Intensive Care Med (2000) 26: 792–795

BRIEF REPORT

S. G. Sakka
E. Huettemann
W. Giebe
K. Reinhart

Late cardiac arrhythmias after blunt chest trauma

Received: 8 November 1999
Final revision received: 1 March 2000
Accepted: 9 March 2000

This work has been presented in part at the 13th European Colloquium on Quantitative Electrocardiology and Vectorcardiography, Paris, 30th September – 2nd October 1999

S. G. Sakka (✉) · E. Huettemann · K. Reinhart
Department of Anaesthesiology and Intensive Care Medicine, Friedrich-Schiller-University of Jena, Bachstrasse 18, 07740 Jena, Germany
e-mail: samir.sakka@med.uni-jena.de
Tel.: + 49-3641-933041
Fax: + 49-3641-933256

W. Giebe
Institute for Legal Medicine, Friedrich-Schiller-University of Jena, 07740 Jena, Germany

Abstract *Objective*: Case reports of two patients who developed fatal cardiac arrhythmias several days after blunt chest trauma.
Design: Case reports.
Setting: Surgical intensive care unit of a university hospital.
Patients: A 23-year-old man and a 9-year-old girl with blunt chest trauma and multiple further injuries following car crashes were transferred to our institution. Although ECG on admission was normal, both patients developed fatal cardiac arrhythmias after 6 and 4 days, respectively. In both patients, post-mortem analysis confirmed myocardial contusion without coronary artery lesions. Histological findings included severe interstitial oedema, haemorrhages and infiltration of lymphocytes and neutrophils, fresh myocardial necrosis and fatty degeneration.
Conclusion: Blunt chest trauma with myocardial contusion may lead to fatal cardiac arrhythmias even after several days, particularly when other severe injuries are present. Thus, a normal ECG on admission and absence of cardiac arrhythmias during the first 24 h of intensive care treatment do not necessarily exclude the occurrence of life-threatening arrhythmias in the further course.

Key words Blunt chest trauma · Myocardial contusion · Cardiac arrhythmias

Introduction

Myocardial contusion following blunt chest trauma occurs in 7–71% of the cases [1, 2]. Electrocardiographic (ECG) abnormalities following blunt chest trauma have been reported to occur in up to 70% within the first 3 days after hospitalisation [3]. Cardiac arrhythmias are most common, including uniform ventricular premature beats (54%), multifocal ventricular premature beats (16%), atrial fibrillation (6%) and ventricular tachycardia (3%). However, mortality resulting from cardiac arrhythmias is low [3, 4]. Previous studies suggest that 24–48 h of ECG monitoring after trauma is appropriate [5, 6]. In this report, we present two critically ill patients with fatal cardiac arrhythmias occurring several days after chest trauma and histologically proven myocardial contusion.

Case reports

Case 1

After a car crash, a 23-year-old man presented to a local hospital. On admission, the patient was awake and complaining of chest pain. Besides a right-sided pneumothorax for which he received a chest tube, chest X-ray showed ipsilateral fractures of clavicle, scapula and ribs II–IV. Chest computed tomography (CT) revealed multiple bilateral lung contusions, sonography and a CT of the abdomen showed a liver and right-sided renal contusion with a retroperitoneal haematoma. The patient was transferred to our institu-

