CASE REPORT

Occurrence of complete heart block associated with acute renal infarction

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Abstract

Complete heart block (CHB) and acute renal infarction (ARI) are both uncommon diseases and seldom encountered in the clinical practice. We describe a rare case of pre-existing left bundle branch block, presenting simultaneously with CHB and ARI. The possible mechanism depends on prior presence of either CHB or ARI. If ARI occurs first, severe pain and embolism may enhance the vagal tone resulting in decrease in the heart rate and transient intraventricular conduction interruption, which subsequently causes CHB. The opposite scenario, CHB preceding ARI, is also possible. CHB can be physiologic and transient, with higher risk of development in the circumstance of pre-existing conduction system disturbances. Patients with CHB are predisposed to formation of thrombi and thromboemboli, giving rise to ARI. In conclusion, awareness and timely identification of the clinical manifestations of these two diseases may facilitate early diagnosis and prompt management.

KEYWORDS

atrioventricular conduction block, bundle branch block, complete heart block, renal infarction

1 | INTRODUCTION

Complete heart block (CHB) is an uncommon disease with a low prevalence of approximately 0.04% in the general population (Kojic, Hardarson, Sigfusson, & Sigvaldason, 1999). It is a type of arrhythmia, indicating that no impulses from the atria reach the ventricle, in the electrical system of the heart. In this circumstance, specific areas in the ventricles may create electrical signals to cause ventricular contraction. The rate is slower and not coordinated with atrial contraction. The transmission disturbance can be transient or permanent (NHLBI, 2019).

Acute renal infarction (ARI) is a rare disorder, and many other diseases mimic the symptoms of ARI, such as abdominal or flank pain. Therefore, it may be easily misdiagnosed or missed, and the previously reported incidence of 0.004%–0.007% in the emergency department may be underestimated (Kwon, Oh, Ha, Kim, & Do, 2016). However, delayed diagnosis of ARI may lead to renal complications, including acute kidney injury and chronic kidney disease. Owing to the widespread availability of computed tomography (CT) scan, early identification and timely management of ARI is possible.

Herein, we report a case showing simultaneous presence of CHB and ARI. It is extremely rare to encounter a patient with a combination of these two diseases. There must be some special relevance to contribute to this condition. The present case suffered from pre-existing left bundle branch block (LBBB), which may have been one of the underlying factors. We will discuss the possible mechanisms in depth.

2 | CASE REPORT

A 68-year-old man was admitted to an academic medical center because of acute left flank pain and worsening dyspnea.

He had a history of hypertensive cardiovascular disease (HCVD) for about 15 years and developed LBBB at the age of 65 years. The 12-lead electrocardiogram (ECG) taken in the outpatient department 2 years ago is shown in Figure 1a. Two days before admission, he presented with sudden onset of generalized abdominal pain, which subsequently localized to the left side flank area. It was accompanied by nausea and vomiting. In addition, fatigue and progressively worsening dyspnea were noted about the

(a)

Speed: 25 mm/s, Limb: 10 mm/mV, Chest: 10 mm/mV, Frequency band: 0.05–150 Hz

(b)

Speed: 25 mm/s, Limb: 10 mm/mV, Chest: 10 mm/mV, Frequency band: 0.05–150 Hz

FIGURE 1 The 12-lead electrocardiogram shows (a) left bundle branch block at outpatient department and (b) complete heart block at emergency department

(a)

(b)

FIGURE 2 Abdominal computed tomography in (a) pre-contrast-enhanced phase and (b) post-contrast-enhanced phase shows wedge-shaped perfusion defects in the left kidney (white arrow)

same time. He sought medical attention in the emergency department of this hospital.

On physical examination, bradycardia (37 beats per minute) was noted with a blood pressure reading of 146/82 mm Hg. The 12-lead ECG showed a CHB (Figure 1b). Laboratory evaluation revealed a mild increase in the white blood cell count to 11,050/ μ l (83.2% neutrophils), normal levels of hemoglobin, platelet count, alanine aminotransferase

(ALT) (42 U/L; normal 10–49 U/L), serum creatinine level (1.31 mg/dl; normal 0.7–1.3 mg/dl), and elevated serum lactate dehydrogenase (LDH) level (1,023 U/L; normal 98–192 U/L). No electrolyte imbalance was detected. The coagulation profiles, including prothrombin time and activated partial thromboplastin time, were normal. Urinalysis by urine dipstick method showed no hematuria and microalbuminuria (value 2+, 100 mg/dl).

Abdominal contrast-enhanced CT revealed wedge-shaped perfusion defects in the left kidney, compatible with renal infarction (Figure 2). Subsequently, transthoracic and transesophageal echocardiography showed normal-sized left atrium (LA) and concentric left ventricular (LV) hypertrophy, preserved LV systolic function with ejection fraction of 62% and LV diastolic dysfunction, and no evidence of valvular disease; however, no definite LA thrombus was identified.

