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Case Report

Reversible bradycardia secondary to myxedema coma: case-report

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Myxedema coma occurs mostly in patients with long-standing untreated or undertreated hypothyroidism. Bradycardia is a well-known cardiac manifestation for myxedema coma; however, not all bradycardia with hypothyroidism are sinus bradycardia. Sick sinus syndrome is a group of arrhythmias caused by the malfunction of the natural pacemaker of the heart. Tachy-Brady syndrome is considered to be a type of sick sinus syndrome, where the heart alternates between tachycardia and bradycardia, and it is usually treated with pacemaker implantation along with rate slowing medical therapy. Here we report a case of an 83-year-old female who presented with myxedema coma and atrial fibrillation with tachycardia and intermittent slow ventricular response. We attempt to review the relationship between these two diseases and conclude that appropriate diagnosis of myxedema coma, may be beneficial in reducing the need for pacemaker implantation.

Keywords

Atrial fibrillation with slow ventricular rate; hypothyroidism; myxedema; case report

1. Introduction

Bradyarrhythmia is defined as heart rate of less than 60 beats per minute. It can be caused by intrinsic or extrinsic abnormalities. Intrinsic causes, such as age-related degeneration or infarction of sinus nodal tissues, whereas extrinsic causes include negative chronotropic pharmacologic agents, electrolyte imbalances, hypothermia, as well as hypothyroidism (Mangrum and DiMarco, 2000). Approximately one million pacemaker implantation occur worldwide secondary to bradyarrhythmia (Mond and Proclemer, 2011; Raatikainen et al., 2015). Myxedema coma is typically a sequela of chronic thyroid hormone deficiency and is characterized by the deterioration of mental status, hypothermia, hypotension, hyponatremia, and hypoventilation. It is considered to be an endocrine emergency. In the past, the overall mortality rate for myxedema coma was 60%-70%. Early disease recognition and advancements in the care provided to these patients have reduced the mortality rate to 20%-50% (Ueda et al., 2019). A precipitating event on a background history of long-standing, untreated, or inadequately treated hypothyroidism may result in myxedema coma. Infections, drugs such as: narcotics, anesthetics, and sedatives, in addition to lung diseases, congestive heart failure, and gastrointestinal bleeding maybe some of the precipitating events (Dhakal et al., 2015). In this review, we aim to emphasize the importance of recognition of the underlying cause of bradyarrhythmia before proceeding with pacemaker implantation in the setting of myxedema coma.

2. Case presentation

An 83-year-old female was brought to the emergency department after she was noted to have a low oxygen saturation at home. She was recently admitted due to acute kidney injury, and her diuresis was held on discharge. On further questioning, she reported having a non-productive cough and mild shortness of breath for the last few days. She reported no new sick contact or travel history. She has a past medical history of hypertension treated with amlodipine, permanent atrial fibrillation on rivaroxaban for CHADsVASc score of 5, hypothyroidism treated with levothyroxine, lower limb edema on furosemide as needed and hyperlipidemia treated with atorvastatin. It was noted that she had inadequate medication compliance for the last five months. Social history was negative for smoking, alcohol use, or illicit drug use. Family history was significant for diabetes mellitus. She had no known allergies. A review of systems was positive for cold intolerance, puffiness around the eyes, constipation, and sluggish responses. On arrival to the emergency department, she was noted to be hypoxic, requiring 2 liters of oxygen via nasal cannula to maintain oxygen saturation above 90%. Blood pressure and heart rates were 145/61 mmHg and 62 beats per minute, respectively. She was mildly confused to time and place, had an irregular pulse, jugular venous distention, positive hepatojugular reflux, bilateral inspiratory wheezes on lung auscultation, bilateral pitting edema up to the mid-shin, and slow deep tendon reflexes were noted. The rest of the examination was unremarkable. Initial laboratory workup revealed elevated brain natriuretic peptide (Table 1). Electrocardiogram showed atrial fibrillation with a heart rate of 60 beats per minute (Fig. 1). Chest radiograph (CXR) showed mild pulmonary edema (Fig. 2). She was admitted as acute hypoxic res-

