Late Persistent Hiccups After Pulmonary Vein Isolation: A Case Report

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Abstract

Introduction: To the best of our knowledge, the association of hiccups and ablation for atrial fibrillation as a late manifested complication has not been reported.

Case Presentation: The case was a 48-year-old patient presented with refractory hiccups after ten days of successful bilateral pulmonary vein isolation. All investigations did not find any specific cause for hiccups other than late complication of ablation. Hiccups were abolished with a single dose of lidocaine and were suppressed with mexiletine.

Conclusions: Hiccup is a well-defined manifestation of phrenic nerve stimulation, which in the case of ablation is a manifestation of upcoming injury and prompt for withholding the RF application in the focus. The represented case was unique, as late appearance of hiccups after atrial fibrillation (AF) ablation has not been reported before. The proposed mechanism is mentioned in the discussion. Meanwhile, the treatment of this patient with lidocaine and successful suppression of hiccups with subsequent therapy with mexiletine deserves a discussion and future investigations.

Keywords: AF Ablation, Phrenic Nerve Injury (PNI), Hiccup, PV Isolation, Complication

1. Introduction

Phrenic nerve injury (PNI) is a well-defined complication of catheter ablation for atrial fibrillation (AF), which is always of concern particularly during the isolation of right pulmonary veins (PVs). Hiccups, along other alarming signs of upcoming PNI, should prompt the operator to hold off ablation until finding a safe location. Nevertheless, hiccup, as a late consequence of AF ablation, which was the case in our patient, has not been reported previously.

2. Case Presentation

A 48-year-old man with a history of severely symptomatic paroxysmal AF and one episode of 1:1 conducted atrial flutter with flecainide was admitted for AF ablation. Atrial fibrillation was terminated with isolation of left PVs with radiofrequency ablation (RFA) and further ablation was done to isolate the right-sided PVs, which is routinely done to ensure future freedom from AF. The procedure was performed under deep sedation. No hiccup was observed during RF applications and diaphragmatic movements were frequently checked and were normal. The patient was discharged the next day with no complication in sinus rhythm and no anti-arrrhythmic drug (AAD) was prescribed.

Ten days later, the patient encountered devastating non-stop hiccups persisting even during deep sleep. The patient lost five kilograms of his weight in six days, and the hiccups were refractory to usual medications including chlorpromazine, gabapentin, baclofen and haloperidol. No corticosteroid therapy was given. The patient did not complain of dyspnea; meanwhile, chest radiograph revealed the level of diaphragms to be normal. Upper GI endoscopy also revealed no lesion or abnormality. Eventually, 60 milligrams of bolus of Lidocaine was injected intravenously, which surprisingly resulted in termination of hiccups in about five minutes. Hypothesizing the Na channel blockage to be the suppressor of the hyperactive phrenic nerve, treatment with a structurally similar drug, mexiletine, was initiated with 300 milligram twice daily for one week, which suppressed the hiccups effectively. The drug was discontinued thereafter and the patient has not experienced any hiccups during the last six months. One episode of AF occurred at the same time of hiccups and lasted for about four hours, but there has been no other episode since then despite no AAD’s being prescribed.
3. Discussion

The usual consequence of PNI in the context of AF ablation is diaphragmatic paralysis, which can lead to severe transient and sometimes, protracted respiratory problems (1). Hiccup has been appreciated for its alarming role in case of injury to the phrenic nerve. However, it has not been recognized as a late manifestation of PNI and in the absence of any other signs and symptoms. In this case no drug could be responsible for this type of pathologic hiccup, thus finding a cause for it was crucial. Although the central component of the hiccup reflex arch, located in the brainstem, is a potential candidate if taking microemboli into consideration (2), a more plausible hypothesis would be an extension of inflammatory reaction in nearby lesions and progressively invading the phrenic nerve. The inflammation around the nerve can theoretically stimulate the nerve to become pulsatile and lead to hiccups similar to stimulation by heat.

The other finding in this case was the efficacy of lidocaine in treatment of this type of hiccup, which has been shown in some previous reports (3-5). However, we also found mexiletine to be effective as well. This was in contrast to a report by Dunst et al. (3), which stated the efficacy of lidocaine but relapse of hiccup after changing to mexiletine.

References