Refractory Hypertensive Emergency associated with Complete Heart Block Resolved after Permanent Pacemaker Implantation: A Case Report

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Abstract

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Refractory Hypertensive Emergency associated with Complete Heart Block Resolved after Permanent Pacemaker Implantation: A Case Report

Short title: Hypertensive Emergency and Complete Heart Block

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Abstract

Severe hypertension sometimes improves with treatment of bradycardia but this phenomenon is under-reported. Here an elderly gentleman with complete heart block and a hypertensive emergency was refractory to medical therapies and blood pressure only improved following pacemaker implantation. We discuss the possible mechanisms relating to heart rate and artificial pacing.

Key clinical message

When faced with a refractory hypertensive emergency and complete heart block, artificial pacing may improve blood pressure by altering diastolic filling, myocardial contractility and atrioventricular synchrony.

Introduction

Significant hypertension and bradycardia are often seen together in clinical practice. Anecdotally blood pressure (BP) often improves following treatment of bradycardia when caused by atrioventricular (AV) nodal conduction disturbance, however this is under-reported in the literature. Permanent pacemaker implantation is a class I indication for complete heart block (CHB).[1]

We report on a case of a hypertensive emergency that was refractory to medical management and was only controlled following treatment of co-existing CHB with permanent pacemaker implantation.

Case Report

An 88-year-old man was admitted to hospital on the acute medical ward with gradually worsening breathlessness and peripheral oedema. His comorbidities included myocardial infarction and coronary stenting 9 years ago, essential hypertension, hypercholesterolaemia and stage III chronic kidney disease. On initial assessment his BP was noted to be 232/92 with a heart rate (HR) of 52 bpm. There was clinical evidence of congestive cardiac failure with bilateral basal lung crepitations and pitting lower limb oedema. An ECG revealed CHB with a narrow complex escape rhythm at 46 bpm (figure 1). A chest x-ray showed mild pulmonary oedema. Admission blood tests were satisfactory including normal thyroid function and electrolytes. His regular medication included lacidipine 6 mg, candesartan 28 mg, bisoprolol 10 mg, furosemide 20 mg, aspirin 75 mg and atorvastatin 40 mg all taken once daily.

Bisoprolol was immediately stopped in view of CHB and he was referred to the cardiology team to consider permanent pacemaker implantation. An intravenous frusemide infusion of 240 mg over 24 hours was commenced. Despite a good diuresis in the first 24 hours his BP remained 202/69, therefore an intravenous glyceryl trinitrate (GTN) (50mg/50mls at 1-10mls/hr) infusion was started. BP remained 190/90 with 8 mls/hr of GTN.

Secondary causes of hypertension were investigated with normal 24-hour urinary catecholamine and serum angiotensin converting enzyme levels, and normal sized kidneys on renal ultrasound. There was heavy proteinuria with 24-hour urinary collection measuring 6.1 g. An echocardiogram showed normal left ventricular (LV) size and function, moderate concentric LV hypertrophy and mild aortic stenosis.

He achieved a 5.3 kg diuresis over 5 days. Attempts were made to wean off GTN however this only led to BP rising again to 215/89. Indapamide 2.5 mg once daily had a limited effect. Doxazosin was started and gradually increased to 4 mg twice daily. Methyldopa was initiated and uptitrated to 250 mg twice daily.

On day 7 pacemaker implantation was due to go ahead. On arrival to the cardiac catheter lab BP was 180/70 with GTN running at 10 mls/hr. GTN was discontinued shortly before the procedure anticipating that sedation and analgesia might conversely lead to acute hypotension. A dual chamber pacemaker was implanted successfully with no immediate complications. The right ventricular (RV) pacing lead was positioned at the RV apex. BP during the procedure was 169/51. With the device set for atrial sensing and ventricular pacing, HR was maintained around 90 bpm. BP immediately after pacing was 160/80 and remained controlled at this level without GTN. The post-implant ECG showed an atrial-tracked, ventricular-paced rhythm at 90 bpm (figure 2), and post-implant chest x-ray showed satisfactory lead positioning with no acute complications. Bisoprolol was restarted at 10 mg. The final documented BP prior to discharge was 100/52 with a HR of 82.

