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EDITORIAL

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Detection and modification of atrial fibrillation drivers: The utility of late gadolinium enhancement MRI and ExTRa Mapping

Circumferential pulmonary vein isolation (PVI) has become an effective approach for paroxysmal atrial fibrillation (AF) ablation.[1] However, PVI is insufficient as a lone strategy for persistent AF, and additional substrate modification may be needed including the creation of linear lesions in the left atrium and focal ablation to eliminate atrial signals that demonstrate complex fractionated atrial electrograms (CFAEs) during AF and their combination. Therefore, some adjunctive ablation strategies beyond the PVI for the improvement in the long-term success rates have been currently proposed.

A new strategy based on low-voltage areas (LVAs) as detected by left atrial (LA) voltage mapping during sinus rhythm (SR) has recently been reported. An increased amount of fibrosis, which is the substrate for AF perpetuation, as detected by LA voltage mapping has been shown to be a predictor of AF/AT recurrence after AF ablation.[2] Actually, the extent of the LVZ was categorized on the basis of the LA fibrosis grade evaluated by late gadolinium enhancement magnetic resonance imaging (LGE-MRI).[3] LGE-MRI has been developed to visualize and quantify the extent of atrial fibrosis as well as ablation scar.

On the other hand, Ashihara et al have developed a novel online real-time phase mapping system (ExTRa Mapping™; Nihon Kohden Co.) based on the 5-second wave dynamics during AF.[4] They indicated that AF was mostly driven by spatiotemporally meandering rotors, rather than stationary stable rotors. What was totally different from the previous rotor mapping system was that the frequency of the rotational activations was quantitatively assessed according to the value of the non-passively activated ratio (%NP) in real-time. Ablation in these areas resulted in the prolongation of the cycle length of AF and AF termination. Therefore, non-passively activated areas (NPAs), as surrogates for the localized sources maintaining AF, may be potential AF ablation targets.

Various additional LVA-based substrate modification procedures after the PVI have been reported in non-paroxysmal AF patients with LVAs.[2] In many AF patients, individually located sites and/or patchy fibrotic areas targeted specifically for ablation have been found. A homogenization of the LVAs, encircling of the LVAs for isolation, and linear lesions from the isolated LVAs connecting them to anatomic structures to prevent AT have been proposed. However, incomplete linear and excessive lesion sets for substrate modification may lead to totally new atrial arrhythmias. Therefore, a detection of the critical target sites among the LVAs by LGE-MRI may be helpful for avoiding excessive lesion sets for substrate modification in nonparoxysmal AF patients after a PVI.

Nakamura T, et al indicated the effect of ablation at NPAs (the AF drivers) with LGE areas (a preexisting fibrotic substrate) in nonparoxysmal AF patients.[5] They focused on visualizing the area with a "patchy" fibrotic property harbouring an AF driver. However, the relationship of the atrial substrate between LGE areas and NPAs has not yet been evaluated fully. As the mechanism, the electrophysiological effects of slow conduction, heterogeneous refractoriness and rapid repetitive activity have been observed in LVAs. Thus, ablation of harbouring drivers in the areas with a "patchy" fibrotic property of the LGE-MRI in addition to the PVI may be more effective than a PVI alone for persistent AF. The selection of NPAs with LGE-areas as targets may correct the shortcoming of an ablation based on LVAs.

However, LGE areas do not necessarily express the fibrosis, because LGEs were found in the area with a loss of atrial myocytes and increased collagen content. Therefore, LGE-MRI has been used to visualize and quantify the tissue injury caused by radiofrequency lesions and fibrosis formation as well as preexisting atrial fibrosis. Discrimination between two kinds of fibrosis is needed. In addition, it is very difficult to obtain good images of the thin LA wall using LGE-MRI due to the problems with contrast medium. The dose of 0.1 mmol/kg used in this case report was lower than that in the previous reports (a dose of 0.2 mmol/kg), suggesting the fibrosis area could be underestimated.[3,5] An improvement in the imaging quality of the thin LA wall may be needed for the evaluation of the fibrosis area.

CONFLICTS OF INTEREST

Authors declare no conflict of interests for this article.

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