

'Focal' Cardiac Concussion – An Under-Recognized Problem?

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ABSTRAK

Gegaran jantung merupakan hentakan tumpul, mengejut terhadap bahagian dada yang boleh menyebabkan kematian. Walaubagaimanapun, terdapat sebahagian pesakit tidak memenuhi definisi tersebut. Kami melaporkan dua kes yang tidak memenuhi domain definisi tersebut. Dua mangsa lelaki mendapatkan rawatan di Jabatan Kecemasan dengan kesakitan dada sederhana selepas terlibat dalam kemalangan jalan raya. Didapati paras ST meningkat pada led dada V2 dan V3 serta peningkatan paras kreatin kinase dantropnin T. Elektrokardiogram tersebut balik ke paras biasa selepas 24 jam. Mereka discas dengan selamatnya selepas 24 jam rawatan di wad sementara Jabatan Kecemasan. Definisi gegaran jantung (commotion cordis) konvensional dan lebam jantung adalah tidak sesuai untuk menggambarkan keadaan kes-kes tersebut. Oleh itu, kami mencadangkan diagnosis gegaran jantung setempat (fokus). Kami juga ingin menekankan kebolehan Jabatan Kecemasan untuk memantau dan merawat pesakit-pesakit golongan ini di wad sementara jabatan ini.

Kata kunci: commotio cordis, sakit dada, tumpul, elektrokardiograf

ABSTRACT

Cardiac concussion is a sudden, direct blunt trauma cause to the chest which led to death. However, there are patients who did not completely fulfil this definition. We report two cases which did not fit into the definition domain. Two male patients presented to Emergency Department with moderate anterior chest pain after motor vehicle crash showed transient anterior ST segment elevation at chest lead V2 and V3 with raised creatine kinase and normal troponin T. The electrocardiogram changes fully resolved after 24 hours. Both patients were discharged uneventfully after 24 hours monitoring in Emergency Department short-stay ward. Conventional definition of cardiac concussion (commotion cordis) and cardiac contusion may

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be unsuitable to describe these cases. Therefore, we propose the diagnosis of focal cardiac concussion. We also highlighted the ability of Emergency Department to manage these patients in short-stay ward.

Keywords: commotio cordis, chest pain, nonpenetrating, electrocardiography

INTRODUCTION

Cardiac concussion is a sudden, direct blunt trauma cause to the chest which led to death (Koehler et al. 2004). It can be fatal or non-fatal which were reported in several case reports (Kaplan et al. 1993, Maron et al. 1995). However, these definitions seem lack of precision and no universal agreed upon the definition either (Koehler et al. 2004). Following were two cases which did not fit into the definition domain.

CASE REPORT

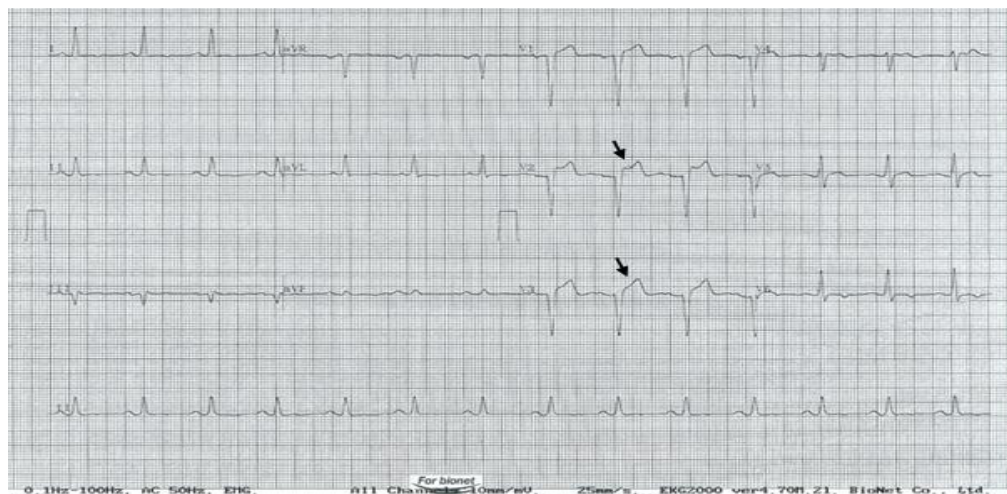
CASE 1

A 34-year-old man motorcyclist was hit by a lorry rear bumper over his anterior chest wall. He presented to Emergency Department (ED) of a university hospital two hours after the accident and complained of moderate chest pain with verbal numerical rating score (VNRS) of seven. Pain worsened on inspiration and it radiated to the back. His blood pressure (BP) was 190/114 mmHg, heart rate (HR) was 83 per minute, afebrile, oxygen saturation under room air (sPO₂) was 98% and respiratory rate (RR) of 19 per minute. Other systemic examinations were unremarkable. Extended Focal Abdominal Sonography for Trauma (eFAST) found no abnormality.

