EDITORIAL

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Prosthetic valve endocarditis: a complex and lethal disease

Endocarditis valvular protésica: una enfermedad compleja y letal

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There is something that needs to be said from the outset, without rhetorical embellishment: if an institutional series of prosthetic valve endocarditis doesn't raise concerns, it's probably being misinterpreted. Because this condition doesn't lend itself to complacent interpretations. It is, in essence, a challenge for medicine, as it implies a failure in the treatment, the device, and, sometimes, the healthcare system as a whole.

The work presented by Cueva-Tutillo et al.[1] in this issue of *Cirugía Cardiaca en México* has a virtue that is not always recognized. It does not attempt to soften or minimize the problem, but rather presents it as it is: a serious and lethal condition. An incidence of 2.9% in a contemporary surgical cohort, with a predominance of mechanical prostheses, a virtually equal distribution between early and late endocarditis, and—above all—a mortality rate of 36.8%, remind us that we continue to operate in the realm of high lethality, not of a treatable complication.

Indeed, prosthetic valve endocarditis (PVE) constitutes the most severe form of infective endocarditis (IE), affecting between 1 and 6% of patients with prosthetic valves.[2]

The importance of the study lies not only in the numbers, but also in how it helps us understand the disease. PVE is not just an infection of the valve itself, but a disease that affects the surrounding tissue, where surgery was intended to repair it. PVE accounts for 20 to 30% of all IE cases.[3] This is why we see complications such as dehiscence, abscesses, and fistulas; these are not just complications, but rather the way the disease manifests. When the infection reaches this level, the problem ceases to be microbiological and becomes structural.

This study shows that surgery for prosthetic valve endocarditis is not an option; it is a necessity. When the infection spreads around the valve, surgery is unavoidable. It is not a matter of being "aggressive" or "conservative"; rather, it is about confronting the severity of the disease. The idea of doing something less invasive does not reflect the reality of the situation. Surgery is the response to the aggressiveness of the infection. The 2023 European Society of Cardiology guidelines for the management of endocarditis present surgery as a Recommendation Class 1, Level of Evidence C for early PVE, by means of new prosthetic replacement and debridement of the infected site.[4]

Another point that deserves attention is the microbiological profile. The predominance of staphylococci (73.6%) is neither a minor nor anecdotal finding. In line with the findings of

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Berisha et al.,[5] patients with mechanical valves are more likely to suffer from PVE caused by *S. aureus*, compared to bioprosthetic valves (36% vs. 17%, $p < 0.001$). This is, in fact, a reflection of an epidemiological transition that should no longer surprise us: prosthetic valve endocarditis is increasingly an infection associated with the healthcare environment, invasive manipulation, and the device itself.[3] *Staphylococcus* not only colonizes; it invades, adheres, and destroys. And it does so with an efficiency that turns every hour of delay into a lost opportunity.

This is where the narrative of "early surgery" takes on a deeper meaning. It is not simply about operating earlier; it is about understanding that time in prosthetic valve endocarditis is not linear. There are anatomical points of no return. The abscess that is contained today may become a fistula tomorrow. Partial dehiscence is the unstable prosthesis of the following week. The window for intervention is not measured in calendar days, but in tissue progression.

The series also identifies classic risk factors—advanced age, diabetes, history of endocarditis. Factors associated with worse outcomes include advanced age, diabetes, healthcare-associated infections, and early PVE.[6] Notwithstanding, it would be a mistake to interpret these as mere adjustment variables. Rather, they are markers of biological vulnerability. In these patients, endocarditis not only occurs more frequently but also progresses more destructively. This necessitates a more nuanced interpretation: not all patients with prosthetic valves have the same threshold for monitoring, nor should they have the same threshold for intervention.

However, the data point that might lead to an optimistic interpretation—a 63.1% survival rate—deserves to be examined critically. Is this high? It depends on the frame of reference. In absolute terms, it remains a pathology with unacceptably high mortality. It is a well-known fact that PVE has a high in-hospital mortality rate, between 20 and 40%.[6]

In addition, compared to mechanical prostheses, the risk of prosthetic valve endocarditis has typically been reported to be higher in patients with bioprostheses, both in unadjusted analysis (hazard ratio [HR], 1.51; 95%CI, 1.31-1.74) and in adjusted multivariable analysis (HR, 1.54; 95%CI, 1.29-1.83).[6] Conversely, in this series by Cueva-Tutillo et al., it was reported that the majority of prosthetic valve endocarditis (PVE) cases developed on mechanical prostheses (84.2%, n = 16), while only 15.8% (n = 3) were observed on previously placed bioprostheses.[1] This could be due to differences in the study population (e.g., age, comorbidities), surgical and local management practices, and patient selection for prosthesis type (mechanical vs. bioprosthesis). In other words, mechanical prostheses were more common in the studied population, which could explain the higher number of PVE in this group.

And that brings us to the core editorial point: the validation of an aggressive and early surgical strategy stems not from its success, but from the absence of viable alternatives.

Prosthetic valve endocarditis left untreated, in the presence of perivalvular invasion, has a predictable course. Surgery, on the other hand, introduces uncertainty… but also possibility. It is in this trade-off—certainty of deterioration versus uncertainty with the possibility of survival—that intervention is justified.[4]

This article, although descriptive and with a limited sample size, fulfills a more relevant function than simply "reporting experience": it establishes a clinical position. And it does so from the perspective of a Mexican center, with its own timelines, resources, and microbiology. That matters. Because one of the most frequent distortions in cardiovascular surgery is the uncritical adoption of results from contexts we do not share.

Finally, the study conveys a lesson that transcends its own findings: PVE is, in many cases, the culmination of a chain of events that began long before the operating room. Suboptimal metabolic control, repeated exposure to healthcare, fragmented clinical follow-up. Thinking of it solely as a surgical problem is to be late to the conversation.

But when the patient is already in the operating room, when the valve ring is destroyed and the infection has spread beyond its boundaries, surgery ceases to be just one option among several. It is the only possible course of action.

And this study makes that clear, with numbers that are not reassuring, but do offer guidance: in PVE, early surgery is not an aggressive approach; it is simply a refusal to be complacent in the face of the disease.

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