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How Long Can One Live Without a Permanent Pacemaker in Complete Heart Block? A Case Report from Rural Kenya

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ABSTRACT

Complete heart block is characterized by a total atrioventricular dissociation resulting in junctional or ventricular escape rhythms. It is associated with high cardiovascular mortality rates without definitive treatment. The current guidelines recommend the placement of a permanent pacemaker to manage all cases of a complete heart block not caused by reversible or physiological causes, irrespective of patient symptoms. The natural history and prognosis of complete heart block not treated with pacemaker placement are unknown. In this study, we present the case of an elderly woman from rural Kenya who was diagnosed with a complete heart block 30 years ago and recommended a permanent pacemaker but declined. She has lived a relatively normal and active life without a pacemaker, albeit with compensated heart failure. This case suggests that a subset of patients with complete heart block may have good clinical outcomes without the need for a permanent pacemaker. It would be worthwhile to do further studies to map this subset of patients' natural history and prognostic factors.

INTRODUCTION

A complete heart block (CHB) (also called third-degree atrioventricular block) occurs when there is no conduction at all from the atria to the ventricles and may be paroxysmal or permanent (Kusumoto *et al.*, 2019). The causes may be reversible or irreversible, including myocardial infarction, myocarditis, infective endocarditis, cardiomyopathy, hyperkalemia, atrioventricular nodal blocking medications (e.g., beta-blockers, calcium channel blockers, digoxin, adenosine, etc.), post-cardiac surgery/procedures, congenital heart block, idiopathic, etc. (Meloy *et al.*, 2022). Patients may present with fatigue, chest pain, dyspnea, palpitations, presyncope and syncope, heart failure, cardiogenic shock, or sudden cardiac arrest (Meloy *et al.*, 2022). They have bradycardia due to the occurrence of escape rhythms, which may be junctional (rates of 40-60 bpm) or ventricular (rates of 20-40 bpm). Patients with CHB have very poor outcomes without prompt and appropriate treatment with pacemaker placement (Knabben *et al.*, 2025). CHB is an independent predictor of in-patient and 6-year mortality (Savic *et al.*, 2021). The prevalence of CHB varies widely based on various populations, e.g., 0.004% in China to 0.04% in Iceland and Michigan (Kojic *et al.*, 1999; Ostrander *et al.*, 1965; Shan *et al.*, 2021). The diagnosis is confirmed on an electrocardiogram, which shows severe bradycardia and evidence of atrioventricular dissociation: the atrial (P waves) and ventricular (QRS complexes) activities are independent of each other. The atrial rate is higher than the junctional or ventricular escape rate. See Figures 1 and 2 below by Yang *et al.* (2018) and Bhasin and Roy (2019), respectively. Electrophysiology (EP) study may rarely be used to make the diagnosis.

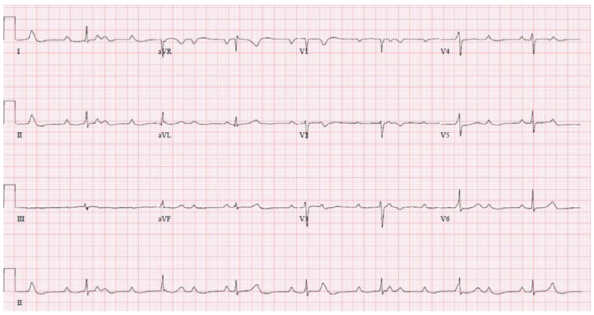

Figure 1: The EKG shows a complete heart block with a junctional escape rhythm, an atrial rate of around 90 bpm, and a ventricular rate of 45 bpm (Yang *et al.*, 2018).

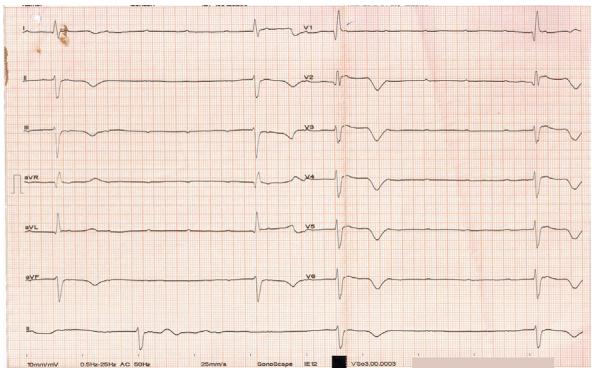

Figure 2: The EKG shows a complete heart block with a ventricular escape rhythm, an atrial rate of about 88 bpm, and a ventricular rate of about 30 bpm (Bhasin & Roy, 2019).

