CASE REPORT

Transient disappearance of left bundle branch block pattern: an unusual ECG presentation of acute pulmonary embolism

S M Athar, B S P Chin, E J Flint

Postgrad Med J 2002;78:555-558

A 61 year old man developed acute pulmonary embolism while in hospital. His previous and admission electrocardiograms (ECGs) showed a typical left bundle branch block (LBBB) pattern. Immediately after the onset of acute pulmonary embolism, LBBB disappeared from his body surface ECG with sinus bradycardia, normalisation of QRS duration, prolonged QT interval, and marked T abnormalities to the right precordial leads. Recovery from pulmonary embolism resulted in reappearance of his left bundle branch pattern. Delayed conduction of the previously unaffected right bundle branch resulting in roughly equivalent onset of ventricular activation is the most likely reason. Rate dependent LBBB is also discussed.

left bundle branch block (LBBB) on an electrocardiogram (ECG) that demonstrated pseudonormalisation of LBBB pattern after acute pulmonary embolism. This unusual ECG presentation is probably secondary to delayed conduction along the right bundle resulting in synchronous ventricular activation onset.

CASE REPORT

A 61 year old man presented initially with one week's history of acute swelling and tenderness in his right calf. This was shown by ultrasound of his right leg veins to be due to a deep vein thrombosis involving the superficial femoral veins. He was bedridden with cerebral glioblastoma multiforme and had received appropriate chemotherapy and radiotherapy. He was on phenytoin and dexamethasone. There was no history of cardiovascular or respiratory disease and he was not on any other regular medications. Blood pressure was 131/77 mm Hg, with a pulse rate of 90 beats/min. His ECG on admission (fig 1) showed LBBB with ventricular rate of 91 beats/min, PR interval 158 ms, QRS duration 142 ms, QT/QTc 430/529 ms, R-T axes 35-24-106. This was comparable to his previous ECG

Lesions in the right bundle or in the left common bundle or its subdivisions, that prevent normal electrical conduction, give rise to recognisable electrocardiographic patterns. These patterns are usually permanent and the result of complete anatomic disruption.^{1,2} Reversible intermittent electrocardiographic patterns indistinguishable from those caused by complete interruption of that bundle may arise from changes in heart rate and other factors that influence intraventricular conduction velocity.³⁻⁷ We report a patient with longstanding

Abbreviations: ECG, electrocardiogram, LBBB, left bundle branch block

Vent. rate	91 bpm	Normal sinus rhythm with occasional premature ectopic
PR interval	158 ms	complexes
QRS duration	142 ms	Left bundle branch block
QT/QTc	430/529 ms	
P-R-T axes	35 -24 106	

Referred by: Unconfirmed

Figure 1 ECG on day of admission.

Vent. rate 57 bpm
PR interval 156 ms
QRS duration 82 ms
QT/QTc 540/526 ms
P-R-T axes 39 -11 73

Sinus bradycardia
ST and marked T wave abnormality, consider anterior ischemia
Prolonged QT
Abnormal ECG

Referred by: Unconfirmed

Figure 2 ECG on day of clinical episode of chest pain.

Figure 3 V:Q lung scan.

done six months previously showing LBBB pattern, ventricular rate 87 beats/min, PR interval 160 ms, QRS 144 ms (not illustrated). He was started on tinzaparin, a low molecular weight heparin, for treatment of the deep vein thrombosis.

Four days after admission, he developed acute pleuritic lower chest pains and dyspnoea. On examination he was tachypnoeic (respiratory rate 50 breaths/min), hypoxic (pulse oximetry reading <90%), drowsy with blood pressure of 95/65 mm Hg, and pulse rate 53 beats/min. An ECG at this time (fig 2) showed sinus bradycardia (rate 57 beats/min), normal QRS duration (82 ms), but prolonged QT interval (QT/QTc 540/526 ms). The PR interval was 156 ms. In addition, there was marked ST/T abnormalities with T inversions to chest leads V1 to V4. A repeat ECG after one hour showed the ST/T abnormalities had not changed or evolved further.

