Asymptomatic Permanent Left Bundle Branch Block (LBBB) complicating Diagnostic Left Heart Catheterisation

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ABSTRACT: This case report describes a routine diagnostic left heart catheterisation (coronary angiography, aortography and left ventriculography) procedure at Sultan Qaboos University Hospital, Oman, which was complicated by the development of new asymptomatic, but permanent, left bundle branch block that was observed incidentally towards the end of the procedure. The patient was completely asymptomatic and haemodynamically stable throughout the procedure and afterwards. Urgent investigations, immediately after the procedure, including routine blood, serial cardiac troponin I, serial electrocardiograms, chest X-ray, and urgent echocardiography were normal and failed to show any possible causation of the LBBB. The results of left heart catheterisation showed two vessel coronary artery disease and severe mitral valve regurgitation. After eight days, the patient went on to have coronary artery bypass surgery and mitral valve replacement surgery both of which were successful. To the best of our knowledge, this is the first case report to describe the occurrence of permanent LBBB after left heart catheterisation. This report describes the case and reviews the literature for the incidence and implications of such a complication.

Keywords: LBBB; Left bundle branch block; Coronary angiography; aortography; Left ventriculography; Left heart catheterisation; Diagnostic heart catheterisation; Case Report; Oman

In general, diagnostic cardiac catheterisation is recommended whenever it is clinically important to define the presence or severity of a suspected cardiac lesion that cannot be adequately evaluated by other non-invasive techniques. Most of the indications for cardiac catheterisations can be collectively classified into management of patients under the following categories: valvular heart disease; chronic heart failure; acute myocardial infarction (AMI), percutaneous coronary intervention (PCI), and coronary artery bypass surgery (CABG). Cardiac catheterisation is a relatively safe procedure, but has a well defined risk of morbidity and mortality. In an analysis of 59,792 patients who underwent cardiac catheterisation and coronary angiography, the following were recognised complications (percentage of risk in brackets): mortality (0.11%), myocardial infarction (0.05%), cerebrovascular accidents (0.07%), arrhythmias (0.38%), vascular accidents (0.07%), arrhythmias (0.38%), vascular
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complications are more common with right heart catheterisation, patients with left main stem (LMS) coronary artery disease (CAD), patients with low ejection fraction (EF) < 30%, and patients with high New York Heart Association (NYHA) class IV. This report describes the case and reviews the literature on the incidence, significance and the implication of this complication during cardiac catheterisation procedures.

Case Report

A 67 year-old man, on regular treatment for hypertension (HTN) for the previous six years, presented at Sultan Qaboos University Hospital, Oman. He had had a history of shortness of breath (SOB) on exertion for the previous few years, but this had recently worsened. The echocardiogram showed a mitral valve prolapse (MVP) with severe mitral regurgitation. He was referred for work-up for mitral valve surgery. He was on the following medications: aspirin 81 mg once a day, ranitidine 150 mg twice a day, bisoprolol 2.5 mg once a day, and simvastatin 20 mg at night.

The patient had an appearance of good health. Heart auscultation revealed a normal first sound, a faint second sound and a pansystolic murmur at the apical region radiating to the axilla. The lungs were clear and the rest of the examination was unremarkable. Initial investigations, including total blood count, urea and electrolytes, coagulation profile, lipids, electrocardiogram (ECG), routine chest X-ray, hepatitis B screening and human immunodeficiency virus (HIV) screening were all normal. Figure 1 shows the baseline ECG of this patient on admission. It shows a normal sinus rhythm, left atrial abnormalities, Rsr' pattern in frontal leads, and a QT interval that was at the upper limit of normal.

The left heart catheterisation procedure was explained and consent was obtained. The procedure was done through the right femoral artery using the Seldinger technique. Coronary angiography was done using 6F, JL4 and JR4 diagnostic catheters. This required multiple small injections of contrast (5–8ml) by hand at high speed and acquisition of coronary images in different projections.

