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GRAND ROUND
*Heart failure*

# Inflammatory-mediated atrial cardiomyopathy diagnosed using multimodality imaging and successfully treated with prednisolone: a case report

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**Background** Atrial fibrillation is a common cardiac arrhythmia and often develops secondary to structural cardiac changes. Both the occurrence of atrial fibrillation and/or structural changes of the heart may lead to development of atrial cardiomyopathy and heart failure (HF). However, isolated atrial cardiomyopathy caused by focal atrial thickening is a rare condition, previously only described in case reports as a result of different aetiologies all linked to inflammation.

**Case summary** A patient with inflammatory-mediated atrial cardiomyopathy causing atrial fibrillation and acute decompensated HF presented as isolated left atrial wall thickening on transoesophageal echocardiography. The diagnosis was confirmed using multimodality imaging with transthoracic and transoesophageal echocardiography, cardiac magnetic resonance imaging, positron emissions tomography/computer tomography scanning and intracardiac echocardiography-guided endomyocardial biopsy. Despite no specific histological aetiology, the observed atrial cardiomyopathy might be associated with type 1 diabetes mellitus. The patient in the present case was successfully treated with prednisolone.

**Discussion** Diabetes mellitus is an important risk factor for developing atrial fibrillation and diabetic cardiomyopathy, due to reduced levels of anti-inflammatory and increased levels of proinflammatory cytokines causing cardiac inflammatory structural remodelling. The regression of the atrial thickening might be due to prednisolone's anti-inflammatory effects and thereby ability to suppress atrial remodelling and reduce the occurrence of atrial fibrillation. However, the effect of prednisolone might only affect the non-manifested inflammatory-mediated atrial remodelling. Due to the rare occurrence of isolated atrial cardiomyopathy a multiple imaging approach during the diagnostic process and follow-ups are essential to determine the aetiology and effect of the treatment.

**Keywords** Atrial wall thickening • Left atrial enlargement • Isolated atrial myopathy • Atrial remodelling • Atrial fibrillation • Diabetes mellitus • Case report

**ESC curriculum** 2.1 Imaging modalities • 6.1 Symptoms and signs of heart failure • 6.3 Heart failure with preserved ejection fraction • 6.5 Cardiomyopathy

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## Learning points

- To consider diabetes mellitus as a cause to inflammatory-mediated atrial cardiomyopathy.
- To consider prednisolone treatment in cases with inflammatory-mediated atrial thickening.
- To consider the occurrence of isolated inflammatory-mediated atrial cardiomyopathy in patients with *de novo* atrial fibrillation and decompensated heart failure with insufficient response to diuretic treatment.
- To consider a multiple imaging approach in case of diagnostic uncertainty and during follow-up to determine the aetiology of the atrial thickening and effect of the treatment.

# Introduction

Atrial fibrillation (AF) is a common cardiac arrhythmia and often associated with structural cardiac abnormalities, e.g. left atrial enlargement and heart failure (HF). Risk factors for AF include ageing, diabetes mellitus (DM), valvular heart disease, HF, and inflammatory disease.[1] A rare risk factor for AF is atrial cardiomyopathy due to alterations in the atrial function and arrhythmogenesis.[2] We report an atypical case of *de novo* AF and atrial cardiomyopathy caused by focal inflammatory atrial thickening. The diagnosis was confirmed using a multimodality imaging approach including transthoracic echocardiography (TTE), transoesophageal echocardiography (TOE), cardiac magnetic resonance imaging (CMI), positron emissions tomography/computed tomography (PET/CT), and intracardiac echocardiography-guided endomyocardial biopsy. Despite no definite diagnosis, the condition was successfully treated with prednisolone.

