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Case Report

Severe calcification in chronic constrictive pericarditis of tuberculous-related a case report and literature review ☆

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ABSTRACT

Chronic constrictive pericarditis is a pericardial affection that causes a severe impairment of myocardial compliance. Among its many etiologies, tuberculosis is the most common cause, mainly in developing countries. Multimodal imaging methods are essential tools for guiding diagnosis. We present the case of a 64-year-old man with no past medical history who presented with dyspnea stage II of NYHA and right heart failure. At admission, he was stable, with normal blood pressure and a normal heart rate. His ECG showed a low voltage of QRS complexes. Transthoracic echocardiography revealed significant pericardial thickening enveloping the ventricles, with significant respiratory flow variation. A thoracic CT scan and cardiac MRI confirmed the presence of pericardial thickening and calcifications. The patient underwent beat-heart pericardial decortication. The anatomopathological examination of the surgical piece revealed Mycobacterium tuberculosis. The postoperative check-up after 6 months showed good clinical and echocardiographic evolution.

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Introduction

Chronic constrictive pericarditis is a relatively rare (0.5%-1% of heart diseases) but serious condition due to the functional impairment it causes [1]. It results from the transformation of the pericardium into a rigid, inextensible, fibrous, and calcified envelope, reducing myocardial compliance and leading to a disorder of cardiac filling pressures.

In the Western world, the main etiologies are viral infections, cardiac surgery, and thoracic radiotherapy [2,3]. While in developing countries, tuberculosis remains the main etiology [4]. Constrictive pericarditis is often underdiagnosed due to its nonspecific and sometimes insidious clinical presentation and progressive evolution. The challenge arises in differentiating it from restrictive cardiomyopathies, which are the primary differential diagnosis, along with other entities responsible for right-sided heart failure [1,5]. Transthoracic echocardiography is the initial examination to be performed, while MRI helps to clarify the diagnosis, and right heart catheterization confirms it [6]. Nevertheless, surgery is associated with high early morbidity and mortality and poor long-term survival [7].

Case presentation

A 64-year-old patient, originally from Northern Morocco, presented to the emergency department of our facility with dyspnea, moderate ascites, and bilateral lower extremity edema. The patient had poorly controlled diabetes; there is no history of trauma, mediastinal radiation therapy, thoracic surgery, known neoplasia, or Tuberculosis (TB).

His symptoms appeared 8 months early, with the onset of NYHA stage II dyspnea and pericarditis-like chest pain occurring in the context of night fever, sweating, and asthenia, with a marked weight loss estimated at 10% of the initial weight.

On admission, he was stable with a heart rate at 82 beats/min, a blood pressure at 117/63 mmHg, a respiratory rate at 17 cycle/min, and his oxygen saturation was 99% on room air. Physical examination revealed signs of right heart failure, including jugular venous distension, hepatomegaly, bilateral lower extremity edema, and moderate ascites. The ECG (Fig. 1) shows a low voltage in the peripheral leads and negative T waves from V1 to V6. The laboratory workup reveals biological cholestasis alkaline phosphatase (PAL) at 314 U/L (normal range : 40-129 U/L), gamma-glutamyl transferase (GGT) at 161 U/L (<45 U/L), and total bilirubin at 36 mmol/l (<20 mmol/l). The acid-fast bacilli (AFB) test in sputum was negative, Quantiferon was positive (TB1-Nil and TB2-Nil >0.35 IU/L), and CA-125 was very elevated at 1325 U/ml (<37 U/ml), indicating inflammation of serous membranes (pericardium and peritoneum). The full blood count, renal function, and liver function were normal.

The Transthoracic echocardiography (TTE) (Fig. 2), revealed significant pericardial thickening enveloping both ventricles, associated with septal bounce, moderate tricuspid regurgitation, and significant respiratory flow variation. The systolic function of both ventricles was well preserved. The thoracic CT scan (Fig. 3), showed pericardial thickening of 17 mm. We performed cardiac MRI (Fig. 4) in our patient, which confirmed the findings of TTE and CT regarding morphological abnormalities of the pericardium, and also showed the presence of pericardial calcifications. Considering all these clinical, echographic, CT scan and MRI criteria, the diagnosis of chronic constrictive pericarditis was established.