An aortogram was done using a pigtail catheter. Thirty-five millilitres of contrast were injected at a rate of 15 ml per second using a power injector. Left ventriculography was done after crossing the aortic valve using a J-shaped guide wire followed by the pigtail catheter. Twenty-five millilitres were injected at 10 ml per second using a power injector. These investigations revealed the following results: the coronary angiography showed two vessels with CAD disease; the first diagonal revealing 75% long stenosis proximally and 75% stenosis distally. The posterior descending artery had 75% stenosis proximally. The rest of the vessels were normal. Ventriculography showed good left ventricular (LV) systolic function, severe mitral regurgitation (MR) grade IV, mildly dilated LV and grossly dilated left atrium. There was no significant aortic pressure gradient across the aortic valve. The aortogram showed no aortic root dilatation and mild grade I aortic regurgitation. Overall the patient had severe MR and two vessels with CAD disease.

Towards the end of the procedure, it was noticed incidentally on the monitored ECG that the sinus rhythm had changed to an LBBB pattern. The patient heart rate was 75 beats per minutes and was completely asymptomatic and haemodynamically stable, and the catheterisation procedure had no
complications. After sheath removal, the patient was moved back to the ward. The patient had no chest pain or SOB. His vital signs were all normal. The physical examination was also entirely normal. A repeat ECG showed a widening of the QRS complex typical for an LBBB pattern [Figure 2]. A chest X-ray was similar to the previous one with a normal appearance of pulmonary vasculature, no pleural effusion, and no signs of lung oedema or consolidation.

An urgent bed-side echocardiogram revealed: 1) LV: upper limit of normal internal dimensions, no regional wall motion abnormality, excellent systolic function, and a normal relaxation pattern, and an intact septum; 2) mitral valve: thickened, fibrosed and calcified with a significant prolapse of the posterior leaflet with severe MR; 3) aortic valve: mild aortic stenosis (AS) with trivial aortic regurgitation (AR); 4) tricuspid valve: normal with trivial tricuspid regurgitation (TR) with estimated pulmonary artery pressure of 20 mm Hg; 5) thickened pericardium with no effusion; 6) no intra-cardiac masses or thrombi noted. Serum cardiac troponin I (CTnI) was 0.03 µg/L four hours after the procedure and, when repeated next day, was again 0.03 µg/L (normal ≤ 0.03 µg/L). As the patient was asymptomatic and clinically and haemodynamically stable, the investigations did not reveal any specific cause for this new LBBB (normal troponins and echocardiographic findings); he was therefore treated conservatively. He was allowed to proceed with surgery during the index admission. A CABG (one vein graft) with mitral valve replacement (MVR) (tissue bioprosthetic valve) was carried out successfully 8 days after the cardiac catherisation. The postoperative course was only complicated by transient atrial fibrillation (that eventually reverted to sinus rhythm after a short course of amiodarone and an on-demand pacemaker), and abdominal pain (a computed tomography (CT) scan of the abdomen showed no major pathologies). The patient was discharged home in a stable condition 12 days after surgery. The pre-discharge ECG still showed a LBBB.

Discussion

The appearance of transient LBBB in the setting of cardiac catheterisation (and specifically during left ventriculography) is a well known, although very uncommon complication. In the pivotal article of Bourassa M and Noble J, in which 5.250 coronary arteriographies were reviewed, the incidence of LBBB was 0.17%. However, LBBB in all the above cases subsided either spontaneously or responded rapidly to appropriate drug therapy. An iatrogenic left bundle branch block is a rare complication of left ventricular catheterisation and coronary arteriography because unlike the branches of the right bundle, which pass superficially within the thin-walled right ventricle, the left bundle is a fan-like structure that radiates from the left interventricular septum through the thicker-walled left ventricle. This structure results in relatively greater protection of the left bundles from focal mechanical injury. In contrast to catheters frequently used in the right ventricle, left heart catheters such as “pigtails” are usually spatially located in the left ventricle so that the interventricular septum is spared mechanical contact. We have reviewed the available literature, but did not find anything on this topic. A permanent LBBB complication as a result of diagnostic cardiac catheterisation has, to the best of our knowledge, not been reported before. Our case was admitted for a routine procedure. The patient was not classified into high risk category for the development of arrythmias. These categories include patients with left main stem (LMS) lesions, heart failure and recent acute coronary syndrome (ACS) events. He had no renal or liver dysfunction, and the basic electrolytes (potassium, calcium, and magnesium) were normal. None of the medications he received were known to be associated with the development of this complication.

