Spontaneous recovery of Right Bundle Branch Block (RBBB) in Acute Anterior wall Myocardial Infarction with Bifascicular Block

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A case of Acute Anterior Wall Myocardial Infarction with Right Bundle Branch Block (RBBB) and Left Anterior Hemi Block (LAHB) - Bifascicular Block had been observed to have undergone spontaneous recovery of RBBB and the plan for transvenous temporary pacemaker insertion was withheld.

Keywords: Right Bundle Branch Block (RBBB), Anterior wall Myocardial Infarction, Acute Myocardial infarction, Bifascicular Block, Left Anterior Hemi Block (LAHB)

BIFASCULAR BLOCK: LITERATURE REVIEW

Bundle Branch Block occurs in eight to thirteen percent of patients with Acute Myocardial Infarction (AMI) and is mainly associated with Anterior Wall AMI. Norris et al. found RBBB in seven percent of patients and Left Bundle Branch receiving single blood supply from a branch of Left Anterior Descending Artery, which is a slender discrete structure with a long subendocardial situation, liable to more frequent involvement in contrast to the Left Bundle Branch receiving dual blood supply with short and diffuse course.

In Anterior Wall AMI, RBBB occurs in ten percent of patients and Complete Heart Block (CHB) in five percent of patients. About one in three patients with Acute RBBB in Anterior Wall AMI develops CHB. Most of the patients with CHB after Anterior Wall AMI give history of Stokes Adams’ attack and therefore pacing is necessary. Unfortunately the degree of myocardial damage sustained in patients with anterior wall AMI with CHB is severe and in spite of transvenous pacing, hospital mortality is sixty to seventy percent and fifty percent of survivors die within next year.

Bifascicular Block either in the form of RBBB and LAHB or RBBB and LPHB (Left Posterior Hemi Block) occurs in eight to thirteen percent of patients with AMI. LAHB and RBBB is more common combination occurring in three to four percent of patients with AMI as both of these structures are supplied by Left Anterior Descending artery. As an isolated finding LAHB alone does not increase hospital mortality. When found with RBBB it is called Bifascicular block and in such condition chance of going into CHB is high and hospital mortality increases. In all patients

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with Anterior Wall AMI with new RBBB, second degree heart block, CHB, Trifascicular Block (3 fascicular block with prolonged PR interval on admission or Bifascicular block with prolonged PR interval on admission or Bifascicular block developing acutely. Temporary admission or Bifascicular block developing acutely, temporary pacing is indicated. Pacing in new RBBB with AMI has been recommended in many cardiac centres. It has been found that AMI with RBBB has high chance of going into CHB mainly in anterior wall AMI and in such cases prophylactic pacing reduces mortality. All patients with Anterior Wall AMI with CHB should undergo permanent pacing whether CHB resolves or not. This has significantly reduced sudden death after hospital discharge.

In a study of one hundred cases of AMI by Dr. A. Sayami, thirteen percent patients with AMI had RBBB (four RBBB on admission, five RBBB on the first day later to admission and four RBBB appeared on the second day). There are no significant differences of RBBB in inferior wall and 6 percent with anterior wall AMI and three percent with inferior wall AMI. Nine percent of patients developed CHB (six percent with anterior wall AMI and three percent with inferior wall AMI). Out of the six percent of patients with anterior wall AMI among them who had CHB, three percent had bifascicular block (two with RBBB and LAHB and one with RBBB and LPHB), two percent had a preceding block and one percent had second degree atrioventricular block Mobitz type 2. Out of two patients with RBBB and LAHB going into CHB, one reverted to sinus rhythm after injection of Atropine but one patient with RBBB and LAHB as well as one with RBBB and LPHB expired later on.

**CASE REPORT**

A muslim businessman of forty years of age was admitted in the coronary care unit with complaints of severe burning type of substernal chest pain for twenty eight hours duration associated with mild sweating and vomiting. He was a heavy smoker smoking twenty cigarettes per day for the last twenty years as well as a heavy alcoholic.

On examination he was restless in agony with pulse rate of 120 per minute, regular with arterial wall thickening. He was febrile with temperature of 102 F. Pallor, icterus, clubbing, cyanosis, oedema, lymphadenopathy were absent. Cardiovascular examination revealed tachycardia with normal first and second heart sounds without third heart sound or murmur or pericardial rub. Respiratory system examination revealed normal vesicular breath sound without nasal crinkles. Abdomen and nervous system examination were unremarkable.

