A Case of Traumatic Cardiac Tamponade Showing Sudden Spontaneous Disappearance of the Pericardial Fluid

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Traumatic cardiac tamponade must be treated by pericardial drainage as soon as possible. We recently encountered a rare case of traumatic cardiac tamponade in which the pericardial fluid disappeared spontaneously immediately before the planned drainage. This case is reported in this paper.
The patient was a 22-year-old male who was transported to our hospital after he sustained injuries in a traffic accident. The patient was diagnosed to have a facial bone fracture, bilateral lung contusions, myocardial contusion (suspected), injury to the spinal cord at the L3-L4 level, injury to the left kidney and pelvic fracture. After TAE was performed to deal with the bleeding from the injured pelvis, the patient was immediately hospitalized. About 6 hours after the injury, pericardial fluid accumulation began to be noted, and about 18 hours after the injury, the patient went into shock, responding poorly to fluid resuscitation and treatment with pressor agents. At this time, a diagnosis of cardiac tamponade was made and emergency operation was arranged for. However, just before this could be executed, the patient's blood pressure showed a sharp rise, accompanied by disappearance of the pericardial fluid. He continued to show steady improvement and could eventually be discharged from the hospital.

Key words: delayed traumatic cardiac tamponade, spontaneous drainage

INTRODUCTION

One of the most life-threatening conditions potentially associated with chest injuries, is traumatic cardiac tamponade, which must be treated by pericardial drainage as soon as possible [1, 2]. Pericardial effusion complicated by cardiac tamponade can arise from various causes. Since the condition involves the risk of cardiac rupture, the medical facility dealing with patients with this condition should be in readiness to perform emergency surgery when needed [1-3]. We recently encountered a rare case of multiple trauma in which cardiac tamponade developed about 18 hours after the injury, but before immediate pericardial drainage could be executed, the pericardial fluid disappeared spontaneously, with complete recovery by conservative treatment. This case is presented in this paper.

CASE REPORT

The patient was a 22-year-old man involved in a traffic accident. Major complaint: Lumbar backache. Disease history: Not significant. History of present illness: While riding a motorbike, he was unable to successfully negotiate a curve and hit against a wall. He was transported by ambulance to the Emergency Room of our hospital.

Findings at arrival: GCS, E4V5M6; respiratory rate, 36/min; heart rate, 96/min; blood pressure, 120/60 mmHg. Physical examination revealed tenderness in the lumbar and pelvic regions, and paresis in both lower extremities. A mild bruise was noted in the precordium, but auscultation revealed clear respiratory sounds, with no difference in the intensity of the respiratory sounds between the right and left sides. There was no cardiac murmur. No other abnormalities were noted.

Hematological test data: The serum Hb was low (8.2 g/dl). Elevation of the serum CK (695 IU/l) and myocardial troponin I (4.20 ng/ml) levels was noted. There were no other laboratory abnormalities (Table 1).

Diagnostic imaging: Echocardiography revealed no signs of fluid in the thoracic, abdominal or pericardial cavities. Plain X-ray and CT scans revealed a facial bone fracture, bilateral lung contusions, burst fracture

Table. 1 Laboratory data on admission

Hematology	Blood biochemistry	Blood gas analysis (O_2 10 L/min)
WBC 14,700 /mm^3	CK 695 IU/l	pH 7.357
RBC 2.52×10^4/mm^3	GOT 53 IU/l	$PaCO_2$ 40.5 mmHg
Hb 8.2 g /dl	GPT 26 IU/l	PaO_2 391.9 mmHg
Ht 24.2 %	LDH 578 IU/l	HCO_3^- 22.2 mmol/L
Plt 10.5×10^4 /mm^3	troponin-I 4.20 ng/ml	BE -2.0 mEq/l

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of the third and fourth lumbar vertebrae, injury to the left kidney, pelvic fracture and hematoma of the surrounding areas. No pericardial effusion was noted (Fig. 1 and 2-(1)).

Diagnosis at arrival: The patient was diagnosed as having a maxillary fracture, bilateral lung contusion, myocardial contusion, burst fracture of the third and fourth lumbar vertebrae, injury to the left kidney and pelvic fracture.

Course after arrival (Fig. 2): Contrast-enhanced CT scans revealed extravascular leakage of the contrast material from the site of injury in the pelvic and lumbar vertebrae. Transcatheter arterial embolization (TAE) was carried out to control the bleeding (Fig. 2-(2)). The patient was admitted to the ICU about 4 hours after the injury.

Course after admission: About 6 hours after the injury (Fig. 2-(3)), mild pericardial effusion was diagnosed by echocardiography (Fig. 3a) and CT (Fig. 4). However, the patient remained hemodynamically stable and no physical abnormalities were noted. Therefore, the patient was followed up conservatively, without active treatment. About 8 hours after the injury, the patient's blood pressure began to decrease gradually. At this time, I.V. fluid therapy, blood transfusion and administration of a pressor agent was initiated. The blood pressure improved for a while, but began to decrease gradually again. Around 17 hours after the injury, the patient complained of chest discomfort, and sweating and cyanosis were noted. At 18 hours after the injury (Fig. 2-(4)), the blood pressure was 78/56 mmHg and heart rate was 124/min, and engorgement of the neck veins and paradoxical pulse were noted. The patient responded poorly to rapid I.V. fluid infusion and administration of the pressor agent at increasing doses. At this time, echocardiography (Fig. 3b) revealed marked increase in the size of the pericardial effusion, and the amplitude of the waves on the electrocardiogram was found to be lower than that in the ECG recorded at admission (Fig. 5). On the basis of these findings, the patient was diagnosed to have cardiac tamponade, and the medial team began to prepare for emergency surgery (pericardial incision and drainage, combined as needed with exploratory

