Pulmonary thromboembolism (PTE) is one of the cardiovascular pathologies that put the life of our patients at greater risk. It is evident from recent registries that at least in the United States, with 600,000 annual cases, PTE is more prevalent and produces a higher mortality rate than acute myocardial infarction with ST segment elevation (5% vs. 10% to 15%).

Delay in the diagnosis is certainly involved in the unfavorable outcome of many patients, and once this is made, inadequate risk stratification. The causes are evident: one is the diagnostic difficulties of this disease, frequently present in patients with other pathologies with superposition of symptoms, such as respiratory disease, heart failure and cancer. Another not lesser cause is the relative lack of knowledge we have of this disease, due to the scarce number of prospective studies performed with adequate technology, the reduced space it is dedicated in graduate and postgraduate study plans, and often in underdeveloped countries, the insufficient availability of adequate diagnostic methods.

Once the diagnosis is confirmed, risk stratification is essential to determine the appropriate therapy, avoiding both insufficient treatments as excesses that might jeopardize the patient’s life.

Several studies have shown that patients with shock, sustained hypotension or syncope have elevated mortality, which according to the assessed case series varies between 15% and 50% and increases to 65% if they have suffered circulatory arrest.

It is agreed that these high risk patients must be referred without delay to high complexity units (ICU-CCU) and are potential candidates to aggressive therapies: thrombolytics or mechanical thrombi treatment eventually associated with thrombolytics and performed in the hemodynamics laboratory.

On the other hand, hemodynamically stable patients ought to be adequately classified, firstly to decide where they should be treated and eventually if therapy should progress. Some authors, as Autjesky, postulate that low risk patients may be treated at home or with brief hospitalizations at low complexity units. This recommendation is based on results showing low mortality in these patients 1%.

Clinical protocols and several combinations of laboratory and imaging studies have been designed to identify these patients. Jiménez et al published in 2008 a study on troponin I in 318 hemodynamically stable patients and found no correlation with 30 day mortality. A year later, in a meta-analysis, the same author and collaborators found increased mortality in positive troponin patients.

In 2011, Konstantinides and his team found that ultrasensitive troponin T was able to predict a favorable outcome in a population of 526 hemodynamically stable patients. An explanation for the discrepancies among different studies with troponins can be found in the diverse laboratory methods employed in the analysis of this variable. According to these authors, ultrasensitive troponin T would be more efficient than troponin I or current troponin T to stratify these patients.

Given the high prevalence of coronary disease in elderly patients, it is possible that assessment of these biomarkers is distorted by the coexistence of both pathologies in a considerable number of patients.

From a different point of view, it is probable that some of the patients seen in the clinical practice with elevated troponins and no evident ethiology, are carriers of inadvertent evolving thromboembolic disease.

Because the greatest proportion of acute PTE mortality is attributable to right ventricular failure, it is understandable that better accordance will be obtained with BNP and NT-proBNP determinations. Stable patients with negative values have a better prognosis. The same occurs with right ventricular dysfunction assessed echocardiographically, although in this case too, the diverse methodologies used in the analysis hamper an adequate assessment of the method. It is possible that standardization of the study methods, especially with the inclusion of variables such as TAPSE and the Tei index, will allow more homogeneous results.

The recent development of clinical scores is very interesting for our daily practice. In the middle of the last decade, the score developed in Geneva proved its utility. Later, researchers of the RIETE study found that their own score, the PESI (Pulmonary Embolism Score...
Index) had greater negative predictive value than the former. Recently, the same team, restricting the number of variables, have designed a simplified version, the sPESI, (14) which includes age, cancer history, previous history of cardiopulmonary disease, heart rate (equal to or greater than 110 per minute), systolic blood pressure (lower than 100 mm Hg) and hemoglobin saturation (less than 90%). It is important to emphasize that the negative predictive value of this index encompasses overall mortality, including both patients dying due to the direct effect of PTE as those who even suffering acute PTE die from other causes.

Despite these advances in diagnosis and stratification, one out of every 10 ten patients with acute PTE admitted to the hospital, dies. The moderate risk group of patients that could benefit from a more aggressive therapy has not been accurately identified. This is the purpose of the PEITHO study directed by Stavros Konstantinides and his team of researchers. (15) The same objective is pursued in the study published by Dr. Jaimovich with the Cardiology team at the Hospital Italiano of Buenos Aires. (16) This article has special interest, as it brings the spotlight on a somewhat forgotten pathology in our setting and in addition focuses on a key aspect: risk assessment. His moderate-high risk patients are the most difficult to stratify. As stated by the authors, it is unquestionable that patients with RV dysfunction and elevated BNP are at greater risk. Regarding troponin, it is possible that a change in the laboratory test will improve the results. The number of cases is very good for a single center, with the advantage of homogeneous diagnostic and therapeutic conducts in a prospective registry. Their contribution to a better understanding of what happens in our population is very important. If they pursue this research, they might provide answers to allow better implementation of earlier and more efficient therapies. For the moment, the search continues.

REFERENCES

15. Steering Committee. Single-bolus tenecteplase plus heparin compared with heparin alone for normotensive patients with acute pulmonary embolism who have evidence of right ventricular dysfunction and myocardial injury: rationale and design of the Pulmonary Embolism Thrombolysis (PEITHO) trial. Am Heart J 2012;163:33-38.e1.