# Summary figure

| Time | Events |
|---|---|
| Day 0 | A 70-year-old woman with hypertension, type 1 diabetes mellitus (DM), fibromyalgia, as well as previous strokes admitted to the Emergency department (ED) with newly diagnosed normofrequent atrial fibrillation (AF) and acute decompensated heart failure. |
| Day 3 | Discharged from ED. |
| Day 13 | Readmission to ED due to worsening of dyspnoea, intermittent fever, fatigue and loss of appetite. |
| | Transthoracic echocardiography (TTE) with left ventricular ejection fraction of 60%, normal atrial sizes and right ventricular function and dilated inferior vena cava with reduced respiratory collapse. |
| Day 15 | Computer Tomography scan with no signs of malignancy. |
| | Thoracocentesis with no tumour cells in the pleural fluid. |
| Day 16 | Discharged from the Department of Internal Medicine. |
| Day 26 | Readmission due to worsening of dyspnoea. |
| | Transthoracic echocardiography with left ventricular ejection fraction of 60% and biatrial dilatation. |
| Day 27 | Transferred to the Department of Cardiology. |
| | Transoesophageal echocardiography (TOE) demonstrated abnormal thickening of the atrial endocardium including the left appendage. |
| Day 30 | Fluorodeoxyglucose (FDG) positron emissions tomography/computed tomography (PET/CT) scan demonstrated strong FDG-uptake in the left atrium (LA) and to a lesser extent the right atrium with no involvement in either of the ventricle. |
| Day 34 | Cardiac magnetic resonance imaging confirmed left atrial dilatation with wall thickening, while T1 and T2 mapping sequences revealed interstitial fibrosis and oedema of the atrial myocardium. Biventricular function was preserved with no scarring in the myocardium of the left ventricle. |
| Day 35 | Broad array of serological rheumatological parameters were tested, where only C-reactive protein and interleukin-2-receptor levels were increased. |
| | Intracardiac echocardiography-guided left atrial biopsy was performed and send to histomorphological, histochemical, immunohistochemical, and molecular pathological analyses. |
| Day 36 | Due to suspicion of inflammatory-mediated cardiomyopathy treatment with oral 37.5 mg prednisolone daily was initiated. |
| Day 44 | Prednisolone treatment was reduced to a dose of 25 mg daily. |
| Day 45 | Histological results of the atrial biopsy showed a chronic inflammatory infiltrate dominated by B-lymphocytes with a background of small macrophages, fewer T-lymphocytes and a negligible amount of plasma cells. Pathological diagnosis rendered was sub-endocardial fibrosis and chronic rheumatoid-like inflammation with possible association with DM. |
| Day 47 | The patient was discharged with prednisolone treatment gradually tapering over the following 11 weeks. |
| 2-months follow-up | Spontaneous conversion of AF to sinus rhythm as well as full regression of the PET/CT FDG-uptake, while the atrial thickening was nearly completely regressed verified by TOE. |
| 5-months follow-up | No signs of relapse on TOE, but occurrence of persistent AF and LA impairment. |