A working diagnosis of pulmonary embolism was made. This was later supported by positive findings on V:Q lung scanning of an area of mismatched perfusion and ventilation in the posterior segment of left upper lobe (fig 3). The nature of the chest pain was not typical of cardiac ischaemia. Furthermore, plasma levels of creatine kinase MB isoenzyme measured at five hours and 15 hours from the onset of pain were not raised (8 IU/l and 1 IU/l respectively; normal range 0–18 IU/l). NB: Serum troponin measurement were not available locally at the time this presentation.

The patient continued with anticoagulant therapy and other supportive measures. His chest pain improved, his shortness of breath got better, and he subsequently recovered. A further ECG on the next day (fig 4) showed increased ventricular rate (89 beats/min), normalisation of QTc, but evidence of LBBB with QRS duration of 144 ms identical to his admission ECG. The PR interval was 160 ms.

DISCUSSION

This case highlights an unusual ECG presentation of acute pulmonary embolism in that a LBBB pattern with prolonged QRS duration normalises temporarily. The following changes were observed in the ECG after acute pulmonary embolism (fig 2) compared with earlier (fig 1) and subsequent (fig 4) ECGs:

1. The ventricular rate was slower: sinus bradycardia at 54 beats/min.
2. The QRS duration shortened to within normal range at 82 ms.
3. The LBBB pattern had disappeared.
4. T wave inversions were noted in the anteroseptal chest leads.
5. The QT interval became prolonged.
6. The PR interval was unchanged.

Two explanations involving a delay in electrical conduction through the previously unaffected right bundle may account for these changes. Slowing of conduction via the right bundle branch is a recognised complication of acute pulmonary vascular obstruction and right ventricular strain.⁸ It is possible therefore that right bundle branch conduction velocity was

Vent. rate 89 bpm
PR interval 160 ms
QRS duration 144 ms
QT/QTc 414/504 ms
P-R-T axes 44 -28 129

Normal sinus rhythm
Left bundle branch block
Abnormal ECG

Referred by: DR ATHAR Unconfirmed

Figure 4 ECG showing evidence of LBBB.

transiently slowed to that of the left bundle after acute pulmonary embolism. If the conduction velocities along both right and left bundles were roughly equivalent, then activation of the two ventricles would be nearly synchronous.^{3,4} Assuming the initial LBBB pattern was caused by a conduction delay rather than a complete anatomic block and the peripheral branches of the intraventricular conduction system were unaffected, then the entire QRS complex may be of normal duration after activation of both ventricles.

The problem with this explanation is that the resulting bilateral bundle branch conduction delay effectively amounts to complete atrioventricular block, and therefore normally associated with prolongation of the PR interval. This was not seen in our case, although a sinus bradycardia was observed.

In fact the slowing of heart rate may provide an alternative explanation, as slower heart rate could slow conduction along the right bundle, while not affecting conduction along the atrioventricular node.^{6,7,9,10} The abnormal and delayed repolarisation pattern (as evident by marked T wave abnormalities and prolonged QT) would support this explanation. Marked T wave inversions in the absence of coronary lesions have been well documented during transient normalisation of LBBB previously.^{11,12} It is therefore also possible but less probable, that conduction velocity along the right bundle had slowed down relatively more than the left resulting in roughly equivalent conduction velocities along both bundles.¹⁰

A third reason, also dependent upon heart rate, may be that LBBB itself is related to heart rate acceleration.¹³ When the heart rate quickens, the R-R interval becomes progressively shorter and a descending impulse may find the left bundle branch still in its refractory period. A "block" is then registered. The rate at which conduction changes is called the "critical rate". It will persist until the cycle lengthens enough for normal conduction to occur. Rarely, rate dependency of bundle branch block may be revealed when a sudden lengthening of the ventricular cycle causes the disappearance of a previously present bundle branch block pattern. A common example of this is seen at the end of the lengthened cycle after an extra systole. One of the interesting features of rate dependent bundle branch block is that the critical rate at which block develops are different (faster) than the rate at which, once established, the bundle branch block disappears. PR interval is not

Summary points

- Pulmonary embolism may result in permanent or transient ECG abnormalities.
- Conduction delay along the right bundle may result from acute pulmonary vascular obstruction and right ventricular strain.
- Normalisation of LBBB in pulmonary embolism is a rare but known phenomenon resulting from equivalent delay along the right bundle branch.
- The appearance and disappearance of LBBB may also be rate dependent.
- Marked T inversion in precordial leads is a manifestation of massive pulmonary embolism.

prolonged. The features of rate dependent LBBB fits with our patient where all ECGs with the registered bundle branch block pattern also showed heart rates exceeding 85 beats/min while the ECG with normal intraventricular conduction is associated with a heart rate less than 60 beats/min.

