Valve Excrescences: Harmless and Common or Strokes-in-Waiting?*

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The report by Roldan et al. (1) in this issue of the Journal raises some troubling questions regarding echocardiographic detection of potential cardioembolic sources. Several of their findings are surprising and clearly at odds with previously published work in this arena, much of which unfortunately has been compromised by retrospective and variable methodology. A major limitation of most studies that attempt to investigate embolic phenomena is the uncertain degree to which each and every potential cardiac abnormality was specifically investigated. The study by Roldan et al. has several advantages over previous work in this field, in that echocardiography was performed in a specific search for cardiac valvular pathology. They also used more than the usual evaluation of valvular structure, including routine use of narrow field, high frame rate imaging at shallow depths to maximize both resolution and detection rates. Potentially this degree of enhanced imaging will result in a higher prevalence of “abnormalities” being detected than is seen with routinely performed clinical studies.

Their study provides several unexpected findings. The first and perhaps most obvious is the astounding prevalence of valvar strands noted in this study. Independent of age, or patient group, a fairly uniform prevalence of valvular excrescences of 38% to 47% was noted. In previous reports of transesophageal echocardiography in patients with presumed cardioembolic phenomena, the prevalence of valvar strands has ranged from <1% (2) to a high of 22% (3). The details of echocardiographic methodology and the intensity with which valvular structures were interrogated in these earlier studies is in large part unknown. Previous studies have all been based on the assumption that truly normal valves have homogeneously smooth edges, without mobile densities or “strands.” The study reported in this issue of the Journal appears to have taken a more unbiased and objective look at the issue of valve excrescences. As noted, Roldan et al. (1) specifically evaluated valves by means of a more detailed transesophageal echocardiographic technique than usual. Even so, the prevalence of strands fully twice that of the highest previously reported prevalence (2–5) is surprising and will certainly require confirmation.

One might initially assume that the more detailed methodology used here was responsible for the 2- to 40-fold increase in the prevalence of valvar excrescences. This assumption would be easy to accept if the excrescence size was relatively small; however, the average length of lesions reported by Roldan et al. was almost 10 mm. It is difficult to image that lesions of this size have been routinely overlooked by previous investigators.

Equally surprising was the finding that the prevalence of valve excrescences was virtually identical independent of age and was seen in normal volunteers to an equivalent degree to that seen in patients with connective tissue disease and in those with presumed cardioembolic disease. Encouragingly, on follow-up, which averaged >4 years, these lesions did not appear to be associated with subsequent embolic events. These data alone would suggest that the excrescences are an innocent bystander and that no cause and effect relation can or should be established between neurologic events and valve excrescences. If the data are taken at face value, one must also assume that there is no causal link between this type of valvular thickening and connective tissue disease. Both of these assumptions fly in the face of conventional wisdom; conventional wisdom unfortunately being only one step removed from folklore.

In two respects the study by Roldan et al. (1) is a model of how echocardiographic surveillance studies should be performed. The findings were evaluated both prospectively and in detail, and prospective follow-up was performed in the patient cohorts. Additionally, a series of these patients underwent repeat transesophageal echocardiography to assess serial changes in valve excrescences. The apparent stability in the number and size of the excrescences is somewhat surprising in view of the hypothesized mechanism of their formation. If indeed these excrescences represent minor tears of the endothelial lining at the coaptation edge, one would expect further traumatic disruption with dislodgment or, alternatively, further fibrosis and a change in appearance. In the study reported here, neither of these phenomena appeared to occur within the study time frame. The lack of an association with embolic phenomena when patients were prospectively followed up after transesophageal echocardiography is likewise encouraging in that the potential of the excrescences to dislodge and embolize distally is low.

Of the dozen or so cardiac anomalies that have been associated with cardioembolic disease, some have a plausible link to cerebrovascular or other embolic phenomena, and others are far less plausible. It is easy to understand, and logical to assume, that left atrial or left ventricular thrombus can result in embolic phenomena. Mechanistically, it is easy to understand how an entity such as an atrial septal defect or even a large patent foramen ovale, if present in conjunction with occult small venous thromboembolism, could result in paradox
embolization. Other entities, such as isolated mitral valve prolapse without vegetation, are far less easily rationalized as an independent cause of neurologic events or cardioembolic phenomena. Many investigators consider that valvular strands fall into a middle ground for which a cause and effect link between the abnormality and an event is plausible, although less likely. The study by Roldans et al. (1) would tend to push one in the direction of downplaying any cause and effect link between events and this anomaly. The study reported here is an ideal model of the manner in which cardioembolic phenomena in general should be approached. A similar approach to the “gray zone anomalies” of isolated atrial septal aneurysm, patent foramen ovale with only transient right to left shunting and isolated mitral valve prolapse is clearly needed as well. In view of the dramatically different prevalence of the abnormality and total lack of association of neurologic events, it may be premature to accept all their conclusions on face value. The present study should cause us to at least question many of the premises of previous work in this arena. Further confirmatory studies will obviously be needed; hopefully, some will include an equally rigorous approach to many of the other presumed cardioembolic entities.

References