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A case of atrial fibrillation complicated by complete atrioventricular block

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Abstract

Atrial fibrillation and complete atrioventricular block are two well-established arrhythmias that can share common aetiologies and risk factors. Although the two arrhythmias can co-exist, only a limited number of cases of atrial fibrillation complicated by complete atrioventricular block have been reported. Correct recognition is essential due to the risk of sudden cardiac death. A 78-year-old female with known atrial fibrillation presented with a 1-week history of shortness of breath, chest tightness and dizziness. On assessment, she was bradycardic with a heart rate of 38 bpm, despite the absence of any rate-limiting medication. Electrocardiography revealed an absence of P waves with a regular ventricular rhythm, consistent with a diagnosis of atrial fibrillation complicated by complete atrioventricular block. This case highlights the diagnostic electrocardiography features of co-existing atrial fibrillation with complete atrioventricular block that are often misinterpreted, leading to a delay in correct diagnosis and initiation of definitive management. Upon diagnosis, it is essential to exclude the reversible causes of complete atrioventricular block before considering permanent pacing. In particular, this includes rate-limiting medications in patients with pre-existing arrhythmias such as atrial fibrillation and electrolyte disturbances.

Keywords

Atrial fibrillation, complete heart block, bradycardia, arrhythmia, pacemaker

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Introduction

Atrial fibrillation (AF) is the most common type of cardiac arrhythmia, characterised by uncoordinated and dyssynchronous atrial contraction resulting in irregular and often rapid ventricular excitation.[1] Its prevalence has continued to increase on a worldwide scale, with a two-fold increase in the number of individuals with AF anticipated by 2050, and a lifetime risk of 22% by the age of 80.[2] Its classic electrocardiography (ECG) findings are well established as an irregularly irregular ventricular rhythm with narrow QRS complexes (<120 ms) with an absence of P waves.

In comparison to AF, complete or third-degree atrioventricular (AV) block is a rarer arrhythmia, caused by a complete loss of electrical impulse transmission between the atria and ventricles, with absent AV nodal conduction.[3] This results in a failure of heart rate control and uncoordinated atrial and ventricular activity, which can compromise cardiac output. Complete AV block can present with significant dyspnoea and chest pain, and if left undiagnosed can lead to acute heart failure and sudden cardiac death. Severe bradycardia with independent atrial and ventricular rates is seen on ECG.[4] The definitive management is insertion of a permanent pacemaker to synchronise atrial and ventricular activity.

Although the pathophysiology and ECG features of both AF and complete AV block are well described, it is important to note that the two arrhythmias are not mutually exclusive, and can co-exist.[5,6] Despite this, the diagnostic ECG characteristics of AF complicated by complete AV block are less well recognised and can often be misdiagnosed, compromising patient management and giving risk of acute complications.

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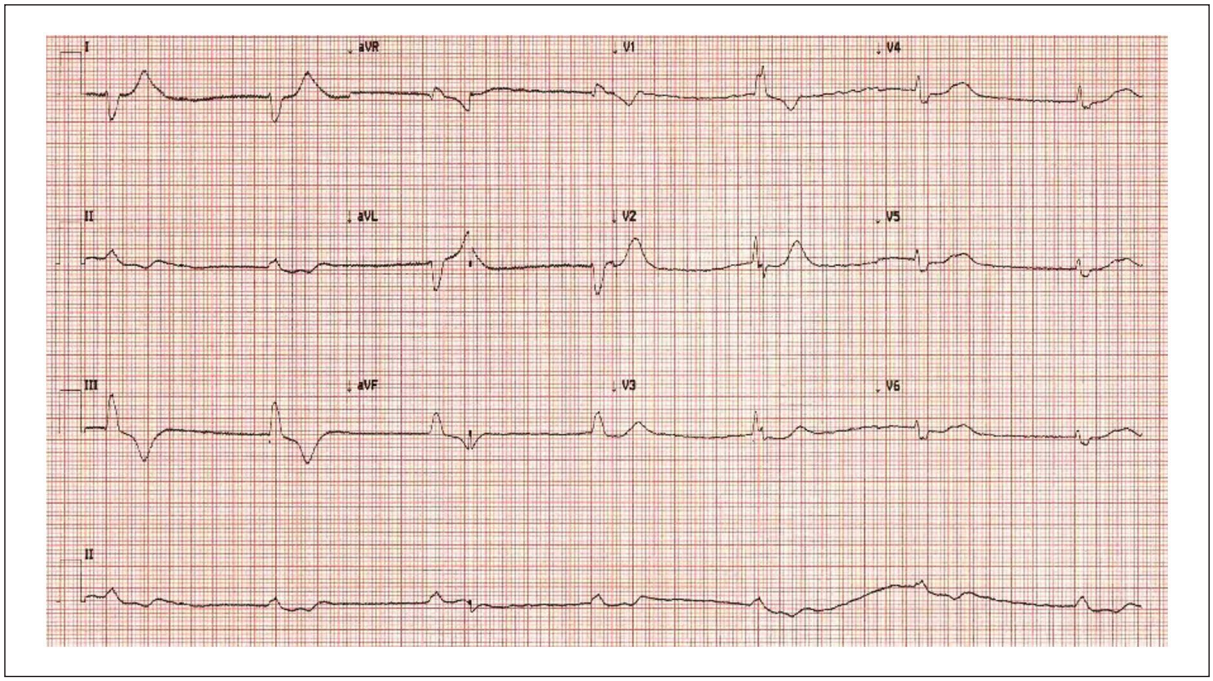