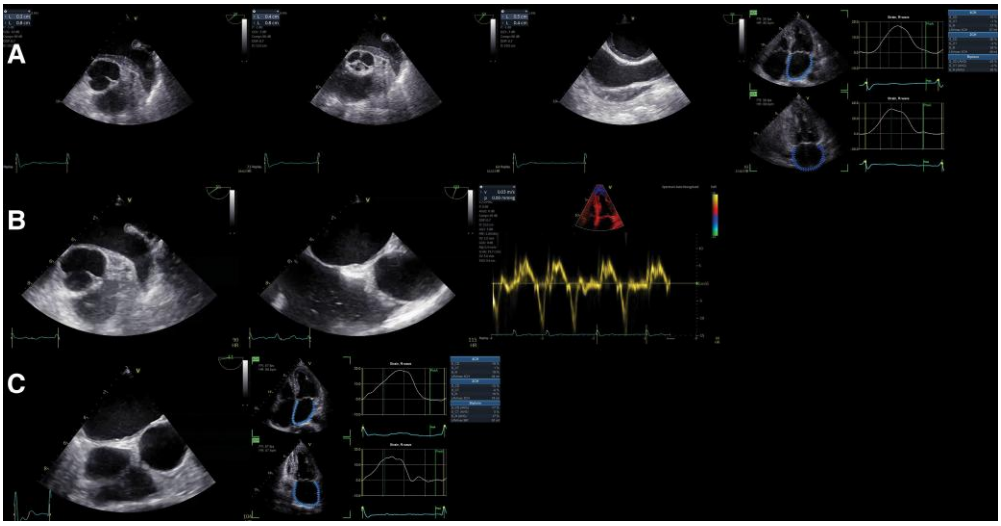


**Figure 1** Transoesophageal echocardiography and left atrial strain before and after prednisolone treatment. (*A*) TOE and left atrial strain at second readmission. (*B*) TOE and a' velocity following prednisolone treatment at two-months follow-up. (*C*) TOE and left atrial strain at three-months follow-up after ended prednisolone treatment. TOE, Transoesophageal echocardiography.

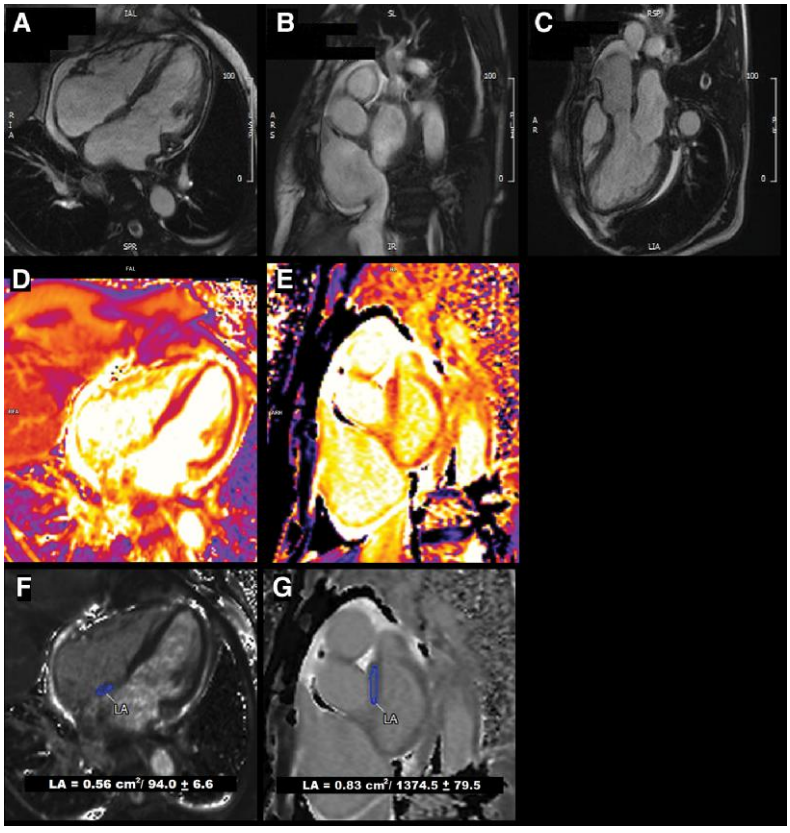


**Figure 2** Cardiac magnetic resonance imaging before prednisolone treatment. Cardiac magnetic resonance imaging confirming left atrium dilatation with extensive wall thickening. Mapping sequences revealed fibrosis suggestive of pathological inflammatory activity of the left atrium. (*A*) Cine sequence four-chamber view. (*B*) Cine sequence short axis view. (*C*) Cine sequence three chamber view. (*D*) T2 mapping four-chamber view. (*E*) T1 mapping short axis view. (*F*) T2 mapping four-chamber view with left atrial region of interest. (*G*) T1 mapping short axis view with left atrial region of interest.