We managed the patient's treatment with furosemide 250 mg/24 h and then 500 mg/24 h, but there was no clinical improvement. However, despite medical treatment, systemic congestive symptoms and dyspnea still persisted, and surgical pericardectomy was indicated for diagnostic and therapeutic considerations.

The patient underwent beat-heart pericardial decortication, and macroscopic examination of the pericardium revealed significant pericardial calcifications (Fig. 5). The anatomopathological examination of the surgical piece revealed

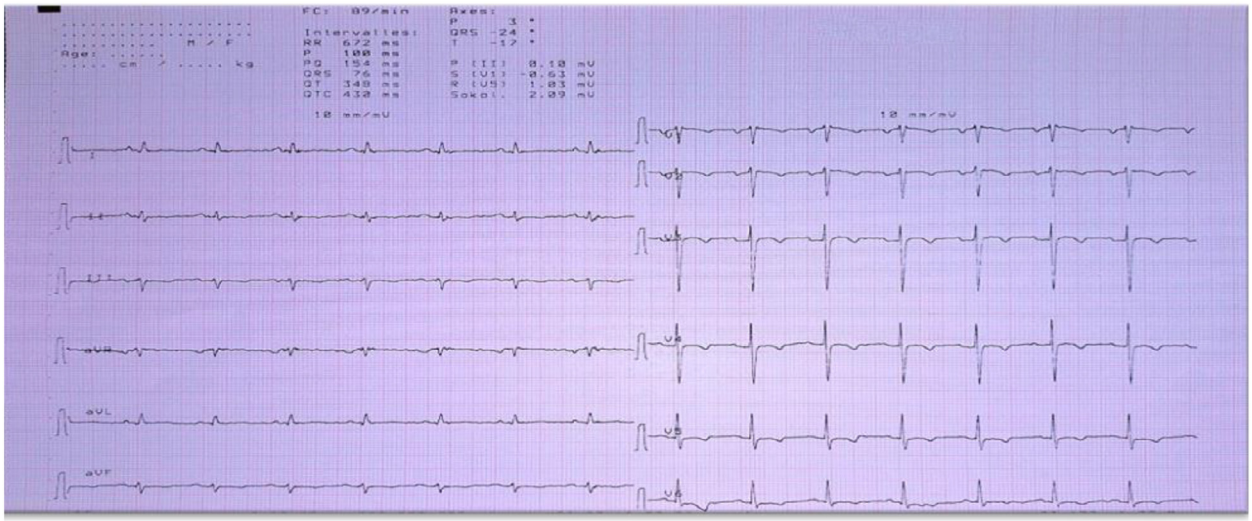

Fig. 1 – ECG showing low voltage of QRS in peripheral leads and negative T waves from V1 to V6.

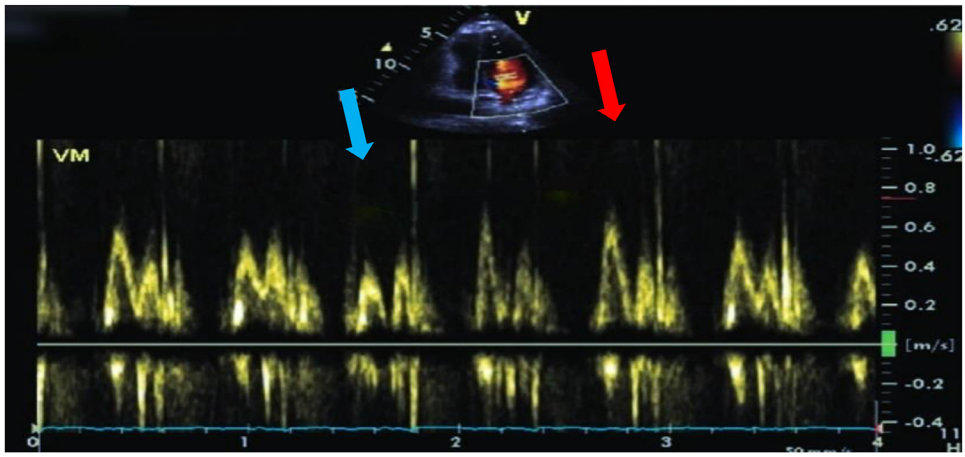

Fig. 2 – Doppler echocardiography showing mitral inflow velocity with respiratory variation; Inspiration (blue arrow), Expiration (red arrow).

