

Arrhythmia coming from the transplanted heart: What problems does it generate?

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We have recently read with great interest the article by Myrda et al. [1]. The authors described a case of a 63-year-old patient who was diagnosed in the second year after heart transplantation with asymptomatic intermittent preexcitation syndrome and subsequently underwent radiofrequency catheter ablation of a left-sided accessory pathway (AP). We would like to emphasize a few more aspects of the issue.

Although the problem of cardiac arrhythmias due to pre-existing AP or dual atrioventricular nodal physiology in donor hearts is not new and occurs in less than 0.5% of patients [2], it still raises some controversies. Usually, the donor does not have a history of supraventricular arrhythmia or it is not known, and the first episode of arrhythmia in the recipient may be provoked by changes in autonomic tone (due to cardiac innervation and catecholamine excess) which affect the substrate [3, 4]. Symptomatic patients should be diagnosed and treated in the same manner as non-transplanted patients. In asymptomatic recipients, even with a negative result of an invasive electrophysiological study, there remains the possibility that the clinical significance of arrhythmia is underestimated, especially if the heart was harvested after a donor's death caused by accidental head injury. In those cases, the loss of consciousness due to an arrhythmia episode often cannot be excluded.

Classic indications for ablation of accessory pathways include supraventricular tachyarrhythmia or a short refractory period [5]. Moreover, in patients after heart transplantation, psychological aspects of recipients' attitudes should be taken into account. An elevated level of patient anxiety could be expected as soon as the patient is informed that "the new

heart is not completely healthy". The decision about ablation or its abandonment should be made after careful consideration of risks related to arrhythmia, intervention, and patients' preferences. At this point, it is worth noting that an invasive strategy has an advantage over pharmacotherapy in patients receiving immunosuppressive treatment. Drugs such as verapamil, diltiazem, and propafenone interact with calcineurin inhibitors, increasing the risk of complications of immunosuppressive agents.

As mentioned above, some patients remained asymptomatic and, on the other hand, the first signs or symptoms of arrhythmia coming from the transplanted heart can occur months or years after transplantation. When the episodes are short and not frequent, the time to diagnosis may be extended. Thus, we would like to ask the authors if they have any protocol in standard care settings for monitoring arrhythmias occurrence in the early and late post-transplantation period.

Based on our center's experience, we have observed that in the last few years on average one patient annually is diagnosed with an AP or dual atrioventricular nodal physiology, which was practically not observed more than 10 years ago. Do the authors have any knowledge about the prevalence of this type of arrhythmia in their center?

And last but not least, we would like to encourage the discussion about harvesting a heart from a donor with a known AP. In our opinion, it is a completely acceptable solution as long as the patients give their informed consent and monitoring is provided along with the possibility of invasive treatment. In the era of a shortage of organ donation, when more and more national programs of heart

donation after cardiac death are implemented, an easily treatable electrophysiological abnormality should not be a contraindication for harvesting a heart.

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