This case has important clinical implications as ECG changes of disappearing and reappearing LBBB, with marked T wave changes in the anterior leads in patient with chest pain and shortness of breath could mimic acute myocardial infarction resulting in inappropriate thrombolysis.

This case is unusual and interesting in that the ECG presentation of acute pulmonary embolism was the temporary disappearance of a LBBB pattern. Only one other case in the last 30 years (based on a Medline search) had reported a similar finding.¹⁴ Our case is different in that marked T inversions were also seen in the precordial leads, suggesting abnormal repolarisation for the same reasons elaborated above.

Authors' affiliations

S M Athar, B S P Chin, Medical Assessment Unit, Russells Hall Hospital, Dudley DY1 2HQ, UK
E J Flint, Cardiology Department

Correspondence to: Dr Athar;
enid.humphries@dudleygoh-tr.wmids.nhs.uk

Submitted 30 November 2001
Accepted 4 April 2002

REFERENCES

- 1 **Lenegre J**. Etiology and pathology of bilateral bundle branch block in relation to complete heart block. *Prog Cardiovasc Dis* 1964;**6**:409–16.
- 2 **Lev M**, Unger PN, Lesser ME, *et al*. Pathology of the conduction system in acquired heart disease: complete right bundle branch block. *Am Heart J* 1961;**61**:593–9.
- 3 **Dressler W**. Delayed conduction in the bundle branches. *Am Heart J* 1945;**29**:728–31.
- 4 **Bauer GE**. Transient bundle branch block. *Circulation* 1964;**29**:730–5.
- 5 **Wallace AG**, Laszlo J. Mechanisms influencing conduction in a case of intermittent bundle branch block. *Am Heart J* 1961;**61**:548–52.
- 6 **Tavazzi L**, Salerno JA, Chimienti M, *et al*. Tachycardia-dependent and bradycardia-dependent intraventricular conduction defects in acute myocardial infarction: electrocardiographic, electrophysiologic and clinical correlates. *Am Heart J* 1981;**102**:675–85.
- 7 **Canon DS**, Goodman DJ, Harrison DC. Electrophysiological studies in patients with rate-related intermittent left bundle branch block. *Br Heart J* 1974;**36**:653–9.
- 8 **Daniel KR**, Courtney DM, Kline JA. Assessment of cardiac stress from massive pulmonary embolism with 12-lead ECG. *Chest* 2001;**120**:474–81.
- 9 **Dressler W**. Transient bundle branch block occurring during slowing of the heart beat and following gagging. *Am Heart J* 1959;**38**:760–8.
- 10 **Rosenbaum MB**, Lepeschkin E. Bilateral bundle branch block. *Am Heart J* 1955;**50**:38–46.
- 11 **Denes P**, Pick A, Miller RH, *et al*. A characteristic repolarization abnormality with intermittent left bundle-branch block. *Ann Intern Med* 1978;**89**:55–7.
- 12 **Gould L**, Reddy CV, Singh B, *et al*. T-wave changes with intermittent left bundle branch block. *Angiology* 1980;**31**:66–8.
- 13 **Marriot HJ**. *Practical electrocardiography*. 7th Ed. Baltimore: Williams & Wilkins, 1984: 230–5.
- 14 **Obeid AI**, Fruehan CT. Normalization of prolonged IV conduction time in a patient with acute pulmonary embolism. *Chest* 1973;**63**:289–92.

Want full access but don't
have a subscription?

Pay per access

For just US\$25 you can have instant access to the whole website for 30 days. During this time you will be able to access the full text for all issues (including supplements) available. You will also be able to download and print any relevant pdf files for personal use, and take advantage of all the special features *Postgraduate Medical Journal* online has to offer.

www.postgradmedj.com