baseline and following completion of the active treatment. The comparison of the lesion characteristics at baseline with the ones evaluated just prior to the intervention demonstrated a decrease in the angiographic presence of thrombus in the patients treated with abciximab compared to placebo. The presence of TIMI flow 0 or 1 decreased in the abciximab group from 9.6% to 6.7% and in the placebo group from 7.7% to 6.7%. Overall, there was no difference in improvement from TIMI 0 or 1 flow or in reaching TIMI 3 flow between abciximab and placebo-treated patients.

Interestingly, when the final TIMI flow in the culprit artery after angioplasty was less than 3 the incidence of death and myocardial infarction at 30 days was 11.5% in placebo and 4.1% in abciximab patients. This finding supports the role of abciximab.