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The management guidelines for CHB have been proposed by the European Society for Cardiology, the American College of Cardiology, the American Heart Association, and the Heart Rhythm Society (Glikson *et al.*, 2021; Kusumoto *et al.*, 2019). In summary, patients with complete heart block not caused by physiologic or reversible causes should undergo a permanent pacemaker placement irrespective of the symptoms (class I recommendation with level C evidence). The long-term outcomes of CHB are not well documented, probably because they are usually treated with pacemakers in urgent-care settings (Knabben *et al.*, 2025). The prognosis is most likely influenced by the underlying etiology of the CHB (reversible vs. irreversible causes), the severity of the clinical presentation, and the occurrence of hemodynamic instability (this can lead to death if not promptly treated by temporary and/or permanent pacemaker placement) (Knabben *et al.*, 2025).

Case Summary

Clinical history and physical examination

A 79-year-old widow, a mother of 10 and a farmer from Mbaruk, Nakuru County, Kenya, first presented to us in 2021 with clinically decompensated biventricular congestive heart failure and severe bradycardia (heart rate of 34 bpm) on a background of poorly controlled isolated systolic hypertension, which was diagnosed some 30 years prior (baseline home BPs of 163-170/71-73 mmHg during this presentation.). She had been non-compliant on the prescribed amlodipine-losartan-hydrochlorothiazide combination tablet. She was overweight (BMI of 28.8 kg/M^2) but with no other cardiovascular risk factors. She had been diagnosed with hypertension and a "slow heart" in a peripheral facility in 1995 and was put on unknown medications and referred to a cardiologist for a possible pacemaker placement. She defaulted on the medications and did not go for her referral. She continued to have "occasional" dizziness thereafter with her heart rates ranging from 30 to 40 bpm whenever they were checked. In the year 2008, she had several syncopal episodes and was referred to the national referral hospital, where she was diagnosed with "severe hypertension" and a complete heart block. Six months before this, her husband had died, and her home and property had been destroyed in the 2007-2008 Kenyan post-election violence. Subsequent multiple cardiologist reviews and recommendations for a permanent pacemaker placement were futile as she declined "the placement of any gadgets in me that God did not put in there Himself!" She underwent serial counseling for the same, but she declined. She describes herself as a "staunch Christian with unshakable faith in God." Reportedly, the etiology of the CHB was never found despite "multiple blood tests," and it was thus labeled idiopathic. No invasive tests or procedures were done during the etiological workup. She subsequently defaulted on her discharge medications (unknown) and was not on any treatment until 2 years before her presentation to us, when she was put on an antihypertensive and referred to a cardiologist, who again failed to talk her into getting a pacemaker upon re-confirming a complete heart block. Interestingly, she reported no further pre-syncopal or syncopal episodes after 2008 except for on-off palpitations with heavy physical exertions. She had no history suggesting heart failure or acute coronary syndrome before her presentation to us.

Presently, she was in decompensated biventricular heart failure at New York Heart Association (NYHA) dyspnea scale grade 3 precipitated by a community-acquired pneumonia (CURB-65 score of 2). Her BP was 163/79 mmHg, pulse rate of 32 bpm (which did not change following a trial administration of 1.2 mg of atropine intravenously), with an oxygen saturation of 97% in room air. She was afebrile. She had grade 3 bi-pedal pitting edema, elevated JVP with the classical cannon 'a' wave, a hyperactive precordium with variable but normal S1, S2, and an S3 gallop rhythm and no murmurs, bi-basal crackles on the lung auscultation with right mid-lung zone crepitations, and a tipped tender hepatomegaly on abdominal examination. The rest of the physical examination was unremarkable.

Work-up, Management, and Follow-up

Her baseline laboratory tests were all normal, including a complete blood count, renal, thyroid, and liver panels, serum electrolytes including calcium, blood sugar, lipid profile, and a urinalysis. Serum troponins were negative. The COVID-19 antigen and PCR tests were negative. Her EKG showed a complete heart block with a ventricular escape rate of 36 bpm, some premature ventricular contractions, and no evidence of acute ischemic changes. See Figure 3 below. Her CXR showed cardiomegaly with pulmonary edema and right lung mid-lobe opacification. An interval echocardiogram showed grade 2 diastolic dysfunction, dilated left atrium and ventricle with a left ventricular ejection fraction of 87%, no regional wall motion anomalies, normal valves, no thrombus, and no pericardial effusion. Another echocardiogram done 12 months later showed a small apical thrombus.

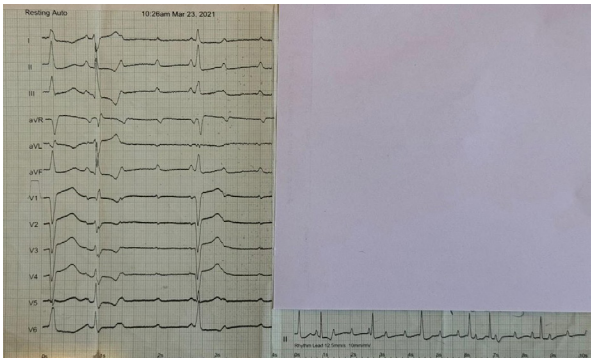

Figure 3: The EKG done in 2021 shows a complete heart block with ventricular escape rhythm; the atrial rate is about 90 bpm, while the ventricular rate is about 36 bpm. Two premature ventricular contractions are noted.

