In the last 30 years, the number of cardiovascular diagnostic and therapeutic invasive procedures has progressively increased in many countries around the world. According to the American Heart Association, almost 1,03 million coronary angiographies, 955 thousand percutaneous coronary interventions (PCI) and 397 thousand heart surgeries were performed in the United States (US) alone in the year 2010 [1]. Although since the year 2006 the number of angioplasties seems to be stable in the US, in other countries such as Brazil the procedure count continues to rise [2]. This trend underscores the importance of concurrently analyzing the behavior of intervention related complications, particularly because PCI associated in hospital mortality remains at around 1.31%, notwithstanding the total complication rate which is even more significant. [1] In this context, post-procedural enzyme elevation is a frequently overlooked complication that has an important independent prognostic value.

The diagnostic criteria for post PCI myocardial infarction (MI) has changed considerably in the last two decades, becoming progressively stricter. In the year 2000, a consensus document on the redefinition of MI, developed by the European Society of Cardiology and the American College of Cardiology, proposed that any post procedural enzyme elevation was to be labeled as a MI. [3] In 2007, a new universal definition was published, classifying different MIs according to distinct clinical situations. Infarctions associated with PCI were defined as type 4a, and diagnosed solely if post procedural enzyme elevation was greater than 3 times the 99th percentile of the upper reference limit (URL) of the assay. [4]

In 2012, an update to the 2007 document was released and continues to be the mainstay by which MIs are defined. Many changes were proposed, particularly in the definition of post PCI events, and troponins officially became the preferred biomarker. Hence, type 4a MI is currently only diagnosed if the troponin level rises above 5 times the 99th percentile of the URL in the first 48 hours after the intervention, and is accompanied by either ischemic symptoms, evidence of ischemia in non-invasive tests or compatible coronary angiography findings. An isolated troponin rise above the predefined threshold without the occurrence of other diagnostic criteria is now defined as “myocardial injury”. Also, if pre-procedural levels were already elevated, the post-intervention value has to be at least 20% greater to be considered relevant.[5]

Despite these new criteria, the mechanisms involved in enzyme elevation during and after PCI justify why it performs well as a surrogate for future adverse clinical events, even as a sole criterion. According to data derived from magnetic resonance studies, the myocardial injury during the procedure can be classified as type 1, when it involves the myocardium adjacent to the coronary lesion being unobstructed or as type 2 when the distal myocardium and microcirculation are damaged, mainly by embolization of plaque debris. Type 2 injuries account for 75% of enzyme elevations in this scenario. Furthermore, it is known that clinical, angiographic and procedure related variables also influence these mechanisms, both by positive and negative associations. Advanced age, diabetes, heart failure, systemic atherosclerosis, anemia, renal insufficiency and active infection are the main patient related risk factors. Conversely, multivessel
disease and intervention, bifurcation lesions, vessel tortuosity, plaque calcification, intraluminal thrombus, vein graft angioplasty, extensive stenting, coronary dissection and no-reflow are potential angiographic and procedural determinants [4].

Establishing the precise prevalence of periprocedural MI is challenging mainly because of the different definitions that have been employed over the years. In earlier studies creatine kinase was used as the reference biomarker instead of troponin, and variations in the diagnostic cutoff point make any comparison even more complicated. The clinical scenario is another variable that has to be analyzed, since elective procedures carry a lower risk of enzyme elevation than acute coronary syndrome interventions. Even when these aspects are considered, the prevalence of post PCI MI as a clinical diagnosis has been reported to be as high as 30%, although isolated enzyme elevation could be observed in up to 73% of interventions [4,6].

For more than a decade, troponins have been established as the biomarkers of choice for diagnosing myocardial necrosis, and this is also valid for post-procedural events. Many studies have analyzed the prevalence of enzyme elevation in this context, while also determining the prognostic value of such a finding. These results are significantly represented by 3 large meta-analyses published between 2008 and 2011 (table 1), all of which primarily involved elective procedures [7-9]. Contrary to what more recent and rigorous definitions of MI attempt to convey, all 3 studies concluded that any post-procedural troponin elevation is a marker for adverse future cardiovascular events, considering up to 18 months of follow up. Additionally, an increase greater than 3 times the 99th percentile URL appeared to predict an even worse prognosis, suggesting a dose-response relationship. Recent publications have shown that high sensitivity troponins perform similarly in this context, with elevations occurring in almost 30% of patients and also representing a worse outcome [10]. Thus, although some controversy exists regarding this issue, it is becoming increasingly evident that troponins should consistently be interpreted as a marker of myocardial necrosis after PCI, and therefore predicts adverse clinical events.

As medical technology advances and becomes readily available throughout the world, it is imperative that physicians not only observe the possible benefits of any given intervention, but also what risks patients are exposed to under these circumstances. In cardiology, percutaneous interventions have experienced important breakthroughs since coronary angioplasty was first introduced almost 40 years ago. However, there still seems to be much to learn about possible complications that were previously disregarded, such as post-procedural enzyme elevation. Perhaps instead of adopting stricter definitions that would undoubtedly reduce the prevalence of this outcome solely from a criteria point of view, future studies should be dedicated to identify new risk factors and possible preventive measures. Until then, meticulous pre-procedural clinical assessment and selection combined with routine post-procedural troponin evaluation, are the most effective ways of both preventing and diagnosing this vital complication.

**Declarations of Interest**
The authors declare no conflicts of interest.

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