The guide wire used was J wire, which means it has a J-shaped soft curve at the terminal end. This protects against left ventricular wall trauma or puncture. This type of guide wire rarely causes trauma to the aorta or LV wall [Figure 3A]. Other guide wires for example straight-tip guide wires which are sometimes used (but not in this case) to cross heavily calcified stenotic aortic valves, may sometimes cause LV wall trauma or perforation or LMS dissection if they are manipulated aggressively. Performing a left ventriculogram or an aortogram requires the injection of a large volume of contrast solution using a powered injector at high speed into the LV and aorta respectively through a pigtail catheter that has a hole at one end and many small side holes to allow the contrast to leak out [Figure...
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3B). The end of the catheter is placed near (but not in contact with) the apex and away from the free wall. This is very important to avoid the powered injection jet being injected directly into the wall of the LV. Inadvertent injection of contrast solution into the free wall of the LV under high pressure may lead to free wall rupture or perforation with subsequent cardiac tamponade or free septal wall rupture with free left to right shunt. The presence of many side holes in the pigtail catheter is a very important safeguard to reduce the force of the jet by allowing the contrast to leak side ways if the end hole were inadvertently placed in direct contact with the wall.

This LBBB complication appeared during heart catheterisation suggesting a direct contribution of the latter to its development. The exact cause(s) of the LBBB in this case is not known, but could be due to one of the following several possible mechanisms: 1) during coronary angiography or aortography, it is possible that a small amount of foreign body material (air, thrombus, coronary plaque, cholesterol or calcium) may have embolised distally and blocked the blood supply to the left bundle branch or some of its fibres; 2) it is possible that manipulation of either the guide wire or the pig-tail might have produced a minimal, localised, septal contusion (without any troponin elevation or pathologic findings in the echo study) leading to LBBB onset; 3) the powered injection of contrast medium into the left ventricle may have caused trauma to the septum or left ventricle and the left bundle branch or one of its distal branches. The overall findings suggests that the trauma to the heart (if any) may have been non-mechanical (as per the echocardiogram) and most probably was very slight, because there was no subsequent significant cTnl rise in the serum.

LBBB is rare in normal individuals and is most commonly seen in patients with CAD; however, as many as 12% of patients with LBBB have no demonstrable heart disease.\(^\text{10}\) Even among patients without overt heart diseases, LBBB is associated with a higher than normal risks of cardiovascular events and all cause mortality.\(^\text{11}\) It is associated with high grade atroioventricular (AV) block and sudden cardiac death.\(^\text{12}\) The abnormal ventricular activation pattern of LBBB induces an abnormal systolic function with reduced ejection fraction and stoke volumes and abnormal diastolic function.\(^\text{13}\) It carries a more serious prognosis than right bundle branch block, but neither form requires specific treatment.

Conclusion

This case report describes the development of a de novo permanent LBBB after a left heart catheterisation procedure. We have reviewed the literature and, to the best of our knowledge, this has not been described before. We have offerred several possible hypotheses to explain the association between left heart catheterisation and the development of LBBB. It is difficult to predict the long-term prognosis of such a complication in this patient. In general, LBBB, whatever the cause, has a poor prognosis and carries significant morbidity and mortality risks. It remains to be seen whether these risks also apply to the specific case described in this report.

Acknowledgments

We would like to thank the patient and all the nursing staff in the Catheterisation Laboratory and in Ward Two Purple of Sultan Qaboos University Hospital, Oman, who were involved in the management of this case.

References