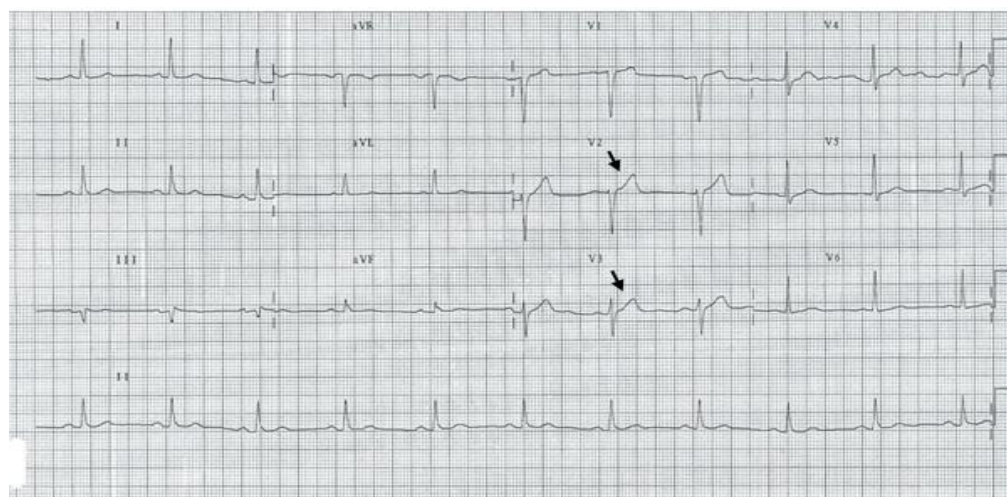
Bedside echocardiography showed no abnormality. Chest X-ray was normal. Initial electrocardiography (ECG) showed ST segment elevation of lead V₂ and V₃ (J point = 3mm elevation) and T inversion over lead I and V₆ (Figure 1). The initial creatine kinase (CK) was minimally raised (169 U/L), but troponin T was normal. He was administered intravenous tramadol and the chest pain resolved (VNRS zero). Patient was admitted to ED short-stay ward (EDSSW) for 24 hours observation. The subsequent serial of ECG's did showed resolution of ST-segment elevation to baseline at lead V₂ & V₃ with persistent T inversion at lead I & V₆. Repeated CK after 6 hours showed no further increment and the troponin T still remained undetectable. Patient was discharged after 24 hours of observation. The ECG on discharge showed normalised ST segment and T wave in all leads (Figure 1). CK level repeated three days later was 285 U/L. The troponin T and ECG remained normal.

CASE 2

A 21-year-old male car driver was brought in to ED after a head on collision with another car. He did not fasten his seatbelt. His anterior chest hit the steering wheel. He presented to ED of with the complaint of right sided headache & central chest pain



(a)

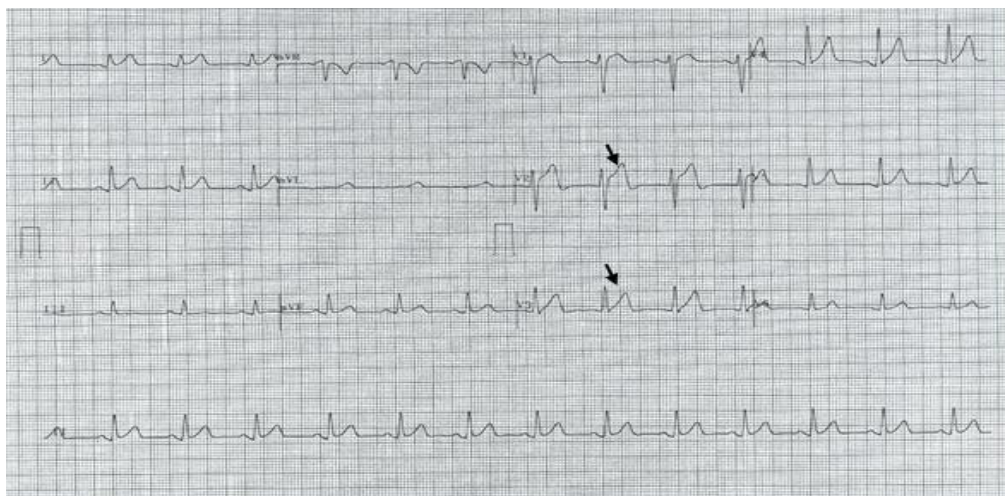


(b)