We successfully managed her as an outpatient with furosemide, spironolactone, losartan, an antibiotic (co-amoxiclav), and supportive therapy. We added rivaroxaban later on following the finding of an apical thrombus. We counseled her several times on the need for a pacemaker, but she has declined. It is now 4 years since she has been on follow-up at our clinic and 30 years since she was diagnosed with a complete heart block. She remains in complete heart block (see figure 4 below) and in compensated heart failure (NYHA 1-2, current baseline), but with no pacemaker. She has had no admissions for heart failure, no chest pains, no syncope, but occasional mild dizziness while working on her farm. She had an episode of orthostatic hypotension in 2023 following an acute diarrheal illness, which resolved with careful hydration and temporary withdrawal of the diuretics. She still does almost all activities of daily living independently.

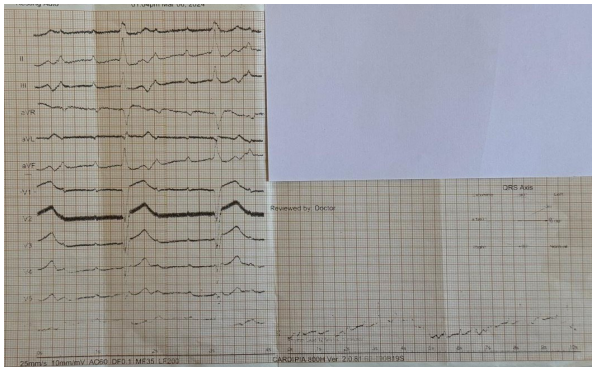

Figure 4: The EKG done in 2024 shows a complete heart block with ventricular escape rhythm; the atrial rate is about 84 bpm, while the ventricular rate is about 30 bpm.

Discussion

Patients with CHB who are managed with a permanent pacemaker have an overall good prognosis. Alpert and Katti demonstrated overall survival rates of 91% at 1 year, 63% at 5 years and 41% at 10 years (Alpert & Katti, 1982). This is influenced by patient demographics and the underlying cause and/or complications of the CHB, e.g., advanced age, congestive heart failure, ischemic heart disease, etc., which were associated with lower survival rates compared to the presence of hypertension, diabetes, or valvular heart disease in this study (Alpert & Katti, 1982).

Little information is recorded in the literature about the prognosis of patients with CHB who are not treated with a pacemaker. A ten-year retrospective study by Edhag and Swahn of 101 patients with arrhythmic syncope and CHB not treated with pacemakers (68 of them had CHB) showed that of those who had CHB, survival was 68% at 1 year and 37% at 5 years (Edhag & Swahn, 1976). There are several case reports of patients who were diagnosed with CHB and managed without a permanent pacemaker placement who led relatively active lives. For example, a 23-year-old Chinese woman diagnosed with congenital CHB at the onset of an episode of Adams-Stokes attack declined a pacemaker and led an active life during a 28-year follow-up period in which she had an uneventful pregnancy (Su *et al.*, 2022). A 26-year-old primigravida with CHB remained stable throughout the pregnancy and underwent a cesarean section under spinal anesthesia without a pacemaker (Swain *et al.*, 2022). Our patient has seemingly followed a clinical course almost similar to the Chinese woman (although the latter had an initial temporary pacemaker and later on had a relatively good compensation for her CHB from the junctional escape rhythm). Our patient was diagnosed with CHB during an Adams-Stokes attack and recommended for a permanent pacemaker placement but declined due to personal and religious reasons. She has lived for about 30 years now with CHB without a pacemaker and has been active throughout, including in her old age presently, where she is still doing most of her activities of daily living independently. She has survived several episodes of presyncope and syncopal attacks and has been in compensated heart failure with preserved ejection fraction since we started following her 4 years ago. The reasons for her relatively stable clinical course remain largely speculative for now, including the possible roles of unknown cardiovascular compensatory mechanisms and her religious faith. We are unable to do any further etiological and prognostic studies for her CHB (e.g., cardiac MRI, biopsy, etc.). Undoubtedly, more studies are needed to correctly map out the natural history of CHB and elicit more reliable and predictable prognostic markers in this subset of patients who decline permanent pacemaker placement but seemingly remain relatively stable over many years.

CONCLUSION

The case of our patient adds to a subset of known (and unknown) patients with CHB who surprisingly led a relatively stable and active clinical course over many years without any pacemaker placement. This may suggest that some patients with CHB may not necessarily require a permanent pacemaker, as is the current evidence-based recommendation by various guidelines. Accordingly, more studies are needed to map out the natural history of CHB in this subset of patients and to determine more reliable markers of prognosis to influence decisions regarding true indications and timing of pacemaker placement.

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