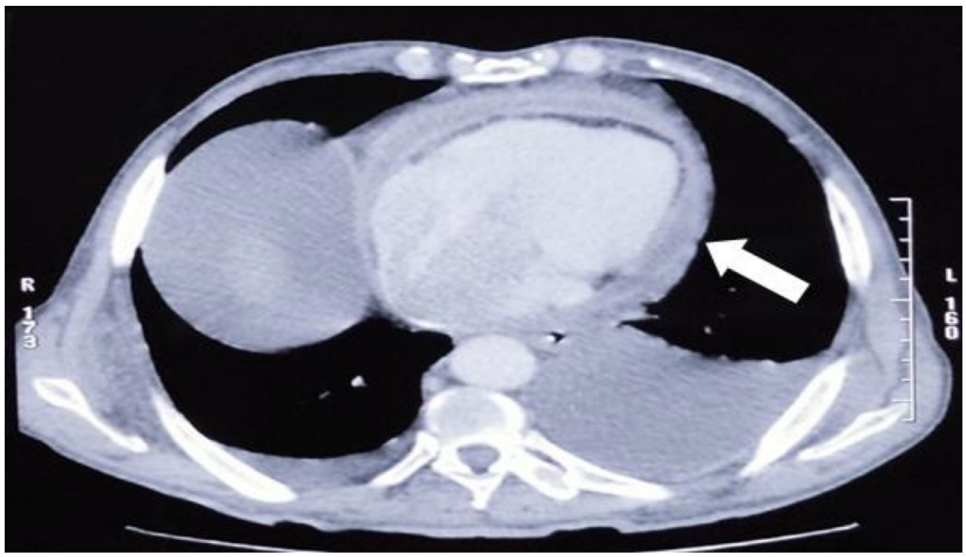

Fig. 3 – Computed tomography demonstrates pericardial thickening (white arrow).

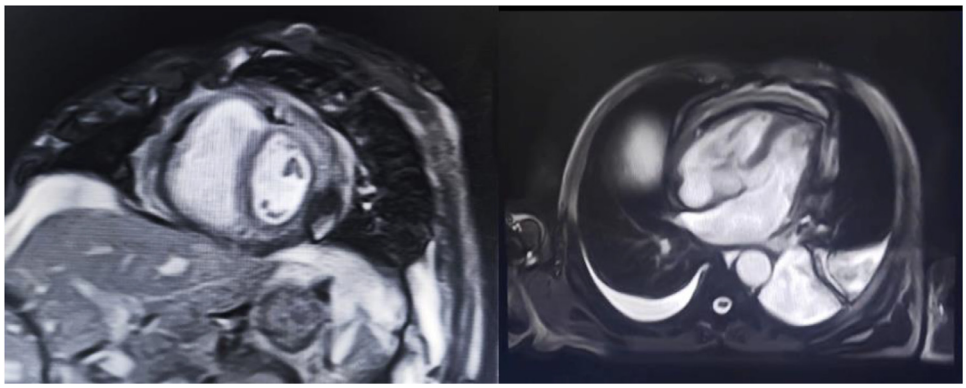

Fig. 4 – Cardiac MRI shows pericardial thickening and calcifications mostly adjacent to the right ventricle.

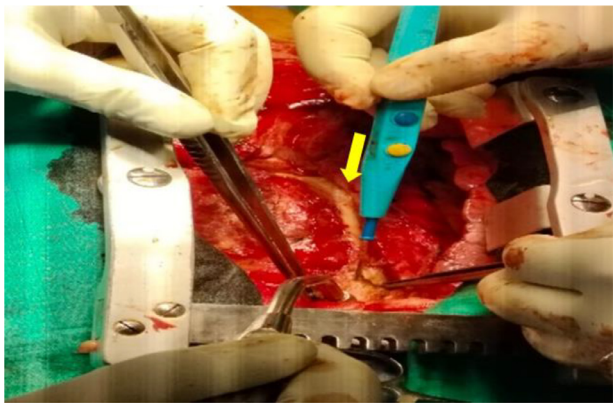

Fig. 5 – Intraoperative image showing pericardial thickeness and calcifications.