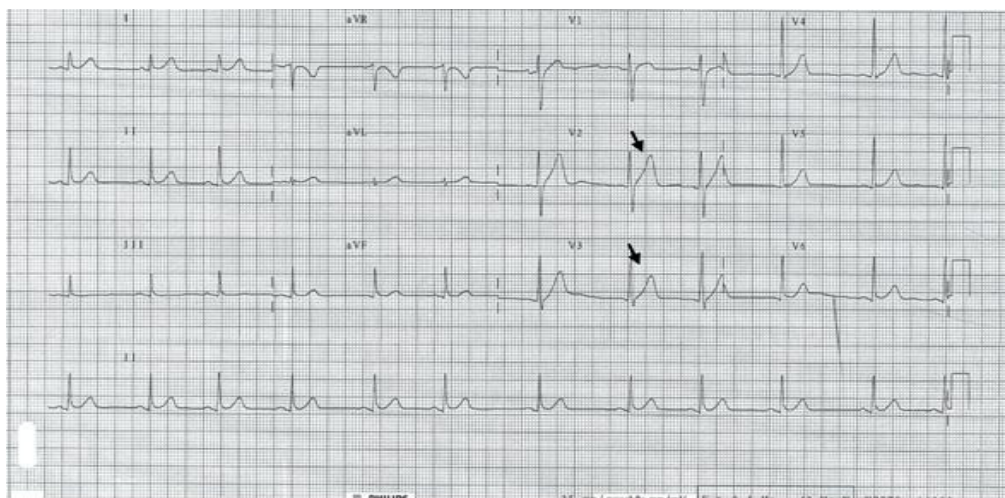
Figure 1: ST segment changes in V2 and V3 case report 1. (a) 2-lead ECG upon presentation showed ST segment elevation of lead V2 and V3 (J point = 3mm elevation) by the red arrow and T inversion over lead I and V6. (b) 12-lead ECG done after 24 hours showed return of the ST segment to baseline marked by the red arrow.

with VNRS of eight. His BP was 127/67 mmHg, HR was 88/min, RR was 18/min and sPO₂ was 97% under room air. His Glasgow Coma Scale score was 14 out of 15. There were multiple abrasion wounds over his face and tenderness over the right iliac fossa. Other systemic examinations were unremarkable. Initial ECG showed ST

segment elevation on lead V2 (J point = 3mm elevation) and V3 (J point = 1.5mm elevation) (Figure 2). EFAST examination showed no abnormality. Computer tomography of brain and cervical spine were performed and reported as normal. His initial CK level was raised (265 U/L) but troponin T level was undetectable (<0.013ug/L).



(a)



(b)

Figure 2: ST segment changes in V2 and V3 case report 2 (a) 12-lead ECG upon presentation showed ST segment elevation in lead V2 and V3 marked by the red arrow (b) 12-lead ECG done after 24 hours showed return of the ST segmentlead V2 and V3 to baseline marked by the red arrow.

Other blood investigations were normal. He was given intramuscular anti-tetanus toxoid and intravenous analgesia. The chest pain was resolved (VNRS zero). He was then admitted to EDSSW for cerebral concussion care and cardiac concussion monitoring. The serial of ECG had shown resolution of ST segment V2 and V3 to baseline

(Figure 2). A repeat CK and troponin T after 14 hours post-accident showed mild elevation of CK level (306 U/L) and troponin T remained undetectable. He was discharged well after 24 hours of observation in EDSSW. A follow-up telephone call to patient was made three days later showed uneventful recovery.

DISCUSSION

There are cases which do not fit in the current cardiac concussion definitions domain which are illustrated by the above two cases. These patients have regional ECG changes. They do not fulfil the diagnosis of cardiac concussion (commotiocordis) whereby the ECG changes are global. Therefore, the diagnosis of "focal" cardiac concussion should be considered. The current practice in diagnose and manage "focal" cardiac concussions are mainly based on acute coronary syndrome (ACS) modality. In these cases, the history and significant changes in the anterior ECG leads may indicate that they had cardiac concussion localized to the anterior wall of the heart. Linton et al. described the elevation of the ST segment indicates direct contact of a specific area of myocardium (Bishop et al. 1956). However, "focal" cardiac concussion differs from ACS in term of pathophysiology. "Focal" cardiac concussion involved electrophysiological component compare to ACS, which involved both myocardium perfusion and electrophysiological changes. Therefore, the definition of 'focal' cardiac concussion should take into account both electrophysiological and microcellular pathophysiological changes.

Existing biomarker of cardiac damage is based on injury to the cardiac muscle. They may not necessarily reflect dysfunction of the electrophysiological component of the myocardium. Nonetheless, immune-

histochemical changes may be present (Dettmeyer 2011). In a canine experimental study, patchy focal loss of the myocardial myoglobin, CK-MM and CK-BB were identified with these substances scattered deposited of between myocardial fibres (Dettmeyer 2011). The CK levels were elevated with normal troponin T level. The diagnosis of focal cardiac concussion maybe more appropriate in cases of regional ST segment elevation in ECG with normal troponin T level. A new diagnostic modality and approach should be developed. Further experimentation is recommended to develop a more accurate definition of cardiac concussion. In terms of patient's disposition, there is a dilemma as the prognosis is uncertain. Therefore, managing this group of patients is rather challenging. They were both successfully managed in EDSSW. Thus, it reduces in-hospital admission and can be cost effective.

CONCLUSION

We propose the diagnosis of focal cardiac concussion for these two cases. We also highlighted the ability of ED to manage these patients in EDSSW.

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