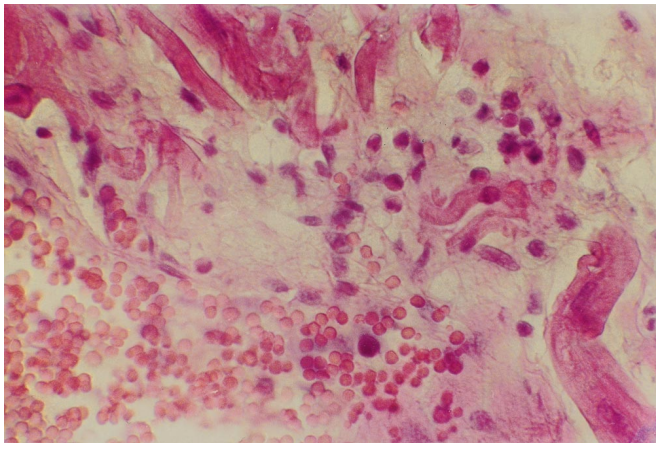

Fig. 1 Small haemorrhages in the myocardium with infiltration of granulocytes

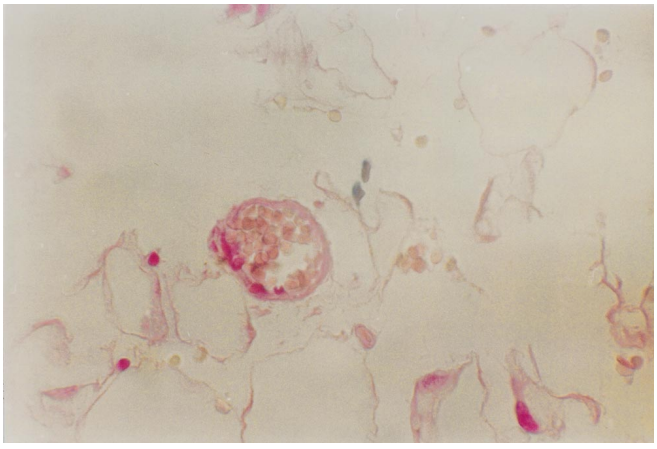

Fig. 3 Moderate haemosiderosis in the subepicardial connective tissue and fatty degeneration of myocytes 4 days after cardiac trauma

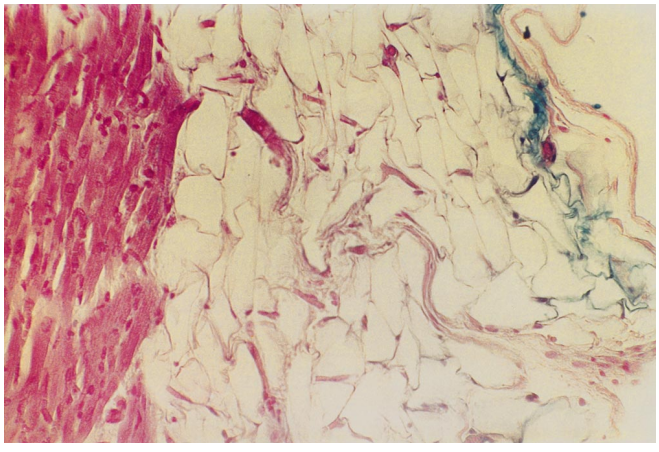

Fig. 2 Haemosiderosis in the subendocardium 6 days after cardiac trauma

tion. On arrival, he was awake, without any vasoactive drugs and required oxygen insufflation (3 l/min) for sufficient oxygenation. However, he required endotracheal intubation due to respiratory failure. After induction and maintenance of anaesthesia, he required 0.08 μg/kg · min norepinephrine for haemodynamic stabilisation. The 12-leads ECG and a rhythm strip showed a normal sinus rhythm without any abnormality. Unfortunately, he developed ARDS and the further course was complicated by systemic inflammatory response syndrome. Six days after the trauma, the patient suddenly developed ventricular tachycardia, intermittent left anterior hemiblock and, ultimately, left bundle branch block as documented by a 12-leads ECG. At this time, serum potassium (5.1 mmol/l), sodium (143 mmol/l) and lactate levels (2 mmol/l) were within normal ranges. Blood analysis showed a normal magnesium level of 0.84 mmol/l (normal 0.4–0.85 mmol/l) and excluded hypophosphataemia. Arterial partial pressure for oxygen (PaO_2) was 90 mmHg and for carbon dioxide ($PaCO_2$) 35 mmHg. Body temperature was 38.6 °C.

The patient had been stable in terms of cardiac rhythm (i.e., normal sinus rhythm on the monitor), global haemodynamics and oxygenation over the past 16 h while receiving norepinephrine (0.32 μg/kg · min) to maintain mean arterial pressure above 60 mmHg. An internal pacemaker was inserted due to the hemiblock syndrome. In the following hours, increasing dosages of norepinephrine (up to 4 μg/kg · min) were required and the patient developed signs of multiple system organ failure. About 10 h after the onset of these arrhythmias ventricular fibrillation occurred. However, sustained cardiopulmonary resuscitation (CPR) remained unsuccessful. Five hours after the start of arrhythmias, serum creatine-phosphokinase (CPK) was 8,100 IU/l (normal range < 150 IU/l) and CPK-isoenzyme MB 128 IU/l (normal range < 6–8 % of CPK). Autopsy revealed multiple contusions of the right lung, a liver and a right-sided kidney laceration. Slices of the myocardium appeared pale with local haemorrhages in the central part of the interventricular septum near the heart base. Microscopy of the myocardium showed severe interstitial oedema and haemorrhages with infiltration of lymphocytes and neutrophils (Fig. 1). Furthermore, fresh necrosis and fatty degeneration were described (Fig. 2).