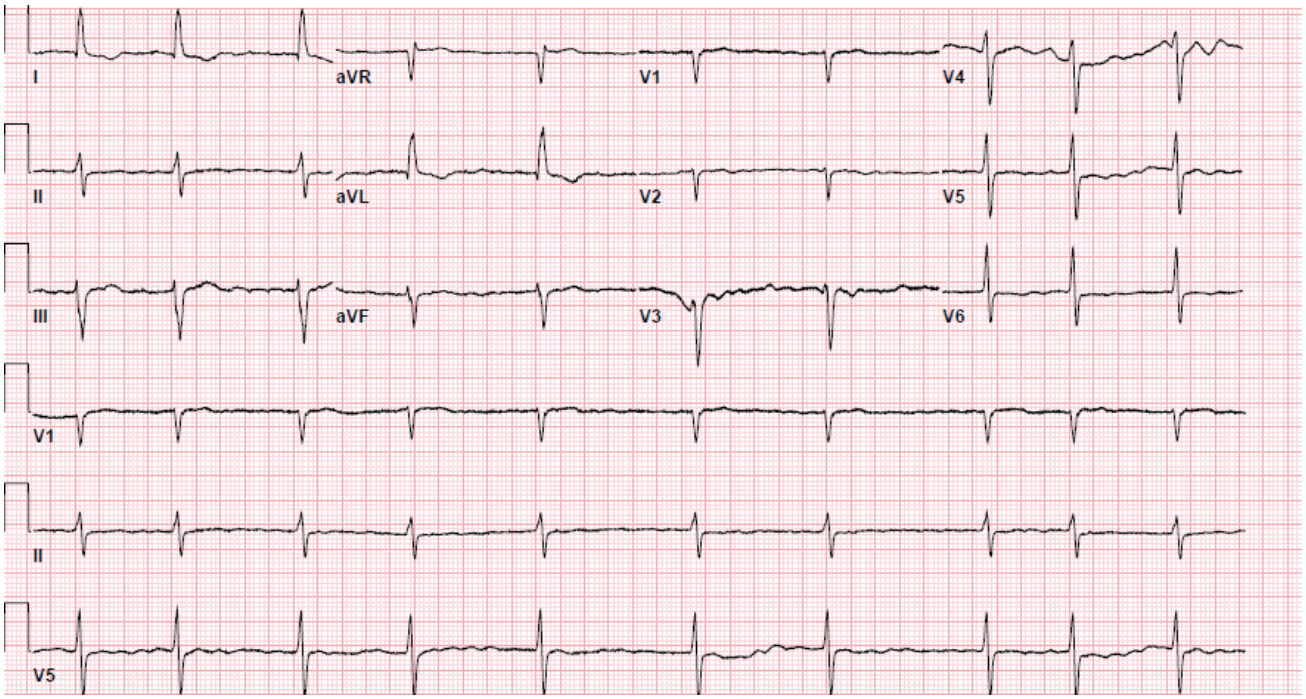

Figure 1. Electrocardiogram was done on admission showing atrial fibrillation, as evidenced by the absence of P-waves, narrow QRS complex, and irregular rhythm, with a pulse rate of 60 beats per minute.

piratory failure secondary to pulmonary edema due to heart failure exacerbation and was subsequently started on intravenous diuretics. Overnight, her heart rate dropped to 13 beats per minute (Fig. 3). Electrocardiogram before admission showed atrial fibrillation with a mildly rapid ventricular rate (Fig. 4), and occasional tachycardia was also noted on telemetry. Cardiac monitor overnight showed evidence of recurrent bradycardia (Fig. 5A,B). Cardiology was consulted for pacemaker implantation in the setting of bradycardia. Echocardiography showed an ejection fraction of 55%, eccentric left ventricular hypertrophy, mild reduced right ventricular systolic function, and right ventricular systolic pressure of 52.9 mmHg. There was trace anterior pericardial effusion noted on the echocardiogram. Thyroid-stimulating hormone was ordered, and it came back significantly elevated at 96.7 UIU/L (Table 1). On further assessment by cardiology service, it was determined that she had congestive heart failure associated with bradycardia, which was attributed to hypothyroidism, and hormone replacement was recommended with no recommendation for pacemaker insertion at that time. On follow-up, the patient was subsequently started on levothyroxine 200 mcg for a minimum of 2 weeks, with a plan to recheck her thyroid-stimulating hormone and free thyroxine level in 2-3 weeks and titrate down to 150 mcg accordingly.