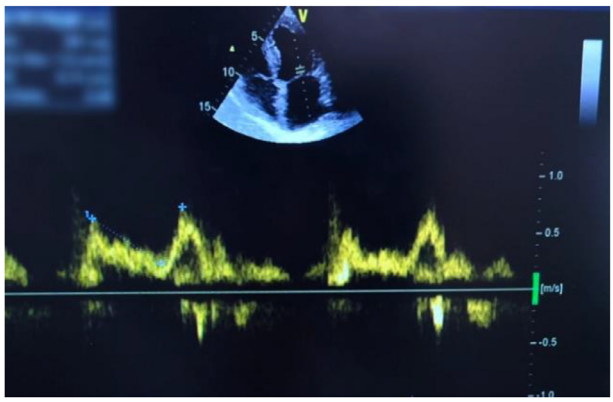

Fig. 6 – Postoperative TTE showing regression of respiratory flow variation.

Mycobacterium tuberculosis. The patient was put on antituberculosis treatment, then reviewed in consultations at 1 month, 3 months, and 6 months, showing good clinical evolution. The postoperative TTE (Fig. 6) check-up after 6 months showed a clear improvement in ultrasound parameters, notably regression of respiratory flow variation and normalization of septal motion.

Discussion

Chronic constrictive pericarditis (CCP) is one of the rare chronic pericardial pathologies but remains a serious condition. Hemodynamically, it leads to dissociation of intrathoracic and intracardiac pressures and increased interdependence of the 2 ventricles, resulting in paradoxal motion of the interventricular septum [1,8].

The etiologies of PCC are multiple, and according to Meganne N. Ferrel et al., in developed countries, the etiologies of PCC are mostly idiopathic, mediastinal radiotherapy, and cardiac surgery [9]. In contrast, tuberculosis is a very common cause in developing countries, as shown by the results of the study carried out by Chowdhury, U. K. et al. from 1985 to 2004, which reported that in 88% of cases, CCP was of tuberculous cause [10].

Table 1 – Diagnostic signs of constrictive pericarditis.*

Signs	Percentages of patients (%)
Jugular veins distension	93
Lower extremity edema	76
Hepatomegaly	53
Ascites	37

* In 135 patients who underwent pericardiectomy at the Mayo Clinic (Rochester, MN, USA) between 1985 and 1995 [1].

The clinical presentation is nonspecific and characterized by significant functional impairment such as dyspnea, asthenia, and sometimes palpitations associated with supraventricular rhythm disorders. Clinical examination reveals signs of right-sided heart failure in 88% of cases. These include jugular vein distention, hepatomegaly, ascites, and lower extremity edema (Table 1) [2,11].

The electrocardiogram may show nonspecific signs of pericardial constriction, including QRS microvoltage, atrial fibrillation, and isolated repolarization abnormalities. However, in addition to their unspecific properties, these signs are only reported in a minority of patients with PCC [12,13]. These clinical and electrical signs are not specific to CCP, so diagnosis requires furthur investigation such as multimodal imaging.

The Mayo Clinic has established several echocardiographic criteria for pericardial constriction. There are 5 main criteria (Fig. 7) that strongly suggest CCP. These include: Paradoxal septal motion (A); inspiratory decrease of the mitral E-wave velocity (B) at Pulsed-wave Doppler with a lower-than-expected E/e' velocity ratios {Annulus paradoxus}; on pulsed-tissue-wave Doppler, the velocity of the E' wave is normal or increased with a medial velocity (C) slightly higher than the lateral velocity (D) {Annulus reversus}; and end-diastolic flow reversals during expiration (E) [14].

In a clinical context suggestive of pericardial constriction, cardiac CT scan is an excellent tool to guide the diagnosis. However, in a study conducted by Talreja DR et al. involving 143 patients with confirmed constriction during surgery, 97 underwent preoperative CT scan; among them, 35% showed pericardial calcification, and 72% showed pericardial thickening [13].

Cardiac MRI serves as a complementary diagnostic tool to the previous ones, providing evidence of morphological and functional abnormalities associated with CCP. Morphological abnormalities include pericardial thickening more than 4-6 mm, pericardial calcifications (although these are poorly visualized on MRI), dilatation of the inferior vena cava, hepatic veins, and right atrium. Functional abnormalities are characterized by septal bounce in protodiastole, increased interdependence of the 2 ventricles, and pericardial adhesions, with systolic limitation of pericardial leaflet shift [6].