The patient was admitted to the hospital, and low-molecular-weight heparin was administered intravenously for anticoagulation. Because he was hemodynamically stable, emergent therapy with atropine or temporary cardiac pacing was not considered necessary. Under continuous monitoring and conservative treatment, CHB recovered spontaneously without any intervention. Finally, his symptoms improved significantly, and he was discharged in good condition with oral anticoagulation therapy using warfarin.

3 | DISCUSSION

Both CHB and ARI are rare diseases and scarcely reported in the medical literature. Although not occurring very often, they are illness which physicians need to be alert and highly suspicious of, especially when the patient has risk factors. We demonstrated a case visiting the emergency room with non-specific chief complaints, and being diagnosed with simultaneous occurrence of these two uncommon diseases. In addition, the patient had a pre-existing conduction system disease involving the left bundle branch. Hence, careful investigation of the underlying mechanism is valuable and significant.

The clinical manifestations of CHB, resulting from reduced cardiac output and slower ventricular rate, are quite variable and include fatigue, dyspnea, chest pain, syncope, and even sudden cardiac arrest (NHLBI, 2019). Atrioventricular (AV) block is caused by physiologic, pathophysiologic, and iatrogenic factors (Sauer, 2018). Physiologic slowing of cardiac conduction in response to enhanced vagal tone, owing to sleep, pain, athletic training, or carotid sinus massage, can result in significant decrease in AV conduction disturbances (Page, Tang, & Prystowsky, 1991; Sauer, 2018). As to the pathophysiologic etiology, conduction system fibrosis and/or sclerosis, which appear idiopathic, occur in about one-half of the cases. It may be associated with other cardiac diseases, like ischemic heart disease and myocarditis, or iatrogenic reasons, such as drugs (Sauer, 2018). LBBB and right bundle branch block (RBBB) occur when the normal electrical activity in the His–Purkinje system is interrupted (NHLBI, 2019). The simultaneous occurrence of LBBB and RBBB leads to CHB.

Patients with ARI typically complain of initial sudden onset of flank pain or generalized abdominal pain, accompanied by nausea or vomiting (Radhakrishnan, Palevsky, & Sheridan, 2017). Laboratory manifestations usually reveal elevated serum LDH (often more than two to four times the upper limit of normal) with little or no increase in serum ALT. This pattern of enzyme elevation is strongly indicative of ARI and can also be seen in other easily distinguishable conditions, such as late myocardial infarction,

hemolysis, and renal transplant rejection. Non-specific findings include mild increase in white blood cell count, increase in serum C-reactive protein, and normal or elevated serum creatinine levels (Radhakrishnan et al., 2017). Urinalysis may reveal hematuria (32%) and proteinuria (12%). The etiology of renal infarction may be classified as cardioembolic disease, renal artery injury, hypercoagulable state, and idiopathic. Among them, cardiogenic causes are the most important (55.7%) followed by idiopathic causes (30.1%) (Oh et al., 2016). Cardiogenic ARI develops from thromboemboli formed in the heart or aorta. Although in situ thromboses may also cause complete occlusion of the artery in the kidney, they are typically nondistensible, irregularly shaped, and smaller in size (Radhakrishnan et al., 2017). Cardiogenic thromboemboli are more likely to be the source of ARI in this case, because of his HCVD and dysrhythmia history.

Several aspects need to be addressed for this rare condition. First, the association between transient CHB and ARI may be causal or coincidental. As this patient had pre-existing LBBB, we speculated that it may play a certain role in the relationship. The underlying mechanism causing this situation would depend on prior presence of either CHB or ARI.

In the presence of ARI, embolism may trigger a hyperadrenergic state, resulting in stimulation of the Bezold–Jarisch reflex causing an increase in the efferent vagal response and decrease in sympathetic tone, leading to a decrease in the heart rate and transient interruption in the intraventricular conduction, including His bundle, LBBB, or RBBB (Elias et al., 2004). For example, RBBB is one of the abnormal ECG findings in acute pulmonary embolism (Boey, Teo, & Poh, 2015). CHB may subsequently develop in the setting of pre-existing conduction system disturbances.

The possibility of CHB preceding ARI cannot be ruled out. This patient had LBBB, which indicated that he was predisposed to high degree AV block. The conduction disorder can be physiologic and transient, and secondary to the abovementioned etiology (NHLBI, 2019; Sauer, 2018). According to Virchow's triad, extreme bradycardia due to CHB, and the resultant increased circulatory time with stasis of blood flow, provoke the formation of thrombi and thromboemboli, giving rise to ARI (Wolberg, Aleman, Leiderman, & Machlus, 2012).

4 | CONCLUSION

Clinicians should be mindful of the occurrence of CHB in association with ARI, especially when dealing with the patients with previous heart conduction disorders, such as LBBB. Owing to the rarity and non-specific symptoms, delay in the diagnosis of ARI may occur. Knowledge about the clinical features and laboratory manifestations of CHB and ARI can greatly assist in establishing immediate diagnosis and initiating adequate intervention.

CONFLICTS OF INTEREST

None.

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