Case 2

Following a car crash, a 9-year-old girl with severe head injury was brought to our institution by the emergency medical service. On admission, she was sedated, intubated and mechanically ventilated. After diagnostic procedures, she underwent surgery for a femoral bone fracture and received a Crutchfield extension due to a cervical vertebral subluxation C4/C5 with a C4 fracture. Furthermore, an epidural probe was placed for the continuous monitoring of intracranial pressure. On arrival at the intensive care unit (ICU), she was haemodynamically stable while receiving 0.28 μg/kg · min norepinephrine to keep mean arterial pressure above 60 mmHg. Three days later she underwent bilateral craniectomy due to intracranial hypertension resulting from brain oedema. However, cardiocirculatory instability due to intermittent atrioventricular dissociation developed during surgery. On her return to the ICU, vasopressor requirements were unchanged (norepinephrine 2.5 μg/kg · min). Heart rate was about 90/min and a 12-leads ECG excluded atrial fibrillation and revealed AV-dissociation with intermittent AV-node extrasystoles.

Within the next hours, global haemodynamics improved following moderate fluid loading and oxygenation was uncomplicat-

ed. An echocardiography was performed that excluded pericardial effusion and showed a normal left ventricular contraction pattern. About 12 h after the end of surgery, the child developed a transient total heart block and became hypotensive. At this time, serum potassium (5.1 mmol/l) was in the normal range, however, sodium (158 mmol/l) and lactate levels (7.2 mmol/l) were markedly increased. The arterial blood gas analysis revealed pH 7.428, BE –5.0 mmol/l, PaO_2 145 mmHg and $PaCO_2$ 28 mmHg. Several hours later, ventricular flutter required defibrillation and a transcutaneous pacemaker system was placed. Recurrent episodes of ventricular fibrillation occurred which, initially, could be successfully terminated. However, finally all therapeutic interventions to convert ventricular flutter and fibrillation failed. Autopsy of the brain showed contusions of the frontal lobe and moderate brain oedema which did not lead to a protrusion of the brain above the craniectomy area. Both lungs had multiple contusions. Furthermore, smaller haemorrhages in the interventricular septum with one near the heart base (about 1 cm large) and moderate subendocardial ecchymosis were described. Histological examination of the myocardium showed many small haemorrhages with fresh necrosis, moderate infiltration of leukocytes and fatty degeneration of myocytes (Fig. 3).

Discussion

Blunt chest trauma leading to cardiac injury is commonly known to be associated with cardiac arrhythmias. Development of arrhythmias may result in considerable morbidity but low mortality [3, 4]. However, the specific mechanisms for arrhythmias occurring after blunt cardiac injury remain unknown. Pathophysiologically, ectopic pacemakers from myocardial stretching, electrical gradients between injured and intact tissue, local hypoxia, ischaemia and/or oedema, and vagosympathetic stimulation or interference with neuromuscular interactions have been discussed [7, 8]. Finn et al. [9] reported an 18-year-old man who fell prone onto a football and developed ventricular fibrillation. During resuscitation, which remained unsuccessful, transient third degree heart block became obvious. Autopsy verified a right atrial subendocardial tear extending through the conduction system which was considered as the cause of death.

Most commonly, arrhythmias are considered to develop within the first hours after trauma [10, 11]. Wisner et al. [4] found, in 3,010 patients with blunt chest trauma, only four patients with arrhythmias requiring therapy. The maximum time interval between trauma and onset of arrhythmias was 48 h. In a study of 17 patients by Mayfield et al. [12], all five deaths occurred within 1.5 h after trauma. Based on a study of 71 patients, Illig et al. [5] proposed an ECG monitoring for 24–48 h. In a case report, Silverman et al. [13] described a 40-year-old man, after a motor vehicle accident, who was asymptomatic for 26 h before he developed ventricular fibrillation. Early direct current cardioversion re-established sinus rhythm. However, 24 h later he developed left bundle-branch block and multiple organ dysfunction. On day 4 ventricular tachycardia occurred, which finally led to death. Post-mortem examination of the heart showed congestion in the left ventricular posterior wall and disruption of myocardial fibres.