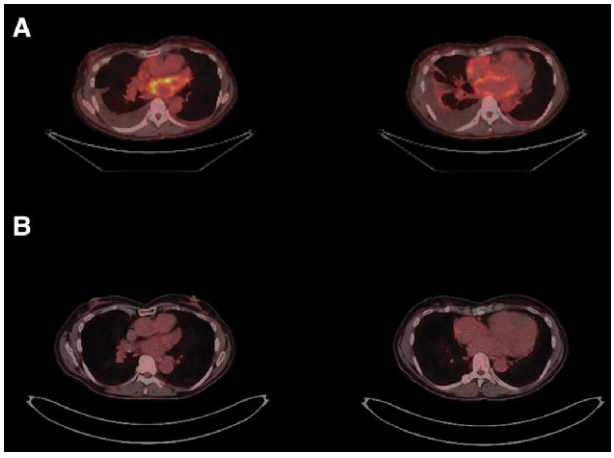


**Figure 3** Positron emissions tomography/computed tomography before and after prednisolone treatment. (*A*) PET/CT before prednisolone treatment. (*B*) PET/CT after prednisolone administration at two-months follow-up. PET/CT, positron emissions tomography/computed tomography.

## Case presentation

A 70-year-old woman with hypertension, type 1 DM, fibromyalgia, as well as previous ischaemic stroke, was admitted to the Emergency Department (ED) with symptoms of dyspnoea and abdominal pain. Heart auscultation demonstrated irregular rhythm without murmur, while auscultation of the lungs demonstrated vesicular respiration and discrete basal crepitations bilaterally. There were no signs of oedema of the lower extremities. Vital signs were with blood pressure of 171/77 mmHg, heart rate fluctuating between 64 and 107 b.p.m., temperature of 37.5°C, respiration rate of 16 per minute and peripheral oxygen saturation of 95% without oxygen supplement. Chest X-ray demonstrated right-sided consolidation of the lung parenchyma and pleura effusion. Symptoms, clinical signs and electrocardiogram (ECG) were consistent with newly diagnosed normofrequent AF and acute decompensated HF. Treatment with apixaban, oral metoprolol, and oral furosemide was initiated. On day-3 the patient was discharged from ED.

On day-13 following the primary contact, the patient was readmitted to ED due to worsening of dyspnoea, intermittent fever, fatigue, and loss of appetite. On admission, she had persistent normofrequent AF. At this point, physical examination demonstrated pitting oedema evolvement of the lower extremities up to knee level as well as occurrence of bilateral crepitations by lung auscultation. Transthoracic echocardiography (TTE) demonstrated left ventricular ejection fraction of 60%, normal atrial sizes and right ventricular function, dilated inferior vena cava with reduced respiratory collapse, no significant valvulopathies or signs of pulmonary hypertension. However, chest X-ray demonstrated progression of right-sided pleural effusion and consolidation. On the suspicion of malignancy, a thoracocentesis was performed, and pleural fluid was sent to cytological examination, from which no tumour cells were detected. No further examination of the pleural fluid was carried out, hence it was not possible to determine whether transudate or exudate was present. Furthermore, a computed tomography scan of the thoracic cavity, abdomen, and pelvis demonstrated a smaller pericardial effusion without signs of malignancy. Initially, 2-day treatment with piperacillin and beta-lactamase inhibitor and intravenous loop diuretic was initiated. The loop diuretic was converted to oral treatment before discharge from the Department of Internal Medicine on day 16.

On day 26, the patient was admitted to hospital for the third time due to worsening of dyspnoea with symptoms and clinical signs still consistent with cardiac decompensation and ECG demonstrating normofrequent AF. This led to a subsequent TTE, this time with the occurrence of biatrial dilatation, diastolic dysfunction with *E*/e' value of 12.6 and a mildly reduced left atrial strain of −23% (*Figure 1A*), while the left ventricular ejection fraction was preserved. Thus, HF with preserved ejection fraction due to AF was suspected. Transoesophageal echocardiography and subsequent direct current cardioversion were planned. However, TOE demonstrated abnormal thickening of the atrial endocardium including the left atrial appendage (*Figure 1A*). Due to the unknown significance hereof, cardioversion was not performed. CMI confirmed left atrial dilatation with wall thickening, while increased values on T1 and T2 mapping sequences revealed interstitial fibrosis and oedema of the myocardium suggestive of pathological inflammatory activity of the left atrium (*Figure 2*). Biventricular function was preserved with absence of scarring in the myocardium of the left ventricle. A subsequent fluorodeoxyglucose (FDG) PET/CT scan demonstrated strong FDG-uptake in the left atrium and to a lesser extent the right atrium with no involvement in either of the ventricles (*Figure 3A*). Due to suspicion of inflammatory-mediated cardiomyopathy, blood samples were tested for a broad array of serological rheumatological parameters. Of these, C-reactive protein and interleukin-2-receptor levels were increased, respectively 13.5 mg/L and 1354 kU/L, consistent with inflammatory activity. All other autoimmune serological parameters were within normal range (*Table 1*). Intracardiac echocardiography-guided left atrial biopsy was performed with histomorphological, histochemical, immunohistochemical, and molecular pathological analyses (*Figure 4*). A chronic inflammatory infiltrate was found dominated by B-lymphocytes with a background of small macrophages, fewer T-lymphocytes and a negligible amount of plasma cells. There was no amyloid deposition, no signs of IgG4-related disease, B-cell lymphoma or BRAF-mutated histiocytosis. Pathological diagnosis rendered was subendocardial fibrosis and chronic rheumatoid-like inflammation with possible association with DM.