Figure 1. Atrial fibrillation complicated by complete atrioventricular block.

Here, we present the case of a patient with known permanent AF who was found to be in complete AV block after presenting with acute breathlessness, chest tightness and dizziness.

Case

A 78-year-old female patient presented to the emergency department with a 1 week history of shortness of breath accompanied with chest tightness, dizziness and vomiting. Her dizziness particularly on standing had resulted in a fall on the day of admission to hospital. Her symptoms were consistent with a class 3 New York Health Association cardiac function.[7]

Her past medical history included endometrial cancer with lung metastasis, Type 2 diabetes mellitus, hypertension, chronic kidney disease and permanent AF, with a total HAS-BLED score of 4 (scoring for the presence of hypertension, renal disease, age >65 years old and alcohol intake of ⩾8 units/week), indicating a high risk of major bleeding with anticoagulation.[8] Importantly, of note, she was not taking any rate-limiting medications for AF. She was, however, dependent on repeated blood transfusions due to chemotherapy- and radiotherapy-induced anaemia. The patient-reported weight loss of 12 kg over the last 3 months, from 74 to 62 kg. Her body mass index was 19.

On examination, the patient looked clinically unwell, with evident skin and conjunctival pallor. Peripheries were cool to touch, and the jugular venous pressure was raised at a height of 7 cm. She was found to be bradycardic with a regular heart rate of 38 bpm. Auscultation of the chest revealed bilateral basal crackles with reduced air entry at both lung bases. Heart sounds were unremarkable on auscultation with no additional gallops or murmurs. Bilateral pitting oedema was apparent in the lower limbs. Her calves were soft and non-tender.

Investigations

Blood tests on admission showed a white cell count (WCC) of 13 ($4.0–11.0 \times 10^9$ L), a haemoglobin (Hb) of 73 (120–150 mg/L) and a C-reactive protein (CRP) of 3.4 mg/L (0–4.9 mg/L). Renal function was acutely impaired, with a creatinine of 124 µmol/L (44–80 µmol/L) and a urea of 12.9 mmol/L (2.5–7.8 mmol/L). Her serial troponin T-tests were 31.6 and 36.4 ng/L (0–14 ng/L), respectively. Creatine phosphokinase-MB (CK-MB) level was 10 IU/L (5–25 IU/L). Electrolytes were unremarkable, with a sodium of 135 mmol/L (133–145 mEq/L), potassium of 4.0 mmol/L (3.5–5.2 mmol/L), magnesium of 0.9 mmol/L (0.8–1.10 mmol/L) and calcium of 2.4 mmol/L (2.2–2.6 mmol/L).

Serum albumin was 27 g/L (35–50 g/L) and total protein was 46 g/L (60–80 g/L). Thyroid function tests were unremarkable, with a thyroid-stimulating hormone (TSH) of 1.97 mU/L (0.27–4.20 mU/L), Free T4 of 14.4 pmol/L (12.0–22.0 pmol/L) and Free T3 of 4.6 pmol/L (3.1–6.8 pmol/L). B-type natriuretic peptide (NT-pro BNP) was elevated at 5000 pg/mL (<2000 pg/mL).

