Revascularization for everyone?

In a recent editorial, Dr Christopher P. Cannon reviewed the published studies comparing invasive with conservative treatment strategies in patients with non-ST elevation acute coronary syndrome (NSTE-ACS). He concluded that it has been very clearly shown that an early invasive approach to all patients with NSTE-ACS is beneficial.

Dr Cannon didn’t mention the Invasive versus Conservative Treatment in Unstable Coronary Syndromes (ICTUS) trial. This study was presented at a Hot Line session at the European Society of Cardiology Congress 2004 in Munich, Germany. This study set out to determine whether an early invasive strategy was superior to a more selective strategy in 1200 patients presenting with NSTE-ACS.

Patients were randomly assigned to either an early invasive treatment strategy or a selective strategy. The early invasive strategy included angiography within 24–48 h, percutaneous coronary intervention within 48 h, or coronary bypass surgery as soon as possible; the selective strategy included medical stabilization and angiography, and revascularization only with refractory angina or ischaemia on pre-discharge exercise testing.

Lead ICTUS investigator Robbert de Winter (University of Amsterdam, The Netherlands) reported that the primary composite endpoint of death, myocardial infarction, or ACS re-hospitalization at 1 year occurred similarly in both groups.

This important study implies that all patients with unstable angina or non-ST elevation myocardial infarction should undergo risk stratification. Patients at higher risk will benefit from an early invasive strategy, and patients at low risk can be equally well managed with an early invasive or early conservative strategy. The latter would include cardiac catheterization and revascularization for patients who develop recurrent ischaemia at rest, or who have evidence of provokable ischaemia on stress testing.

(Note, with regard to ICTUS, my editorial was published on 23 July 2004, one month before this trial was presented, thus I was not able to include it.)

References


Increased left ventricular mass in obese adolescents

We read with interest the paper by Friberg et al., regarding the association between left ventricular (LV) mass and obesity in adolescents. The authors reported that, compared with lean controls, the obese had higher absolute and height-indexed LV mass. This difference was statistically significant in females but not in males and there was a difference in LV mass between genders, as expected. These findings are, overall, similar to our recently published data in young (20- to 40-year-old) adults, and further our understanding of obesity-related cardiac abnormalities in a younger age group. In this sense, the data by Friberg and colleagues represent a significant contribution.

There are a few points we wish to address, which may be important for the interpretation of studies assessing cardiovascular structure and function in relation to body adiposity. Several factors affect LV mass including endocrine, renal and other chronic diseases, medications, and daily habits such as exercise, smoking, alcohol, or illicit drug use. None of these issues was directly addressed by Friberg et al. It is important to ascertain whether all of the above factors, particularly the daily habits that may pertain to the adolescent population studied by Friberg, were either equally distributed between study groups, or were controlled for in the analysis.

The authors performed sub-group analyses between obese and lean males and females with two of the four sub-groups containing <10 subjects. Such analyses should be viewed with scepticism. We believe that the most likely reason why there was no difference in LV mass among the male sub-groups was because of inadequate statistical power. The authors correctly pointed out that in order to have an 80% power to identify a difference in LV mass of 10 g/m (mass/height) between two groups (alpha = 0.05 and standard deviation of 10 g/m), one would need at least 20 subjects in each group.

The authors did not report on the LV volumes in the two study groups, although these data were readily available. We have found that young, healthy, obese males have a higher LV end-diastolic volume (absolute and height indexed), but similar LV end-systolic volume. It would be
interesting to know whether the obese adolescents also had a similar pattern of LV hypertrophy to the one we found in obese adults, characterized by primarily eccentric remodelling.

The difference in fasting insulin concentrations between obese and lean adolescents reported by the authors is striking and is indicative of insulin resistance among the obese. There was no association between LV mass and fasting hyperinsulinaemia, in agreement with similar findings in adults, in addition to data suggesting that insulin resistance is not independently associated with LV mass. As our previous findings have suggested an association between indices of cardiovascular structure and fasting serum ghrelin levels in healthy young adult males, it would also be of interest to investigate this association in adolescents. Such information would help improve our understanding of the interplay between endocrine factors and cardiovascular function in obesity.

References