Treatment with oral 37.5 mg prednisolone once daily was initiated, which resulted in significant symptomatic relief. Seven days following initiation, prednisolone treatment was reduced to a dose of 25 mg once daily, while intensifying the antidiabetic treatment due to diabetic derangement caused by prednisolone.

After 21 days, on day 47 following the first contact, the patient was discharged. Treatment with prednisolone was continued with gradual weaning over 11 weeks since no systemic inflammatory disease was verified, resulting in a total period of treatment of 12.5 weeks, which at two-month follow-up led to spontaneous conversion of AF to sinus rhythm as well as full regression of the PET/CT FDG-uptake, while the atrial thickening was nearly completely regressed verified by TOE (*Figures 1B* and *3B*). Despite the regression of atrial thickening, TTE Tissue Doppler measurement demonstrated a reduced a' velocity of 3 cm/s (*Figure 1B*). At five months follow-up, three months following prednisolone treatment termination, TTE and TOE demonstrated no signs of relapse, but a slight wall thickening remained, the diastolic function with *E*/e' of 11.2 as well as worsening of left atrial strain to −17%, which might be due to subendocardial fibrosis (*Figure 1C*). Furthermore, the termination of prednisolone resulted in better diabetic control and no shortness of breath, even though relapse of normofrequent AF occurred. The AF was accepted as permanent and a yearly follow-up was planned to detect the occurrence of a possible relapse. The rationale for prednisolone treatment was indicated on the occurrence of inflammatory cardiomyopathy in accordance to current European Society of Cardiology (ESC) guidelines and Danish national guidelines for inflammatory cardiomyopathies.[3,4] The choice of dosing and weaning were empirical and based on an evaluation of the severity of the patient's condition as well as a long weaning period aiming for an optimal treatment effect while minimizing the risk for side effects.