An ECG performed on admission highlighted severe bradycardia (at a rate of 38 bpm), an absence of P waves and a regular ventricular rhythm, consistent with a diagnosis of AF complicated by complete AV block (Figure 1). The ECG showed a QRS duration of 151 ms and QTc interval of

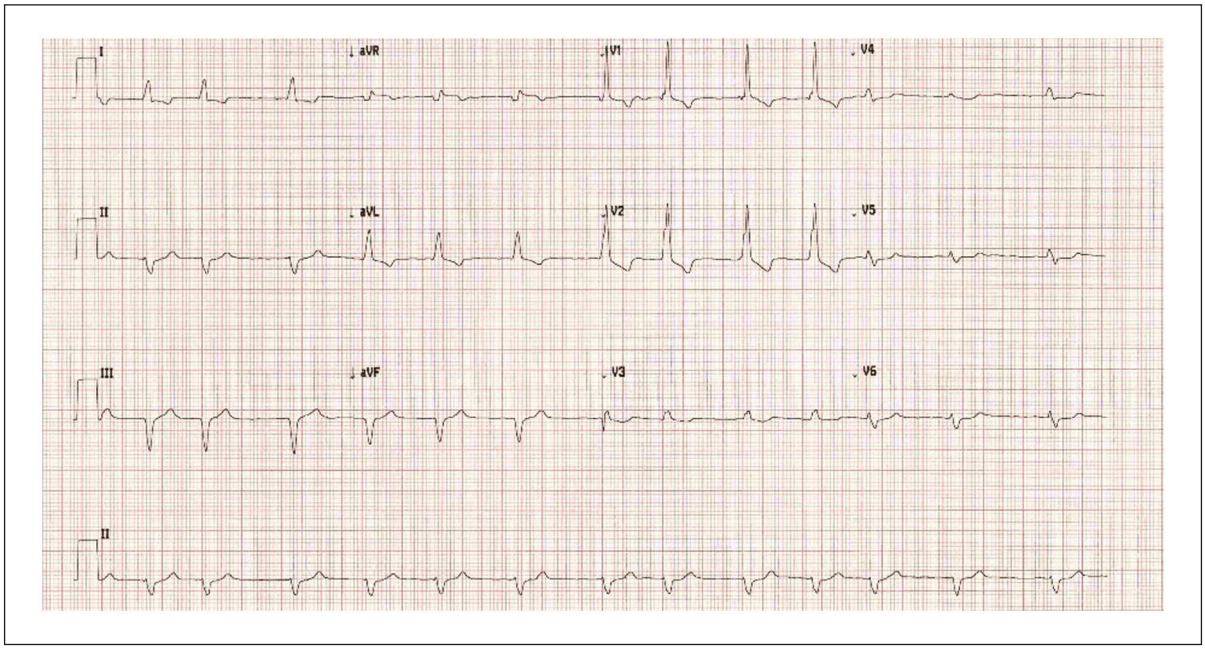

Figure 2. Permanent atrial fibrillation 12 months ago.