However, in challenging cases, right and left heart catheterization may be performed. This is an invasive method allowing the identification of hemodynamic parameters char-

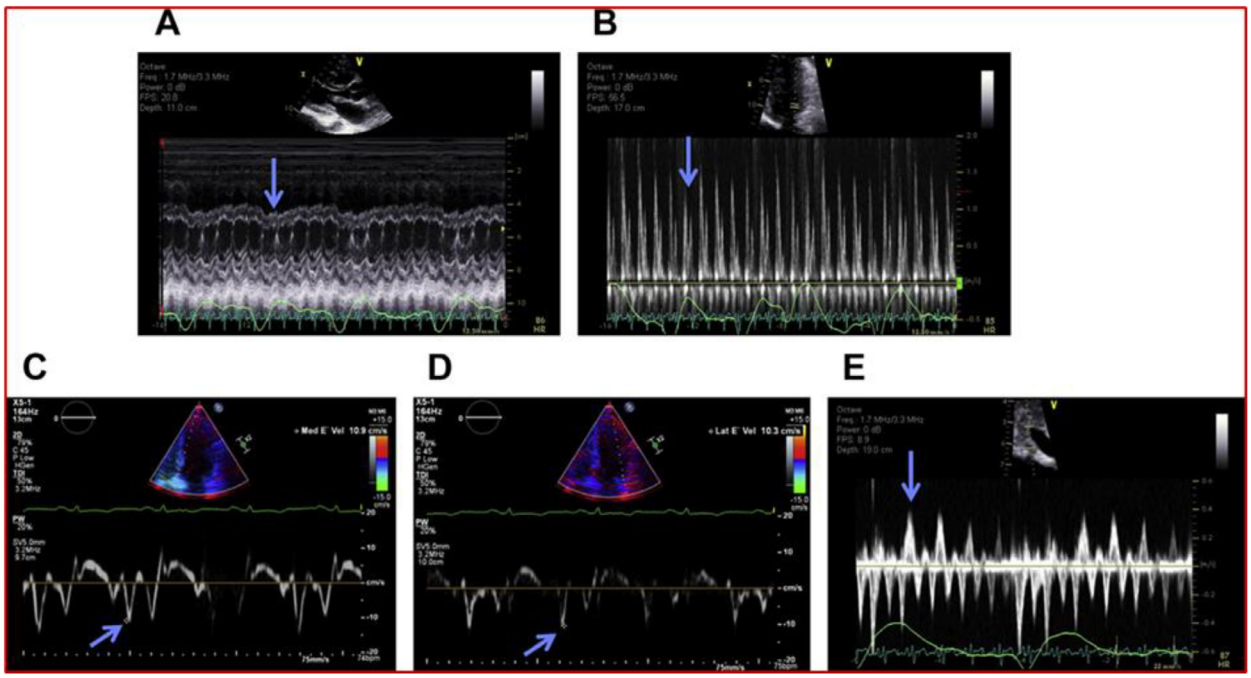

Fig. 7 – Principal echocardiographic findings in constrictive pericarditis.(A) Leftward ventricular septal shift in inspiration (arrow). (B) Inspiratory decrease in early (E) inflow velocity (arrow) at Pulsed-wave Doppler. (C) Medial and (D) lateral mitral annular tissue Doppler recordings. Note (arrows) normal to increased early relaxation velocity (e'), with medial velocity slightly greater than lateral. (E) Pulsed-wave Doppler recording within the hepatic vein. Note prominent end-diastolic flow reversals with expiration (arrow) [14].