Experimental studies [7] have underlined that fatalities due to ventricular arrhythmias and cardiogenic shock occur immediately after injury. Anderson et al. [14] could demonstrate that severity and duration of arrhythmias correlated closely with the amount of impact on the myocardium. In a canine model of myocardial contusion [15], 50 % of the animals died from arrhythmias within a few minutes. No animal developed any life-threatening arrhythmias in the remaining study period. Based on clinical studies, a normal ECG on admission in a stable non-ICU patient predicts excellent outcome [6] and monitoring over 24 h has been suggested as appropriate. In general, the risk of having a clinically significant myocardial contusion is insignificant if the admission ECG is normal [16].

In contrast to these recent evidence-based guidelines [16], we present two critically ill patients suffering from other severe injuries who had normal sinus rhythm on admission but developed late cardiac arrhythmias. In our department, a 12-leads ECG with a 1-min rhythm strip is a standard procedure in every patient admitted to the ICU. Since monitor ECG showed a normal sinus rhythm for several days, follow-up 12-leads ECG examinations were obtained after several days when arrhythmias occurred. Unfortunately, both patients died between 4 and 6 days after the initial trauma from therapy-refractory arrhythmias. Thus, our two patients confirm the statements of two recent articles [17, 18] which both emphasise the possibility of late serious cardiac sequelae that range from arrhythmias to shock and/or death in patients with blunt chest or cardiac trauma undergoing major operations for accompanying injuries. The specific feature of these case reports is the time interval between the trauma and the onset of life-threatening arrhythmias. In the literature, there is one case of late cardiac arrest after myocardial contusion, i. e. on the 5th postoperative day, in which the patient had a thoracic aortic rupture as additional significant injury [19]. Autopsy had confirmed myocardial contusion. Hoppe et al. [20] reported a 29-year-old ice hockey player who developed atrial tachyarrhythmia 2 days after blunt chest trauma. While coronary artery angiography was normal, left ventricular anterior wall was found to be akinetic. The obtained endomyocardial biopsy excluded myocarditis and confirmed myocardial contusion with extravasation of red blood cells and degeneration of myocytes. The left ventricular anterior wall returned to normal function within the following 3 months. Norton et al. [21] reported two patients who developed ventricular fibrillation 36 h after admission and recommended monitoring patients at risk for at least 48 h. Wisner et al. [4] also mentioned one patient with arrhythmias due to supraventricular tachycardia treated 48 h after admission for blunt chest trauma.

In fact, our patients had numerous other injuries in addition to myocardial contusion that may have enhanced the susceptibility for cardiac arrhythmias. Abnormalities of serum potassium levels, i.e. due to renal failure, which may lead to cardiac arrhythmias, were not present in our patients. Unfortunately, only one patient underwent echocardiography by which pericardial effusion and pathological left ventricular contraction pattern were excluded. Among the different methods evaluated for the clinical diagnosis of myocardial contusion, echocardiography and CPK levels showed only a moderate sensitivity [5] while troponin I might be more reliable [22]. Unfortunately, troponin I was not measured in our two patients because this test was not yet available in our department at that time. Nevertheless, neither CPK nor troponin I levels are useful in predicting which patient will have complications related to blunt cardiac injury [16].

In both patients, autopsy confirmed myocardial contusion of the interventricular septum directly below the valvular plain near to the conductive system and coronary artery lesions could be ruled out. Although both patients were receiving norepinephrine, the pathological appearances were not those of catecholamine-induced contraction band necrosis, which can be seen with high-dose inotropic therapy. Since other conditions that may cause dysrrhythmias, such as hypotension, acidosis or hypoxaemia, can be excluded in our patients, blunt cardiac trauma seems to be the main causative factor that led to the fatal arrhythmias. Furthermore, intracardiac catheters as well as pacing wires may cause arrhythmias. In our patients, central venous catheters were in the correct position and pacing wires were placed even after the onset of arrhythmias. Nevertheless, the potentially proarrhythmic effects of high dosages of norepinephrine [23] may have contributed to the development of cardiac arrhythmias. However, a mono-causal relationship seems unlikely and other contributing factors cannot definitely be excluded, since our patients suffered from multiple severe injuries besides blunt cardiac injury.