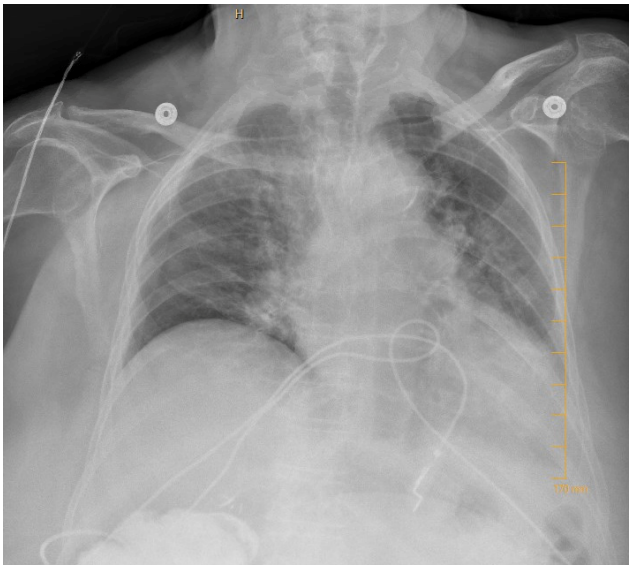

Figure 2. Portable Anteroposterior chest radiograph was done on admission showing interstitial edema as evidenced by increased lung interstitial markings bilaterally, and a small left-sided pleural effusion, suggestive of pulmonary congestion.

A few days later, her symptoms resolved, with the resolution of bradycardia documented by continuous cardiac monitoring. Interestingly, no significant tachycardia was seen following initiation of thyroxine with maximum heart rates around 101-105 bpm with highest rate on day 2 of levothyroxine of 121 bpm. A week later, her thyroid-stimulating hormone level was down to 15.3 UIU/L, and repeat CXR showed resolved pulmonary edema (Fig. 6). She was discharged in a stable condition on a tapering dose of levothyroxine, with close follow-up with her primary care physician and endocrinology.

3. Discussion

Untreated hypothyroidism is known to affect almost all organs, including the cardiovascular system. It is associated with a decrease in cardiac output, stroke volume due to decreased myocardial contractility, and an increase in systemic vascular resistance.

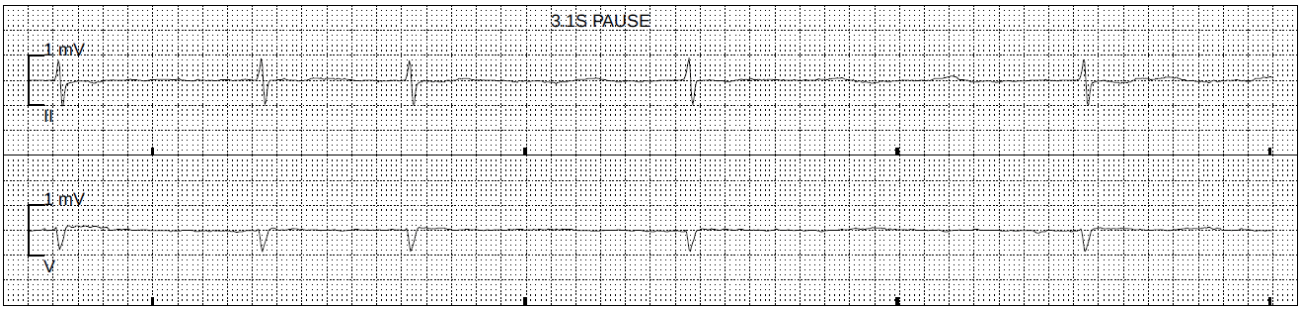

Figure 3. Cardiac rhythm strip recorded during the patient's stay in the intensive care unit, showing atrial fibrillation with a slow ventricular response and a pause of 3.1 seconds.

