EP CASE REPORT

Cardioneuroablation for the treatment of recurrent swallow syncope

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This case report describes a 62-year-old male with arterial hypertension and type 2 diabetes mellitus. The patient has been suffering from multiple episodes of loss of consciousness associated with falls and many injuries for the recent 8 years. All syncopal spells were triggered by swallowing of fluid.[1] The physical examination was unremarkable. Cardiological and neurological workup (including head-up tit test, echocardiography, electroencephalography, and barium swallow test) did not reveal any significant abnormality. Provocation test[2] with a cold carbonated beverage (Coca-Cola) reproduced the presyncope and simultaneous electrocardiogram monitoring documented sinus arrest of 7-s duration.

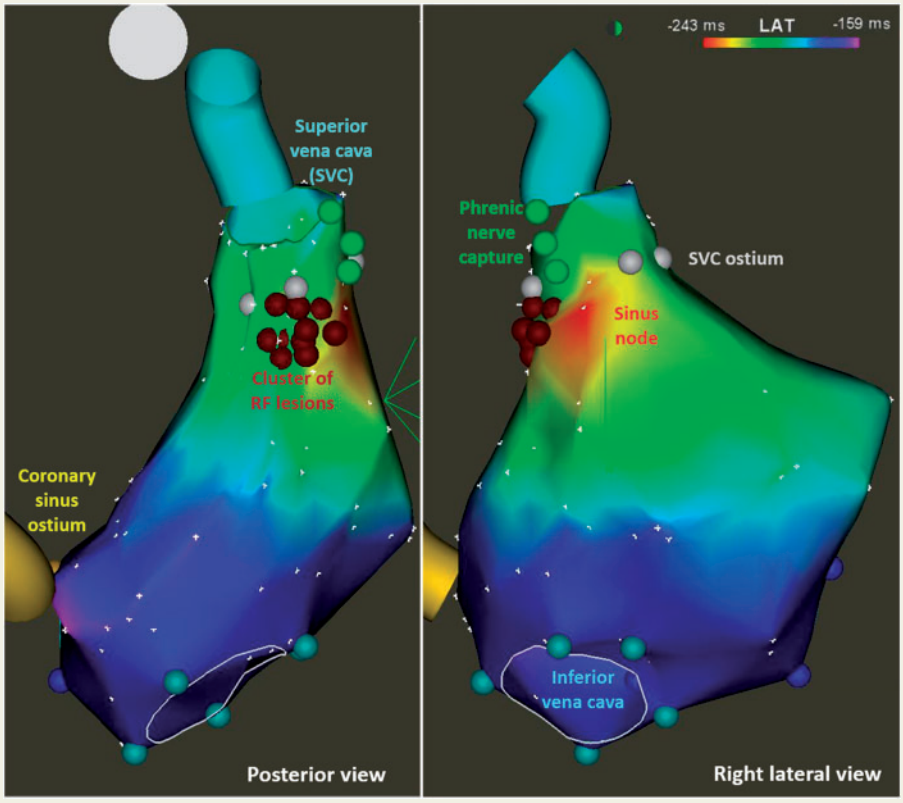

Figure 1 Map of the right atrium with ablations of superior part of the anterior right ganglionic plexus.

Patients with idiopathic deglutition (swallow) syncope can be effectively treated by dual-chamber pacemaker implant, preferentially with rate-drop-response algorithm or closed-loop stimulation function. Our patient consented for an alternative treatment method—radiofrequency catheter ablation of ganglionic plexi (cardioneuroablation)[3]—aimed at modification of parasympathetic input to the sinus node.

Right atrial anatomy was reconstructed using an electroanatomic mapping system (CARTO-3, Biosense-Webster) with tagging of sites with phrenic nerve capture and sinus nodal region. Radiofrequency energy (Navistar Thermocool, 30 W/30 s, irrigation of 20 mL/min) was delivered at the posteroseptal portion of the junction between the right atrium and superior vena cava, i.e. at the superior part of the anterior right ganglionic plexus which is empirically the most effective site for sinus nodal denervation (*Figure 1*). A cluster of 10 lesions (total radiofrequency time of 328 s) resulted in modest but persistent sinus rate acceleration (from 60/min to 72/min). Intraprocedural provocation test by swallowing of cold water was negative and administration of atropine (2 mg intravenously) had virtually no effect on heart rate. The total procedural time was 100 min with a radiation dose of 27 $\mu Gy.m^2$. During the 3-year follow-up, the patient did not experience the recurrence of the syncope, did not manifest other arrhythmias on Holter and did not report any adverse effects attributable to cardioneuroablation.

To the best of our knowledge, this is the first report on swallowing syncope successfully treated by cardioneuroablation. Our patient was not a typical representative of subjects who are more frequently candidates of cardioneuroablation. These are usually much younger and present with recurrent 'common' vasovagal syncope due to enhanced cardiac vagal modulation. On the other hand, syncopes with clear trigger (like in our case or in case of carotid sinus hypersensitivity) have more likely prevailing cardioinhibitory and less pronounced vasodepressor component. Such characteristics favour cardioneuroablation as a treatment method of choice.

Very limited right-sided ablation (avoiding transseptal puncture) targeting sinus node only may be clinically insufficient especially in patients in whom (i) complete sinus nodal denervation can only be completed by contralateral ablation in the anterior antrum of right superior pulmonary vein or (ii) predominant mechanism of the reflex syncope is atrioventricular block (i.e. those requiring ablation of inferior ganglionic plexi).

In conclusion, this case corroborates the clinical utility of cardioneuroablation as an effective and safe treatment alternative to pacemaker implantation in selected patients with reflex syncope. Further studies are warranted to elicit the long-term benefits and risks of cardioneuroablation.

Conflict of interest: none declared.

References

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