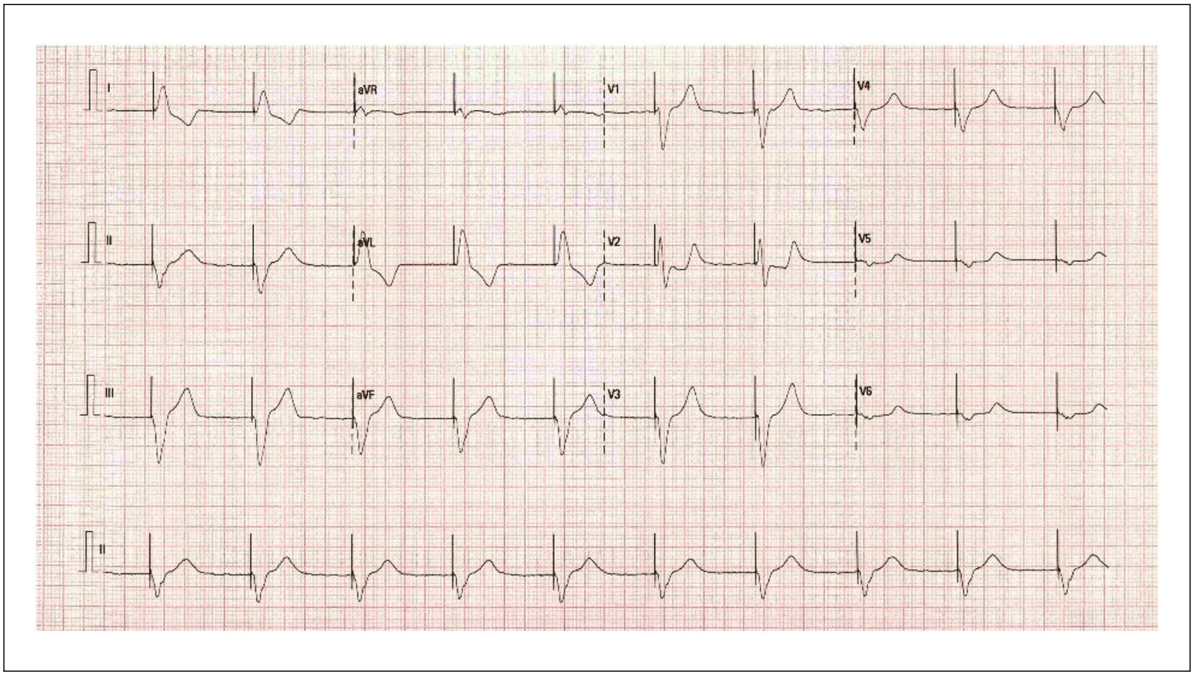

Figure 3. Stimulated and paced rhythm following implantation of a ventricular pacemaker.

453 ms. Right axis deviation with right bundle branch block was also noted.

This was in stark contrast to her baseline ECG 12 months ago, which displayed an irregularly irregular rhythm with an absence of P waves and a variable ventricular rate (60–80 bpm), consistent with AF (Figure 2). QRS and QTc duration were 141 ms and 402 ms, respectively. Left axis deviation with no bundle branch block was noted. Interestingly, pathological Q waves in the inferior leads (II, III, and aVF) along with prominent R waves in the anteroseptal leads (V1 and V2) were noted at present (Figure 1) and 12 months ago (Figure 2). These findings potentially indicate the background of a previous inferior and/or posterior myocardial infarction, and/or chronic ischaemia.

Figure 3 illustrates the paced rhythm following implantation of a ventricular pacemaker. QRS and QTc duration were

148 ms and 480 ms, respectively. Left axis deviation was also visible; however, there was no bundle branch block.

Echocardiography demonstrated good left ventricular systolic function, with an ejection fraction of 55%. Other than mild mitral regurgitation, the appearance of all other heart valves was unremarkable with no evidence of cardiac metastases. There was no evidence of local asynergy.

An erect anterior-posterior chest radiograph revealed bilateral moderate pleural effusions. Gross pulmonary congestion was evident.

A computerized tomography (CT) scan of the chest, abdomen and pelvis exhibited multiple bilateral pulmonary nodules suggestive of bilateral pulmonary metastasis, significant mediastinal, bilateral hilar and supraclavicular lymphadenopathy and bilateral pleural effusions.

Differential diagnosis

The patient's symptoms of shortness of breath, chest pain and dizziness were likely a combination of severe bradycardia secondary to AF complicated by complete AV block, along with anaemia and pulmonary metastases. Furthermore, the presence of bilateral pleural effusions raised the potential of cardiac failure and/or metastatic effusions secondary to her endometrial cancer, which could be exacerbating her shortness of breath.

Treatment

Acutely, the patient was resuscitated with one unit of red blood cells and oxygen therapy. The decision was made for implantation of a single-chamber ventricular pacemaker to manage the bradyarrhythmia of combined AF and complete AV block. Specifically, a Medtronic pacemaker with pacing and sensing activity specific to the ventricles with inhibitor activity (VVI), model MCIAVR1 0.8 cm^3 in volume with encore programming and capture management was implanted.[9] The VVI pacemaker was implanted into the endocavitary of the heart via the right femoral vein.

Intravenous diuretics were also commenced to treat pulmonary congestion and peripheral oedema. The patient remained clinically stable and was discharged 3 days later.

Outcome and follow-up

The patient reported an improvement in her symptoms of breathlessness, dizziness and chest tightness 1 month following permanent pacemaker insertion in the cardiology outpatient clinic. The patient's weight was closely monitored following discharge. A weight loss of 1.5 kg was noted after 1 month (from 60 kg on discharge from hospital to 58.5 kg). Hb remained stable at 85 g/L (pre-discharge Hb of 90 g/L). She is currently able to perform certain activities of daily living independently, which has improved her quality of life.

During her inpatient admission, the patient also underwent a right-sided pleural aspiration for therapeutic and diagnostic purposes. Pleural fluid cytology and immunocytochemistry revealed malignant endometrial cancer cells, consistent with metastatic pleural effusions.

Discussion

This is a rare case of a patient with known AF who was simultaneously found to be in complete AV block, causing profound symptomatic bradycardia. The ECG findings leading to this diagnosis were an absence of P waves with a regular but bradycardic ventricular rhythm. These ECG features have clear contrast to the irregular rhythm and variable ventricular rate that characterise AF.