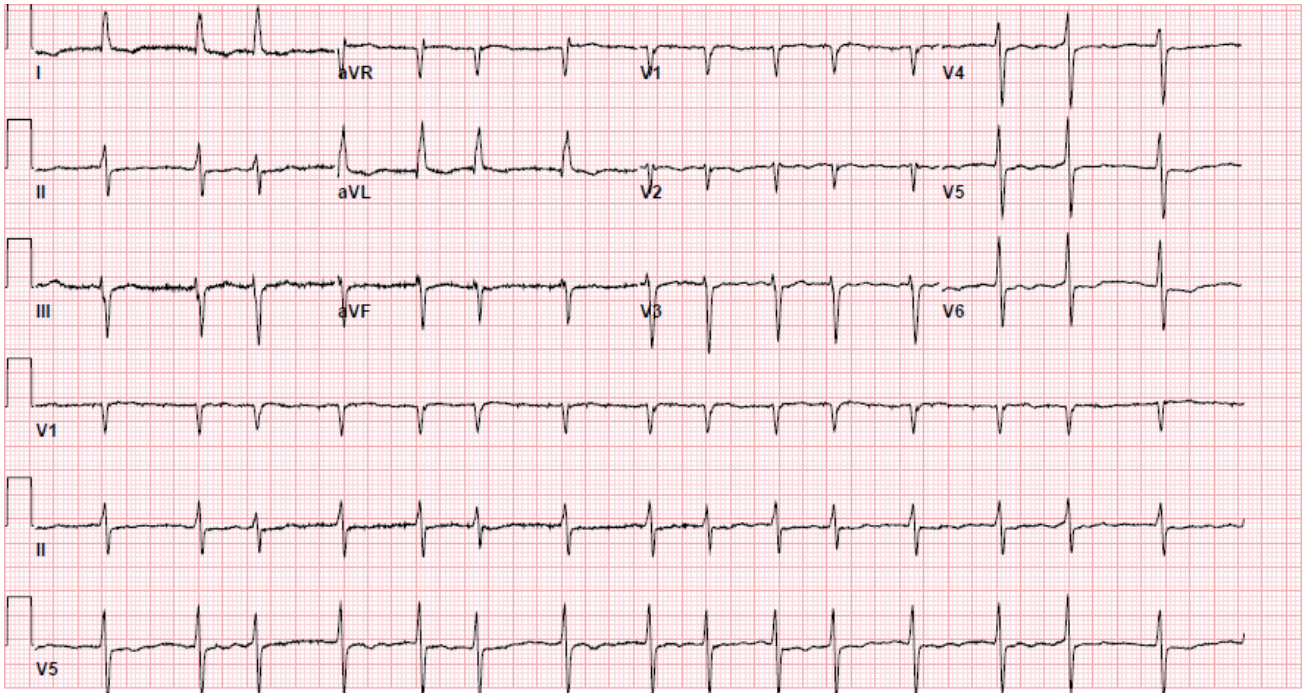

Figure 4. Electrocardiogram from a previous admission showing atrial fibrillation, as evidenced by the absence of P waves, narrow QRS complex, and irregular rhythm, with a pulse rate of 90 beats per minute.

It can cause cardiac arrhythmias, the most commonly seen conduction abnormalities being sinus bradyarrhythmia, heart block, ventricular tachycardia, and torsade de points. In addition, diastolic hypertension has been observed in the early stages due to peripheral vasoconstriction and central shunting of blood. Pericardial effusions may also be present in such cases due to increased vascular permeability (Salhan et al., 2017). Supraventricular arrhythmias, including atrial fibrillation, can be seen in around 50% of patients with sick sinus syndrome or sinus node dysfunction. In our patient atrial fibrillation with slow ventricular response was present. Correction of any possible underlying reversible cause for bradyarrhythmia should be sought first before consideration of pacemaker implantation.

To date, only a few studies have described patients with myxedema coma presenting with bradyarrhythmia's, who were successfully treated with thyroid hormone replacement only, without the need for permanent pacemaker implantation. Harada et al. reported a case of a myxedema coma in an elderly female patient who presented with sick sinus syndrome and type two respiratory failure, which was successfully managed with thyroid hormone replacement (Harada et al., 2019). Also, Waseem et al. (2018) reported a case of an elderly male with a history of undertreated hypothyroidism who presented with atrial fibrillation with a slow ventricular response as well as a high degree atrioventricular block. The atrioventricular block resolved upon increasing the dose of levothyroxine, thus avoiding the need for permanent pacemaker implantation (Lee et al., 1986). Also, Schoenmakers et al. described a case of an elderly female who presented with a complete heart block and was found to be in myxedema coma. She was treated with low dose levothyroxine and required temporary pacing. After several days atrioventricular conduction was restored, and she did not require permanent pacemaker implantation (Schoenmakers et al., 2008). Furthermore, in a study of atrioventricular block in patients with thyroid dysfunction, conducted by Ozcan et al., 24% of patients with hypothyroidism and atrioventricular block had complete resolution of the atrioventricular block

