

Cardiovascular risk scores: Great Tools... When Used by Good Physicians

Puntajes de riesgo cardiovascular: una gran herramienta... cuando son usados por un buen médico

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Risk scores or risk scales are equations designed to determine the likelihood of an event occurring. In cardiology, cardiovascular risk scales are intended to calculate the probability of an individual's experiencing a cardiovascular event over a period. Because of their widespread use, when we refer to risk scales, we are generally talking about scores that predict the occurrence of cardiovascular events related to atherosclerosis. But we must not forget that there are other scores dealing with other types of heart diseases. As the article in question states, (1) these scales are merely mathematical equations based on a series of variables that define risk much better than those same variables separately, as they consider the interactions that exist between their presence and intensity. The variables usually used should be accessible, pragmatic and, at the same time, valid for prediction. The accuracy of a risk equation in predicting the probability of an event occurring is usually calculated using a mathematical index called Harrell's C-index. (2)

Risk scores are very useful clinical tools, but we must be aware of their limitations before using them. Firstly, many important variables are not considered when designing a risk equation; therefore, they will have no effect in determining greater or lower risk. A clear example is seen in equations that include systolic blood pressure but not diastolic blood pressure for risk assessment. (3) What about a patient who has elevated diastolic pressure and controlled systolic pressure? Is his/her risk not increased? We believe this may be an example as easy to understand as the fact that body mass index is not yet included in many risk estimation scores. (3) Secondly, we must bear in mind that risk equations determine probability of experiencing a cardiovascular event in a population and not in a specific subject. Therefore, we can state that a certain probability will be fulfilled in a population of, for example, 1000 subjects, but we will most likely not be able to determine the exact probability of suffering

a cardiovascular event in a specific subject. Furthermore, another important aspect to highlight is that these equations are designed for patients in primary prevention. The question we should ask ourselves when thinking about this detail is the following: is a patient with subclinical atherosclerosis a primary prevention patient or a secondary prevention patient? Should we use these equations designed for patients in primary prevention, that is, without cardiovascular disease, in patients who have subclinical disease? This is another unresolved issue.

Another limitation to consider is that risk stratification is based on the numerical result of the equation. As the authors show in the article, patients with a score < 5%, between 5% and 7.4%, between 7.5% and 19.9%, and ≥ 20% were classified as low risk, "borderline" risk, moderate risk, and high risk, respectively. The limitation, on this occasion, lies in setting these thresholds, since they are completely arbitrary cut-off points, not based on mathematical-epidemiological data or clinical data. This by no means implies that risk stratification is not useful, but we should be cautious and flexible in implementing and using these thresholds.

In the article written by Professor Daniel A. Sinawski et al., (1) the authors try to evaluate whether three modulators, lipoprotein (a) levels, detection of carotid atherosclerotic plaques and coronary artery calcium score, are useful not only for optimizing cardiovascular risk stratification but also for modifying lipid-lowering treatment, specifically use of statins. Undoubtedly, these are challenging questions in our society, which condition clinical and economic considerations, and their results provide us with important conclusions for our daily practice. The study is well designed, based on data obtained from a considerable sample of patients. Importantly, one of the risk modulators, lipoprotein (a), is a causative factor of cardiovascular disease while the other modulators, detection

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of carotid atherosclerotic plaques and coronary artery calcium score, measure the consequence of the effect of risk factors. We would like to give a critical comment on two aspects: the fact that the patients came from a single center and the existing selection bias, since they were subjects who had attended a cardiovascular prevention clinic.

The paper has many positive aspects to highlight. Firstly, and unlike many other studies, more than half of the individuals analyzed are women, so the data provided are more realistic than in other studies with a very high percentage of men. Interestingly, almost one third of the population has elevated lipoprotein(a) levels. Lipoprotein(a), a risk factor involved in both coronary atherosclerosis (4-6) and in the development of aortic valve stenosis, (7) is becoming increasingly important. There is still no specific treatment for elevated lipoprotein(a); nevertheless, new drugs are being developed with great expectations. (8) Another very important fact is the high prevalence of subclinical atherosclerosis, both in carotid and coronary arteries, in a relatively young population. These data may overlap with those found in Spain in the PESA study (9) in a population with no apparent cardiovascular disease and with a slightly lower mean age. Also, the lack of agreement between the presence of carotid artery atherosclerosis and the presence of coronary artery atherosclerosis is a result that was already found in previous articles. (9) This lack of association may be explained because the different risk factors have different tropism for different arterial vascular territories. Thus, hypercholesterolemia has a special tropism for the coronary arteries while hypertension is more directed towards carotid artery disease.

The article also defines the pragmatic approach to risk assessment that has been carried out in these patients. This approach has not only improved cardiovascular risk stratification, but has also led to changing their therapeutic management, specifically by intensifying lipid-lowering treatment.

New developments in artificial intelligence may become a very useful future tool for risk stratification, (11) but nowadays a good score combined with modulators, as those shown by Professor Daniel A. Siniawski's team in his article, are the scientific basis for the management of our patients. We should never

forget that medicine is a science, but it is also an art, and that doctors experience and knowledge of their patients provide a level of excellence that is impossible to achieve by other means.

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