In conclusion, blunt chest trauma causing myocardial contusion may lead to fatal cardiac arrhythmias not only in the very early period but even after several days. Thus, a normal ECG on admission and absence of cardiac arrhythmias during the first 24 h of ICU treatment may not necessarily exclude life-threatening arrhythmias. Further studies are needed to evaluate the pathomechanisms involved.

References

1. Frazee RC, Mucha P, Farnell MB, Miller FA (1986) Objective evaluation of blunt cardiac trauma. J Trauma 26: 510–520
2. Dubrow TJ, Mihalka J, Eisenhauer DM, De Virgilio C, Finch M, Mena IG, Nelson RJ, Wilson SE, Robertson JM (1989) Myocardial contusion in the stable patient: what level of care is appropriate? Surgery 106: 267–274
3. Potkin RT, Werner JA, Trobaugh GB, Chestnut CH 3rd, Carrico CJ, Hallstrom A, Cobb LA (1982) Evaluation of noninvasive tests of cardiac damage in suspected cardiac contusion. Circulation 66: 627–631
4. Wisner DH, Reed WH, Riddick RS (1990) Suspected myocardial contusion. Triage and indications for monitoring. Ann Surg 212: 82–86
5. Illig KA, Swierzewski MJ, Feliciano DV, Morton JH (1991) A rational screening and treatment strategy based on the electrocardiogram alone for suspected cardiac contusion. Am J Surg 162: 537–544
6. Healey MA, Brown R, Fleiszer D (1990) Blunt cardiac injury: is this diagnosis necessary? J Trauma 30: 137–146
7. Doty DB, Anderson AE, Rose EF, Go RT, Chiu CL, Ehrenhaft JL (1976) Cardiac trauma: clinical and experimental correlations of myocardial contusion. Ann Surg 180: 452–460
8. Utley JR, Doty DB, Collins JC, Spaw EA, Wachtel C, Todd EP (1976) Cardiac output, coronary flow, ventricular fibrillation and survival following varying degrees of myocardial contusion. J Surg Res 20: 539–543
9. Finn WF, Byrum JE (1988) Fatal traumatic heart block as a result of apparently minor trauma. Ann Emerg Med 17: 59–62
10. Jones JW, Hewitt RL, Drapanas T (1975) Cardiac contusion: a capricious syndrome. Ann Surg 181: 567–574
11. Harley DP, Mena I, Narahara KA, Miranda R, Nelson RJ (1984) Traumatic myocardial dysfunction. J Thorac Cardiovasc Surg 87: 386–393
12. Mayfield W, Hurley EJ (1984) Blunt cardiac trauma. Am J Surg 148: 162–167
13. Silverman SH, Turner WW Jr, Gilliland MBF (1990) Delayed cardiac arrest after myocardial contusion. Crit Care Med 18: 677–678
14. Anderson AE, Doty DB (1975) Cardiac trauma: an experimental model of isolated myocardial contusion. J Trauma 15: 237–244
15. Stein PD, Sabbah HN, Viano DC, Vostal JJ (1982) Response of the heart to nonpenetrating cardiac trauma. J Trauma 22: 364–373
16. Pasquale M, Fabian TC (1998) Practice management guidelines for trauma from the Eastern Association for the Surgery of Trauma. J Trauma 44: 941–956
17. Trupka A, Nast-Kolb D, Schweiberer L (1998) Blunt chest trauma. Unfallchirurg 101: 244–258
18. Pezzella AT, Silva AT, Lancey RA (1998) Cardiothoracic trauma. Curr Probl Surg 35: 647–789
19. Kram HB, Appel PL, Shoemaker WC (1988) Increased incidence of cardiac contusion in patients with traumatic thoracic aortic rupture. Ann Surg 208: 615–618
20. Hoppe UC, Erdmann E (1997) Contusio cordis – zu selten diagnostiziert? Med Klin 92: 444–446
21. Norton MJ, Stanford GG, Weigelt JA (1990) Early detection of myocardial contusion and its complications in patients with blunt trauma. Am J Surg 160: 577–582
22. Adams JE 3rd, Dávila-Román VG, Bessey PQ, Blake DP, Ladenson JH, Jaffe AS (1996) Improved detection of cardiac contusion with cardiac troponin I. Am Heart J 131: 308–312
23. Tisdale JE, Patel RV, Webb CR, Borzak S, Zarowitz BJ (1995) Proarrhythmic effects of intravenous vasopressors. Ann Pharmacother 29: 269–281