The prognosis of acute myocardial infarction (AMI) has changed radically over the last 20 years. Independent of the diagnostic criteria followed, more than 90% of patients now survive the acute phase. The main reason for this improvement is owed to the effort made in preserving the cardiac muscle via different early reperfusion strategies. Late morbidity/mortality after having been released from hospital is now also much lower, but certainly not negligible. The PRIAMHO II study showed that 9.6% of patients with AMI died in the acute phase, followed by a further 1.8% by day 28, and a further 5.1% between this point and one year.1 In this last period, the main causes of death were heart failure and sudden death. However, important advances have been made with respect to prognosis via improvements in revascularization and pharmacological therapy for preventing the deterioration of ventricular function, and the prevention of sudden death via the prophylactic implantation of automatic defibrillators. The main reason for this success, especially with respect to sudden death (but also with respect to heart failure to a certain degree), has been the concentration of efforts on those patients at greatest risk.

Since the introduction of specialized coronary units, the medical literature has shown that the development of heart failure during the acute phase of an AMI increases the risk of death in both the short and long term, and certainly suffering an AMI increases the risk of developing heart failure at a later date. This is the case even though the diagnosis of AMI has changed over time, and despite the fact that the diagnostic criteria for heart failure have varied greatly in different studies. Some important recent studies have examined the risk factors involved in the development of heart failure during the acute phase of acute coronary syndrome.2,3 However, very few have looked at the risk of developing late-onset heart failure (LOHF) in patients who have suffered an AMI not complicated by symptoms of heart failure, and therefore thought to be at lower risk of complications. A priori it might appear that the risk factors for developing LOHF ought to be very similar to those for developing heart failure during the acute phase of an AMI; any findings that depart from this might initially appear doubtful. In any event, being able to identify patients at greater risk of developing LOHF would allow us to better piece together the puzzle of how to prevent heart failure, the greatest pandemic of the 21st century.

The article by Macchia et al in the current issue of the REVISTA ESPAÑOLA DE CARDIOLOGÍA reports a post hoc analysis of the GISSI Prevenzione trial results, the aim of which was to determine the risk factors associated with the development of heart failure in a group of patients who had suffered a low risk AMI and who presented no symptoms of heart failure during the acute phase. These patients were a subgroup of those included in the GISSI Prevenzione study.4 The results suggest we ought to reflect on a number of points. As expected, the factors associated with the development of LOHF are not very different to those associated with the appearance of heart failure during the acute phase, such as age, a history of AMI or peripheral vascular disease, diabetes, high blood pressure, a high heart rate, and a reduced ejection fraction. This study is the first, however, to associate leukocytosis with LOHF. This ought not to be surprising since this condition has been associated with a poor prognosis in the acute phase of AMI, especially neutrophilia, which has been associated with larger infarctions.5 Recently it has been shown that the enzyme GRK-2, which is present in leukocytes, is involved in the disconnection of cardiac beta-adrenergic receptors, and that its expression is associated with heart failure.6 This might explain this association between leukocytosis and heart failure.

It should be emphasized that the figures referring to the risk of developing heart failure are probably
C reactive protein, a marker of inflammation, is well documented. Although the effect of intensive statin therapy in the CARE and GISSI Prevenzione studies, statins influencing the development of LOHF). At the time of release at first sight suggests that the blocking of the renin-angiotensin-aldosterone system, and treatment with statins. Such treatment may change a patient’s risk profile.

In the article by Macchia et al in this issue of the REVISTA ESPAÑOLA DE CARDIOLOGÍA, one of the findings underlined is the important influence of the ejection fraction on the development of heart failure, especially in this group of patients in whom this variable was quite well preserved; every tiny loss seems to have an impact on a patient’s future. In this population, the patients who did not develop heart failure had a mean ejection fraction of 54±9.8% compared to 49±10.1% in those that did develop this complication. This shows that every gram of myocardium preserved is important.

It is possible that inhibitors of aldosterone receptors may be able to play a prophylactic role with respect to LOHF. The factors identified in the GISSI analysis appear to be related to ventricular remodeling and late-onset neurohormonal activation. However, as shown in a recent analysis of the EPHESUS clinical trial, eplerenone appears to provide greater benefits when given early. Although the latter study was mainly directed towards patients who showed symptoms of heart failure during the acute phase of AMI, it should be remembered that 10% did not have such symptoms and were included because of a combination of diabetes and a reduced ejection fraction. As shown by the results of Macchia et al, both factors are associated with the development of clinical LOHF. Other factors multiply the risk posed.

In conclusion, many variables appear to be associated with the development of heart failure, the largest pandemic of this century. These factors are all pieces of a puzzle which on occasion seems impossible to put together. However, with respect to ischemic heart disease, none of the pieces seem to be mismatched. Although some of these risk factors cannot be modified, such as age, others can be altered by effective therapies such as early revascularization, the blocking of the renin-angiotensin-aldosterone system, and treatment with statins. Such treatment may change a patient’s risk profile.

REFERENCES