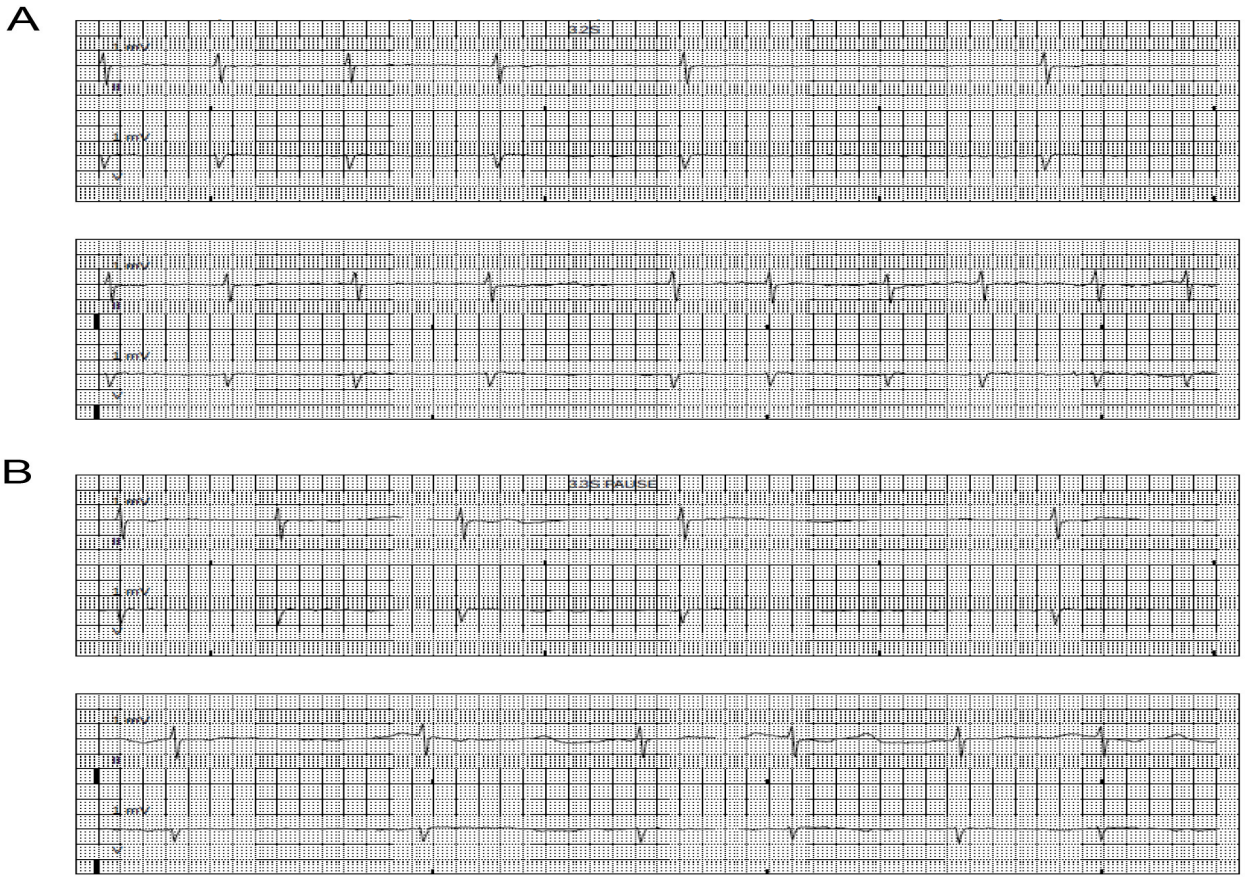

Figure 5. Two cardiac rhythm strips recorded during the patient's stay in the intensive care unit, done 2-hours apart, showing atrial fibrillation with a slow ventricular response. Figure -5A shows a pause of around 3.2 sec. Figure-5B shows a pause of around 3.2 sec.