**Table 1** Blood samples before and after prednisolone treatment

| | Normal range | UNIT | Day 0 | Day 13 | Day 26–47 | | | Two-weeks control | Two-months control |
|---|---|---|---|---|---|---|---|---|---|
| **Organ markers** | | | | | | | | | |
| Troponin I | <47 | ng/L | | | 20 | | | | 10 |
| Creatinine kinase | 50–150 | U/L | | | | | 33 | | |
| Myoglobin | <75 | µmol/L | | | | | 66 | | |
| Pro-brain natriuretic peptide | <300 | µmol/L | | 1142 | 1394 | | | 1306 | 1309 |
| **Hepatology** | | | | | | | | | |
| Alanine transaminase | 10–45 | U/L | 69 | 86 | 61 | | | 39 | 25 |
| Amylase | 10–65 | U/L | 25 | 25 | | | | | |
| Lactate dehydrogenase | 115–255 | U/L | 219 | | | | | 222 | 203 |
| Acid phosphate | 35–105 | U/L | 92 | 124 | 125 | | | 81 | 59 |
| Bilirubin | 5–25 | µmol/L | 7 | 6 | 4 | | | 8 | 6 |
| Triglyceride | <2.0 | mmol/L | | | 28 | | | | |
| **Endocrinology** | | | | | | | | | |
| HbA1c | <48 | mmol/L | 56 | | | | | 70 | |
| Thyrotropin | 0.300–4.50 | IU/L | | | 5.18 | | | 1.34 | 1.76 |
| T3 | 1.10–2.50 | nmol/L | | | 1.1 | | | | |
| T4 | 60–140 | nmol/L | | | 89 | | | | |
| **Nephrology** | | | | | | | | | |
| Potassium | 3.5–4.6 | mmol/L | 3.3 | 3.6 | 4.1 | 4.0 | 4.7 | 3.8 | 4.1 |
| Natrium | 137–145 | mmol/L | 144 | 140 | 142 | 143 | 137 | 140 | 139 |
| Calcium ion | 1.18–1.32 | mmol/L | | 1.18 | 1.24 | | | | |
| Magnesium | 0.70–1.10 | mmol/L | | 0.74 | | | | | |
| Albumin | 34–45 | g/L | | | 28 | | | 36 | 36 |
| Creatinine | 45–90 | mmol/L | 52 | 55 | 124 | 78 | 105 | 81 | 87 |
| Carbamide | 3.1–7.9 | mmol/L | 3.3 | 3.5 | 11.6 | 6.6 | | 5.7 | 5.6 |
| eGFR | >60 | mL/min | >90 | >90 | 38 | | 46 | 63 | 58 |
| **Immunology** | | | | | | | | | |
| CRP | <8.0 | mg/L | 14.8 | 23.1 | 9.6 | | 13.5 | <4.0 | 13.3 |
| Interleukin 2 | 158–623 | kU/L | | | | | 1354 | 796 | 963 |
| Immunoglobulin G | 6.1–14.9 | mg/L | | 14.3 | | | | | |
| Leucocytes | 3.50–10.0 | 10^9/L | 6.9 | 7.2 | 5.7 | 7.0 | 9.6 | 9.2 | 6.8 |
| Neutrophils | 2.00–7.00 | 10^9/L | 5.26 | 4.71 | 3.63 | | 8.28 | | 4.67 |
| Metamyelocytes + Myelocytes + Promyelocytes | <0.05 | 10^9/L | <0.02 | 0.02 | 0.02 | | 0.04 | | 0.03 |
| Lymphocytes | 1.30–3.50 | 10^9/L | 0.91 | 1.48 | 1.14 | | 0.87 | | 1.39 |
| Monocytes | 0.20–0.70 | 10^9/L | 0.61 | 0.77 | 0.66 | | 0.38 | | 0.5 |

*Continued*

**Table 1** *Continued*

| | Normal range | UNIT | Day 0 | Day 13 | | Day 26–47 | | Two-weeks control | Two-months control |
|---|---|---|---|---|---|---|---|---|---|
| Eosinophils | <0,50 | 10^9/L | 0.07 | 0.17 | 0.14 | | <0.02 | | 0.04 |
| Basophils | <0,10 | 10^9/L | 0.05 | 0.09 | 0.07 | | <0.02 | | 0.05 |
| ANA | <1 | ratio | | | | <1 | <1 | | |
| ANA, Hep-2 | | | | | | | unmeasurable | | |
| ANCA | | | | | | | unmeasurable | | |
| Myeloperoxidase | <3.5 | kIU/L | | | | <3.5 | | | |
| Proteinase-3 | <2 | kIU/L | | | | <2 | | | |
| Peptidyl dipeptidase | 12–60 | U/L | | | | | 26 | | |
| Urate | 0.15–0.40 | mmol/L | | 0.3 | | | | 0.42 | 0.34 |
| **Hematology** | | | | | | | | | |
| Haemoglobin | 7.3–9.5 | mmol/L | 6.7 | 7.1 | 6.0 | 6.7 | | 6.9 | 7.2 |
| MCV | 82–98 | fl | 94 | 95 | 95 | | | | |
| MCHC | 19.7–22.2 | mmol/L | 20.5 | 20.3 | 20.1 | | | | |
| Reticulocytes | 31–97 | 10^9/L | 56 | 53 | | | | | |
| Thrombocytes | 165–400 | 10^9/L | 166 | 206 | 188 | | | 140 | 191 |
| Kappa–lambda | 3.3–19 | mg/L | | 36 | | | | | |
| Lambda-chains | 5.7–26 | mg/L | | 43 | | | | | |
| Kappa/lambda-chain | 0.26–1.7 | ratio | | 0.83 | | | | | |