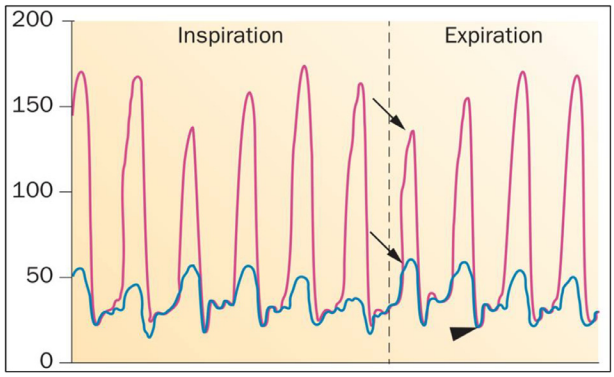

Fig. 8 – Simultaneous left (Pink curve) and right (Blue curve) ventricular pressure tracings in constrictive pericarditis showing a Dip-plateau waveform (arrowhead) [17].

acteristic of CCP. In CCP, cardiac catheterization can show a "dip-plateau" shape (Fig. 8) of right (RV) and left (LV) ventricular diastolic pressure curves, equalization of RV and LV end-diastolic pressures, and parallel ventricular interdependence to inspiration.

It is characterized, during inspiration, by an increase in RV systolic pressure and a concomitant decrease in LV systolic pressure. This is the same pathophysiological mechanism responsible of the respiratory variation in transvalvular flow on TTE [8,15,16]. In our patient's case, we did not use the invasive method of cardiac catheterization.

All these ultrasound, scan, MRI and cardiac catheterization criteria have been summarized by Terrence D. Welch et al. in Table 2 [17].

The initial management relies on symptomatic treatment with diuretics to reduce volume overload and on etiological treatment whenever a cause has been identified. In the absence of an optimal response to medical treatment, surgical pericardial decortication is the only effective treatment and is indicated in patients after 6-8 weeks of medical treatment combining diuretics and anti-tuberculous therapy in tuberculosis-related constrictive pericarditis [18].

Pericardiectomy was first successfully performed in 1913 by the German surgeon Ludwig Rehn and remains the treatment of choice for symptomatic constrictive pericarditis after failing medical treatment [19].

The outcome of patients after pericardial decortication depends on the NYHA stage prior to surgery, left ventricular function, renal function, and the time of intervention. Therefore, the 30-day mortality varies between 5% and 10%, regardless of etiology [11,12].

Previous findings support the importance of a comprehensive management approach for patients presenting with non-specific symptoms of PCC. This requires integrating their data in algorithms that rely on clinical, echocardiographic and multimodal imaging arguments to guide early diagnosis and management of PCC.

Table 2 – Principal diagnostic findings in constrictive pericarditis [15].

Echocardiography
- Two-dimensional: respiration-related ventricular septal shift and plethora of the inferior vena cava
- Hepatic vein Doppler assessment: prominent end-diastolic reversal of flow during expiration
- Mitral annular tissue Doppler : preserved or exaggerated e' velocities (medial e' typically ≥ 9 cm/s), often with medial e' greater than lateral e'
- Mitral inflow Doppler assessment: exaggerated respiratory variation in E velocity
- Speckle tracking myocardial strain imaging: preserved global longitudinal systolic strain, with relative
- reduction in lateral longitudinal strain compared with septal longitudinal strain

Computed tomography
- Pericardial thickening
- Pericardial calcification
- Deformation of cardiac contour
- Dilatation of the inferior vena cava

MRI
- Pericardial thickening
- Myocardial tagging sequences: pericardial–myocardial adherence
- Real-time cine imaging: respiration-related ventricular septal shift (as seen with echocardiography)
- Delayed imaging after gadolinium administration: pericardial enhancement, corresponding with inflammation

Hemodynamic catheterization
- Increased venous pressure
- Elevation and near-equalization of right and left heart diastolic pressures
- Relatively dissociated intrathoracic (pulmonary capillary wedge) pressure and left ventricular diastolic pressure (with inspiration, exaggerated decrease in pulmonary capillary wedge pressure compared with left ventricular diastolic pressure)
- Discordance in systolic pressure changes in the right and left ventricles during the respiratory cycle (with inspiration, right ventricular systolic pressure increases and left ventricular systolic pressure decreases)

Conclusion

Chronic constrictive pericarditis is a rare condition characterized by an insidious progression and notable for its polymorphic and nonspecific symptomatology, leading to difficulties and delays in diagnosis. There are multiple etiologies, and tuberculosis is the most common cause, specifically in developing countries. Advances in multimodal imaging have facilitated the diagnosis of pericardial constriction, especially in challenging cases. Pericardial decortication is the only effective treatment, improving the prognosis of patients who remain symptomatic despite medical treatment.

Patient consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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