There are several explanations for why our patient developed complete AV block in the context of established AF. Both arrhythmias share several common aetiologies, the most pertinent of which is ischaemic heart disease.[5,6,10–20] Although an acute myocardial infarction was excluded with serial troponin T-tests which demonstrated no significant rise and serial ECGs which confirmed an absence of dynamic ischaemic changes, the potential of a previous myocardial infarction and/or ongoing chronic ischaemia cannot be excluded. ECGs performed 12 months ago and on admission (Figures 1 and 2) exhibited pathological Q waves in the inferior leads (II, III and aVF) along with prominent R waves in the anteroseptal leads (V1 and V2). These ECG findings coupled with the patient's established risk factors for ischaemic heart disease (hypertension, Type 2 diabetes mellitus and an advanced age) may well mean the presence of ischaemia in the right coronary artery and/or left circumflex artery that led to impaired conduction at the level of the AV node and ultimately complete AV block.

Another potential trigger to the development of complete AV block is the patient's chemotherapy regimen of paclitaxel and cisplatin in addition to radiotherapy for the management of metastatic endometrial cancer.[12] Chemotherapy and radiotherapy predispose to the development of several arrhythmias including complete AV block through primary or secondary mechanisms. Primary mechanisms involve chemotherapy drugs interrupting specific molecular pathways critical to cardiac condition system, whereas secondary mechanisms involve structural damage to the endocardium, myocardium and/or pericardium through ischaemia, inflammation and radiotherapy.[12] With respect to the patient's chemotherapy regimen, paclitaxel is known to induce arrhythmias through primary mechanisms, specifically by releasing histamine that ultimately leads to QTc interval prolongation.[13] On the contrary, cisplatin predisposes to myopericarditis.[12] Several case reports of cisplatin-mediated complete AV block for the treatment of cholangiocarcinoma and squamous cell carcinoma of the skin have been documented in the literature.[14,15]

As well as cancer treatment being a cause of complete AV block, the presence of cancer itself, either as a primary cardiac tumour or secondary metastatic myocardial disease, can promote the development of arrhythmias including complete AV block.[16] Importantly of note, the potential of cardiac metastases from the primary endometrial cancer was excluded on echocardiography.

The patient's malnutrition and weight loss secondary to metastatic endometrial cancer must also be considered in the aetiology of the complete AV block. Malnutrition in malignancy results from a combination of reduced synthetic function, gastrointestinal malabsorption, increased renal protein losses and eventually attenuated oral intake.[17] Ultimately, this cumulates in myocardial atrophy and a reduction in cardiac output. Impaired myocardial function promotes the development of arrhythmias including complete AV block.[18]

Reversible and iatrogenic causes of complete AV block must also be excluded before proceeding with implantation of a permanent pacemaker. These include electrolyte disturbances, in particular hyperkalaemia and hypermagnesemia, infection and medications.[10,11] It was particularly pertinent in our patient's case to exclude the presence of rate-limiting medications for the management of her pre-existing AF. Digoxin, beta blockers, calcium channel blockers and antiarrhythmic agents from all four classes can impair AV nodal conduction and predispose to the development of complete AV block.[3]

On reflection, the development of complete AV block in our patient with established AF is likely multifactorial. The patient had ECG evidence of chronic inferior myocardial ischaemia, a history of treatment with two cardio-toxic chemotherapy agents in addition to radiotherapy and objective malnutrition.

Conclusion

Complete AV block and AF are by no means mutually exclusive. The former arrhythmia has the potential to complicate patients with acute or long-standing AF due to shared aetiologies and risk factors. These include ischaemic heart disease, electrolyte disturbances, acute infection, cardiomyopathies and valvular diseases. Reversible causes of complete AV block such as medication toxicity and electrolyte disturbances must first be excluded before permanent pacemaker implantation is considered.

Contributors

This case was diagnosed and managed by the cardiology team, of which N.K. was the consultant, and Y.Y. and W.I. were the senior house officers. Y.Y. wrote the abstract, background, case report, investigations, differential diagnosis, treatment, outcome and follow-up, discussion, patient perspective and learning points. W.I. obtained written consent from the patient's next of kin and obtained Figures 1 and 2. N.K. critically reviewed the manuscript and suggested changes to be made. Y.Y. implemented these changes.

Declaration of conflicting interests

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Ethics approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

The authors confirm that written informed consent was obtained from the patient's legal authorised representative – her daughter and formal next of kin on 23 March 2022, for anonymized patient information to be published in this article. Written informed consent was taken from the legal authorised representative because the patient lacked capacity at the time of consent.

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