ANA, antinuclear antibodies; ANCA, antineutrophil cytoplasmic antibodies; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; Hb1Ac, haemoglobin A1c; HEp-2, human epithelial-2; IU, international units; MCHC, mean corpuscular haemoglobin concentration; MCV, mean corpuscular volume; NT-proBNP, N-terminal pro-brain natriuretic peptide; T3, triiodothyronine; T4, thyroxine.

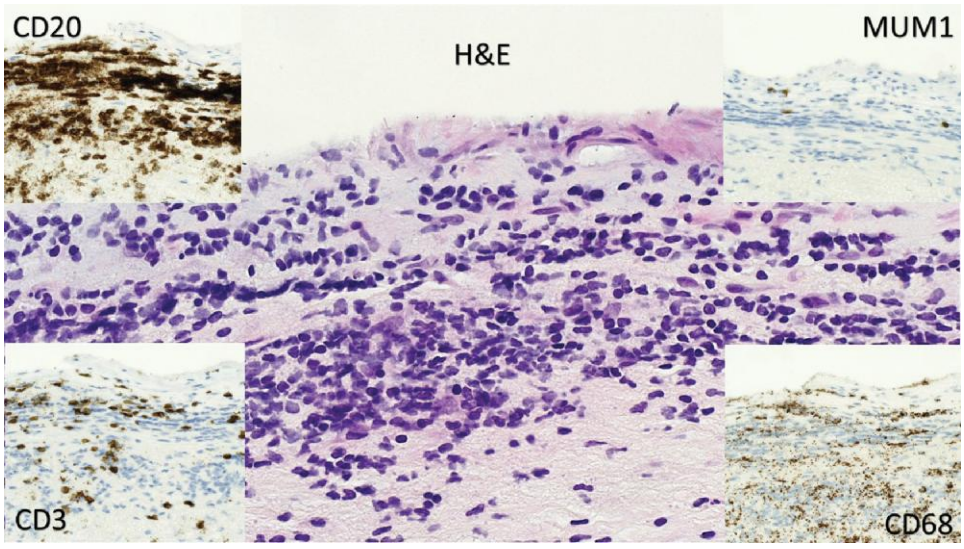


**Figure 4** Endocardial biopsy before prednisolone treatment. Endocardial left atrial biopsy demonstrating extensive chronic inflammation dominated by B-lymphocytes. Haematoxylin Eosin stains as background. CD20, B-lymphocytes; CD3, T-lymphocytes; MUM1, plasma cells; CD68, macrophages.

