Are the data on trends in case-fatality in patients with acute myocardial infarction observed in Scotland applicable across Europe?

Short-term fatality of patients admitted with acute coronary syndromes is the end-point followed most often in randomized pharmacological and interventional studies dealing with acute myocardial infarction or unstable angina[1–4]. Usually, follow-ups in most trials are prolonged up to 1 year. The long-term prognosis beyond 1 year is virtually unknown. Meanwhile short-term (1 month) fatality in patients with acute coronary syndromes depends mainly on acute coronary care, the long-term prognosis concerns adequate secondary prevention. Few studies follow coronary deaths over a long period (1986–1995), as had been done by Capewell et al.[5].

Kloner et al.[6] in Los Angeles County reported on a 12-year population-based analysis of more than 220,000 cases of acute coronary events. Its main focus on monthly variations in death showed that monthly case-fatality was highest in the years 1985–1988 and lowest in 1996, while consistently keeping the U-shaped distribution, with peaks in December–January and troughs in June–September.

It is well known from coronary registries that case-fatality of patients admitted to hospital do not reflect the true burden of acute coronary syndromes[7]; namely, half the cases die from sudden or medically unattended deaths. Therefore medical treatments in the acute phase, in respect of evidence-based guidelines, improve real mortality by only a few percent. Treatment of the acute phase of acute coronary syndromes has changed considerably since 1986. It has developed from monitoring and antiarrhythmic therapies through application of thrombolytics, aspirin and ACE inhibitors to revascularization procedures safely and routinely applied in the acute phase of events in most coronary units in Europe and elsewhere.

It is not surprising that the report of Capewell et al. showed that in a series of more than 100,000 patients admitted to hospital with a first myocardial infarction in Scotland between 1986–1995 that the short-term case-fatality diminished during this time by 46% in men and 27% in women. After excluding death within 30 days in men and women it was shown that deaths were reduced in successive years[5]. This may be attributed to changes in lifestyle and secondary preventive therapies. Improvement in secondary preventive care are anticipated in Scotland and other European countries[8].

The most important factor for median survival was age; median survival fell from more than 10 years in patients younger than 55 years, to 0–1 year in those older than 85 years. This study unequivocally showed that women, in spite of having only about one-third the incidence of disease generally have a case fatality one third higher and a median survival one half that of men. This may be attributed to greater co-morbidity in women, who more frequently have diabetes prior to acute coronary syndromes and more frequently develop severe heart failure subsequent to myocardial infarction.

The ESC’s European Heart Survey Program highlights this issue. The first published data related to the EUROASPIRE I Study in 1995, and EUROASPIRE II has already been completed in terms of changes of risk factors and therapies during last 5 years. The 5 year case fatalities in patients enrolled from 6 months to 18 months after the acute coronary event or revascularization procedures will be published next year. It will show if the differences in 5 year mortality depend on the differences in risk factors and adherence to secondary prevention guidelines in different European countries[9]. Without doubt, the potential to improve long-term survival still exists in Europe and recommended secondary preventive measures are still rarely exploited.

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References


[6] Klaper RA, Poole WK, Perrits RL. When throughout the year is coronary death most likely to occur? A 12-year population-based analysis of more than 220 000 cases. Circulation 1999; 100: 1630–4.


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The Q wave infarct; phenomenon or chimera?

See page 1008 for the article to which this Editorial refers

It is time for the destruction of error — Auden

The study presented in this issue by Abdulla and colleagues[1] should go far to put a final nail in the coffin of one of the most egregious misconceptions in modern cardiology. The terms ‘non-Q MI’ and ‘Q MI’ are in almost universal use and grace the pronouncements of the most prestigious committees of the American College of Cardiology, the American Heart Association and their European and Asian counterparts. Against this background, it is refreshing to read Abdulla et al.’s comment that ‘the electrocardiographic distinction between a Q wave and a non-Q wave MI is most likely meaningless, not only for prognosis but also in terms of therapeutic choices.’ Amen! Even though this statement is in fact supported by abundant other evidence, it represents such a departure from current dogma that a precise review is in order.

The earliest studies in electrocardiography made it clear that the pathological Q wave was a relatively reliable marker for myocardial infarction, but the presence or absence of a Q wave was not supposed to designate any particular clinical or pathological subset. A monumental error intruded in 1954 when Prinzmetal et al.[2] reported that Q wave infarcts were transmural and non-Q infarcts subendocardial. The entire basis for this notion was a study of 17 dogs with induced infarcts studied by means of needle electrodes. What happened next is genuinely astonishing. In 1957 the same workers repudiated their study, admitting errors of technique and further admitting that there seemed to be no reason why subendocardial infarcts could not produce Q waves[3], but the genie was out of the bottle. Nobody seemed to notice the refutation and for a whole generation, texts, lectures and learned studies continue to quote the Q non-Q distinction as revealed truth. This becomes even harder to credit when one considers that every cardiac pathologist who addressed the subject stated in the clearest terms that no such distinction existed. In fact they were at pains to point out that about 50% of subendocardial infarcts do not: the association is random[4]. As one distinguished pathologist commented, even 1 mm subendocardial infaracts can and do produce Q waves[5]. Three reviews between 1980 and 1983[6–8] summarized these facts and substantially demolished this misconception: it is remarkable that even though these three studies are universally accepted and quoted in all leading texts, echoes of the the imaginary Q/transmural correlation can be heard to this day in halls of medical learning. Myths die hard.

The notion that the presence or absence of a Q wave distinguished two categories of infarction was tenacious. Exponents retreated to the clinical arena, alleging that non-Q wave infarcts were clinically