Discussion

This case interested us due to the combination of bradycardia and a hypertensive emergency that was refractory to medical treatments. BP markedly improved following treatment of CHB with permanent pacemaker implantation. A summary of BP readings during the clinical course are shown in figure 3. It

was interesting to note that the lowest diastolic BP before pacing was 51 mmHg and immediately rose after initiation of pacing to 80 mmHg. Pulse pressure before pacing was between 100-140 mmHg and after pacing fell to around 50 mmHg. We suspected this was a direct effect of increasing HR, shortening the diastolic period and limiting the fall in arterial pressure before the next cardiac cycle. We confirmed before pacing that the large pulse pressure did not have an alternative cause such as aortic regurgitation.

Systolic BP also decreased by 20 mmHg following device implantation without the need to restart the GTN infusion and even once the effects of sedation had worn off. A combination of factors may have played a part however the contrast was striking. Whilst it proved impossible to wean off GTN prior to pacing, it was no longer required at all afterwards. The reduction in diastolic filling time could have reduced ventricular preload and stroke volume (SV) to some extent, but it is difficult to say how much this contributed to the reduction of systolic BP.

BP is a function of cardiac output (CO) calculated by the formula CO = HR x SV. A lower HR should therefore reduce CO, but this is mitigated by a rise in SV due to increased diastolic filling time and ventricular preload. Historically, it has been described that as HR slows, pulse pressure widens i.e. diastolic BP drops and systolic BP increases.[2,3] The haemodynamic effects of artificial pacing in CHB have also been described, suggesting that increasing HR lowers SV.[4] Conversely, SV has been shown to increase on induction of CHB in the animal model.[5]

RV pacing may directly affect SV by induction of dyssynchronous myocardial contraction. Prior to pacing in this case, the escape rhythm was junctional in nature, hence LV activation will have been via the normal His-Purkinje system. Change in the pattern of regional myocardial strain by pacing has been demonstrated in dogs by MRI imaging. SV was significantly reduced by RV apical pacing due to dyssynchronous LV myocardial contraction.[6] Another study looking at lengthening AV delay during pacing showed an immediate increase in both SV and BP. However, BP quickly declined after a few seconds whilst SV was maintained, suggesting compensatory peripheral vasodilatation.[7] Artificial pacing algorithms to shorten AV delay have been proposed as a treatment for hypertension, however this has so far remained restricted to clinical studies.[8,9]

BP is also dependent on systemic vascular resistance (SVR) under autonomic control via baroreceptors. Cardiogenic shock induces a sympathetic response to compensate with peripheral vasoconstriction and increases SVR acutely.[10] Chronically raised SVR may have played a role in this case with evidence of longstanding hypertension, echocardiography revealing moderate concentric LV hypertrophy and there was heavy proteinuria. SVR is also known to increase with advanced age associated with atherosclerosis.[11,12] Higher SVR may have exaggerated the BP response to increased SV.

We noted only one previous case report of bradycardia and malignant hypertension. A case of a 65-year-old man presenting with left upper limb weakness, bilateral foot paraesthesia and headache with a BP of 240/90 and an ECG showing CHB at 39 bpm. Brain imaging was normal, and symptoms resolved after BP control with intravenous GTN. After permanent pacing BP improved to 140/80. The authors attributed better BP control to reduced diastolic filling time and SV.[13]

In hindsight, pacemaker implantation was unduly delayed due to a combination of inter-speciality logistics and lab availability. Greater efforts to enable earlier pacemaker implantation may have led to sooner control of BP and shorter hospital stay, and this is a learning point. We could also make mention of alternative pacing sites such as RV outflow tract, RV septum or His bundle pacing which may have preserved more physiological ventricular contraction, and although the haemodynamic effects have been studied with relation to intracardiac function, the effect in this context on systemic BP needs further study.[14]

Conclusion

Severe hypertension often improves following treatment of bradycardia but this phenomenon is underreported. In this case, bradycardia as a result of CHB occurred in the context of a hypertensive emergency refractory to multiple medical therapies. BP was only controlled following permanent pacing with an imme-

diate rise in diastolic BP likely related to a faster HR. A reduction in diastolic filling time and SV may have contributed to better control of systolic BP, although other factors such as pacing-induced dyssynchrony and SVR could have also played a role. Pacing may therefore benefit patients early in their clinical course.

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Figure legends

Figure 1. ECG before pacemaker implantation showing complete atrioventricular dissociation with a narrow complex escape rhythm at 42 bpm.

Figure 2. ECG following pacemaker implantation showing atrial-tracked, ventricular-paced rhythm at 90 bpm.

Figure 3. Blood pressure recordings taken during the clinical course influenced by events during this episode.