The laboratory investigation revealed Haemoglobin 16 gm%, Total leucocyte count 12,700/cmm with 72% polymorphs, 22% lymphocytes and 6% eosinophils, random blood sugar 5 mmol/l, urea 3.8mmol/l CPK 453 (15-125) IU/l, SGOT 136 (5-40)IU/l, LDH 804 (80-320) IU/l and after twenty four hours CPK 534 IU/l, SGOT 144 IU/l and LDH 805 IU/l.

**Photo No.1** First electrocardiograph (ECG) recorded in Emergency department, showing Left Axis Deviation, tachycardia (QRS rate 120/min), regular rhythm with deep Q waves in V1, V2, V3 and ST elevation in V2, V3, V4, V5 and V6.

**Photo No.2** ECG recorded on next day showing RBBB and Abnormal Left Axis Deviation, QRS rate 61/min, regular rhythm, deep Q waves in V1, V2, V3 and ST elevation in V2, V3 reciprocal ST depression in lead II, III, aVF, V5, V6 and inverted T waves are upright. Decreased heart rate may be due to narcotic analgesic, sedation and Atenolol (20mg two doses).

**Photo No.3** ECG recorded six hours after starting intravenous GTN (Glyceryl trinitrate) at the dose of 250ug/min showing no significant change. RBBB and Left Axis Deviation still persisting. Deep Q waves in V1, V2, V3 with ST elevation in V2, V3 and reciprocal ST depression in lead II, III, aVF and V5, V6 still persisting.
Photo No. 4 ECG recorded after forty eight hours of intravenous infusion of GTN showing inverted T wave in V1 has become upright. No other significant changes. RBBB and left axis deviation still persisting.

Photo No. 5 ECG recorded on eighth day of admission and ninth day of AMI showing recovery of RBBB. Left axis deviation is still present. Q waves in V1, V2, V3 and V4 and inverted T waves in V5, V6, II, III, aVF.

Photo No. 6 ECG recorded on fourteenth day of admission showing absence of RBBB, Q waves and ST elevation in V1, V2, V3, V4 and absence of inverted T waves in V4, V5, V6 and II, III, aVF.

DISCUSSION

Patient attended TU Teaching Hospital Emergency department twenty eight hours after the onset of severe chest pain. Initially the patient had features of Acute Myocardial infarction (Antero-septal infarction) with abnormal left axis deviation and tachycardia. The next morning (forty hours of AMI), RBBB was noticed in ECG. His QRS rate was 107/min (tachycardia) despite adequate dose of narcotic analgesic, sedation overnight and Atenolol (two doses of 25mg) which was given due to tachycardia on the day of admission and next morning. There was no bifascicular block on the day of admission. Although Atenolol can increase the degree of atrio-ventricular block it has not been reported to induce RBBB. The RBBB did not disappear even after stopping Atenolol for seven days in this patient.

As the patient was continuously complaining severe chest pain so Glyceryl trinitrate GTN infusion was started on the second day of admission and continued for forty eight hours but no apparent ECG changes including RBBB was noticed. He did not develop complete heart block during his fourteen days hospital stay and his heart rate was always in upper limit (around ninety beats per minute) and initially had tachycardia mostly likely due to severe chest pain of AMI. On the eighth day of admission, Spontaneous recovery of RBBB took place. This event was preceded by change of inverted T wave in V1 into upright T wave. This may be the sign of recovering RBBB. It was noticed two days before recovery of RBBB. On the fourteenth day of admission, RBBB was absent but inverted T wave in V5, V6 II, III, aVF were still present despite oral Isosorbide mononitrate 20mg TDS dosage.

Though new RBBB in anterior wall AMI is considered to be a warning sign of ensuing CHB and prophylactic pacing is said to be indicated, Spontaneous recovery is possible. Even if CHB appears, it may revert back to sinus rhythm simply with injection of Atracurium. Hence careful observation for appearance of CHB may be enough in hospitals without pacing facility and referral centres with pacing facility being quite far away. It should be remembered that very high mortality rate (sixty to seventy percent) in Anterior Wall Myocardial Infarction with CHB can occur even in patients who are paced. Patient may be referred to nearest possible referral centre with Isoprenaline drip as an alternative.

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