## Discussion

Isolated atrial thickening has been described in previous cases and studies as a result of giant cell myocarditis, lymphocytic myocarditis, IgG4-mediated atrial cardiomyopathy, rheumatic heart disease, or of unknown aetiology.[5–9] Inflammatory-mediated atrial cardiomyopathy with focal thickening is a rare condition, previously only described in case reports with different aetiologies all linked to inflammation and with clinical effect after prednisolone administration.[10,11] While the FDG PET/CT scan and myocardial biopsy revealed inflammatory atrial cardiomyopathy, we did not find any definite cause of the isolated atrial thickening in the present case. However, the myocardial biopsy suggested a possible association between the atrial wall thickening and type 1 DM, which at the time of admission was dysregulated. DM may resolve in diabetic cardiomyopathy and is an important risk factor of AF.[1,2] The electrophysiological properties of the left atrium change with myocardial thickness, thus structural changes in the atrial wall may lead to AF. The possible association with DM in this case is due to the negative serological rheumatological blood arrays as well as the overabundance of B lymphocytes, which is a common finding in antibody-associated immune diseases including DM, and known to secrete reduced levels of anti-inflammatory cytokines in DM while the secretion of proinflammatory cytokines remain increased.[12] Early diabetes-induced cardiac inflammatory structural changes are localized to the epicardial adipose tissue with propagation to the atrial wall.[12,13] The proarrhythmogenic properties are a result of cardiac insulin-resistance and local inflammation. The local inflammation stimulates to an increased production of advanced glycation end-products, which results in increase of oxidative stress in the cell, thus resulting in apoptosis, as well as cross-linking of the extracellular matrix proteins leading to activation of fibroblasts and connective tissue involvement. Furthermore, the insulin-resistance also reduces the insulin-stimulated production of nitric oxide in the cardiac microvasculature resulting in microvascular dysfunction. Overall, this leads to diabetes-induced cardiac remodelling comprised of cardiac stiffness, myocyte hypertrophy, and fibrosis. The structural changes may cause atrial cardiomyopathy, which then may lead to AF, due to alterations of the atrial function and arrhythmogenesis.[2,14,15] Normally, diabetic cardiomyopathy involves both atria and ventricles. However, it is possible that the focal atrial involvement in the present case could be a result of early onset of diabetes-induced cardiac remodelling. Initially, IgG4-related disease, lymphocytic heart disease, Erdheim-Chester disease as well as amyloid disease were suspected, but these were not verified by the blood samples, CMI or the myocardial biopsy, while the histological evaluation of the atrial myocardial biopsy with findings consistent with long-term chronic inflammation with sub-endocardial fibrosis. Moreover, the patient underwent full-body PET/CT scanning without evidence of underlying inflammatory systemic disease. Furthermore, in this case the rather fast development of atrial dilatation is noticeable, although we cannot with certainty arbitrate whether the wall thickening was present during the first TTE. Therefore, the specific underlying pathophysiological mechanisms causing the focal atrial thickening in the present case remains speculative and might not solely be due to DM.

Previous studies have found prednisolone to be able to suppress atrial remodelling and reduce the recurrence of AF due to its potent anti-inflammatory effect.[16,17] The patient in the present case was treated with prednisolone monotherapy with full regression of inflammation assessed by FDG PET/CT imaging. While prednisolone treatment did not result in complete atrial wall thickening regression, this most likely represents sub-endocardial fibrosis resistant to prednisolone treatment. Brain natriuretic peptide is secreted from both ventricular and to a lesser extent atrial myocytes. Despite successful prednisolone treatment with full symptomatic and inflammatory relief, TTE demonstrated impaired LA function as assessed by LA strain and a' velocity, and likely as a consequence hereof a rise in N-terminal pro-brain natriuretic peptide level at follow-up.[18–20] Thereby, the anti-inflammatory effects of prednisolone might only affect the non-manifested inflammatory-mediated atrial remodelling, which despite successful treatment with prednisolone on the patient's symptoms and inflammation, the inflammatory atrial cardiomyopathy led to atrial fibrosis with persistent AF and impairment of LA function.

## Conclusion

We documented the occurrence of inflammatory-mediated atrial cardiomyopathy causing atrial fibrillation and acute decompensated HF

successfully treated with prednisolone. The diagnosis was confirmed using a multimodality imaging approach. Despite no specific histological aetiology, the observed atrial cardiomyopathy might be associated with type 1 DM.

## Lead author biography

Birgitte Cabuhn Larsen is a medical doctor from Aarhus University, Aarhus, Denmark and graduated in 2020. Since 2021, Birgitte Cabuhn Larsen has held junior resident positions within the field of cardiology. Initially, at the Department of Cardiology, Aarhus University Hospital and currently at the Department of Internal Medicine, Regional Hospital Randers.

## Patient consent

The authors confirm that written consent for submission and publication of this case report including the images and associated text have been obtained from the patient in line with COPE guidance.

**Conflict of interest**: None of the authors had a conflict of interest.

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## Data availability

The data underlying this case report are incorporated into the article.

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