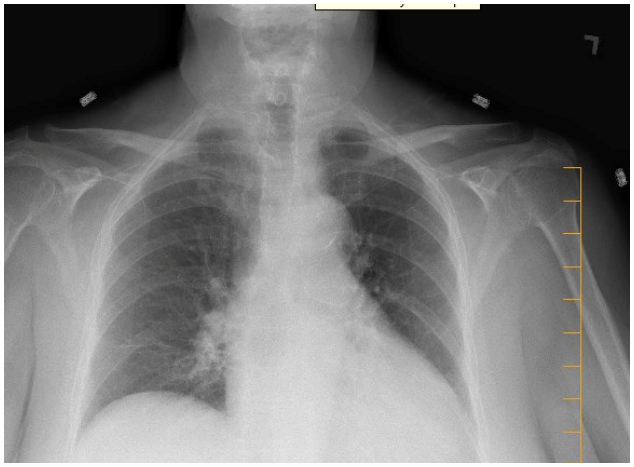

Figure 6. Chest radiograph was done one-week after initiation of thyroid replacement therapy and diuretics, showing resolution of the previously noted pulmonary congestion and the left-sided pleural effusion.

after treatment with thyroid hormone replacement (Ozcan et al., 2012). Older studies have also described an atrioventricular block in patients with myxedema, that resolved with thyroid hormone supplementation, without the need for cardiac pacing (Schantz and Dubbs, 1951; Singh et al., 1973).

4. Conclusion

Our case demonstrates the importance of recognizing severe hypothyroidism, and particularly myxedema coma, as a cause of bradyarrhythmia, especially in elderly patients presenting with nonspecific symptoms such as fatigue, presyncope, syncope or palpitations. Mortality rates associated with myxedema coma with cardiac complications is high. So, appropriate treatment of these patients with thyroid hormone replacement can spare them the need for permanent pacemaker placement, as well as reduce procedure-related risks and complications.

Authors' contributions

- Omar Kousa, Mohamed Mansour, Dana Awad, Amr Essa and Abdallah Qasim had substantial contributions to the conception or design of the work, formulated the tables and figures, and drafting the work.
- Arindam Sharma and Mark Holmberg critically revised, edited the manuscript substantially and approved the final version of the current version.
- All authors agree and are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

Table 1. Laboratory work up on presentation.

Test	Result	Reference Range
White Blood Cell	6.1 k/mL	4–12 k/mL
Hemoglobin	11 gm/dL	12–16 gm/dL
Hematocrit	36.20%	36–48 %
Mean Corpuscular Volume	99 fL	80–100 fL
Platelet	288 k/mL	140–440 k/mL
Glucose	90 gm/dL	74–106 gm/dL
Sodium	140 mmol/L	137–145 mmol/L
Potassium	3.1 mmol/L	3.5–5.1 mmol/L
Chloride	105 mmol/L	98–107 mmol/L
Carbon Dioxide	30 mmol/L	22–30 mmol/L
Urea	30 mg/dL	9–20 mg/dL
Creatinine	1.36 gm/dL	0.7–1.2 gm/dL
Total Protein	7 g/dL	6.3–8.2 g/dL
Albumin	4.1 g/dL	3.5–5.0 g/dL
Calcium	9.7 mg/dL	8.4–10.2 mg/dL
Total bilirubin	0.9 mg/dL	0.2–1.3 mg/dL
Aspartate Aminotransferase	70 IU/L	15–46 IU/L
Alanine Transaminase	78 IU/L	4–50 IU/L
Alkaline Phosphates	92 IU/L	38–126 IU/L
Creatine Kinase	43 IU/L	55–170 IU/L
Brain Natriuretic Peptide	4856 pg/ml	$<$ 449 pg/ml
Thyroid-stimulating hormone	96.7 UIU/L	0.4–3.8 UIU/L
Troponin	$<$ 0.04	$<$ 0.04
Influenza antigen A & B	negative	negative
Early morning cortisol	14.2 ug/dl	5.3–22.4 ug/dl
Thyroid peroxidase antibodies	47 u/ml	$<$ 59u/ml

Ethics approval and consent to participate

Our institution does not require ethical approval for reporting individual cases or case series. The consent was not obtained from the patient(s) due to unavailability. Any potential patient's identifiers have been removed from the description/images and this paper has been anonymized not to cause any harm to the patient.

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Conflict of interest

The author declares no conflicts of interests.

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