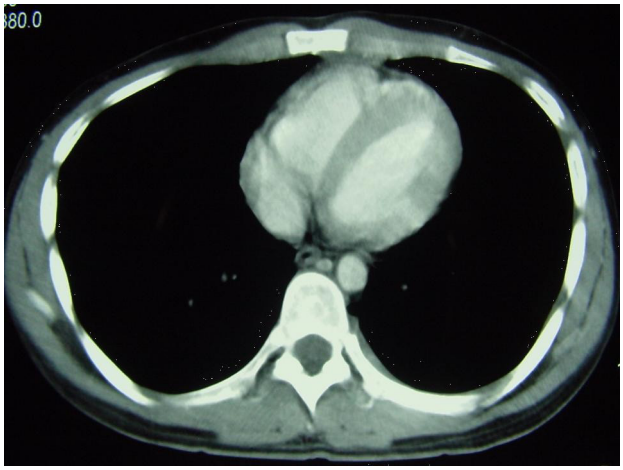

Fig. 1 Chest CT at arrival (about 1 hour after injury). No sign of pericardial effusion is noted.

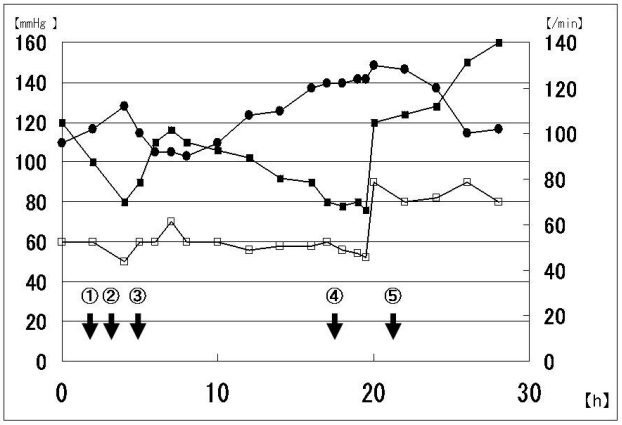

Fig. 2 Time-course of changes in the blood pressure and heart rate. The time of injury is denoted as 0. Filled circles (●) indicate the heart rate. Filled squares (■) indicate the systolic blood pressure. Open squares (□) denote the diastolic blood pressure.
① At arrival (about 1 hour after the injury). ② At the time of TAE (about 2 hours after the injury). ③ 6 hours after the injury. ④ 18 hours after the injury. ⑤ 21 hours after the injury

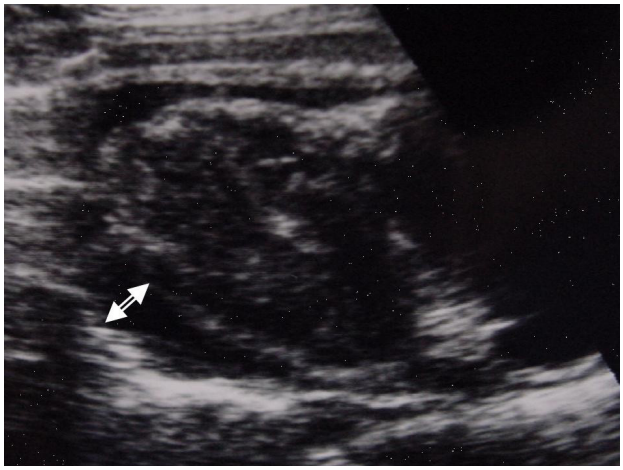

Fig. 3a Echocardiography at 6 hours after the injury. Slight pericardial effusion is noted (⇔).

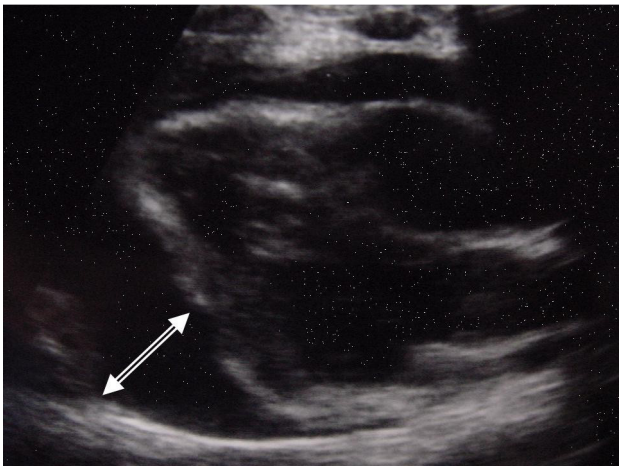

Fig. 3b Echocardiography at 18 hours after the injury. Marked increase in the size of the pericardial effusion (⇔).

thoracotomy).

However, at 19 hours after the injury (immediately before the emergency surgery could be executed), the patient's blood pressure began to rise suddenly, with steady improvement of the clinical symptoms. The pericardial fluid had disappeared almost completely on the ultrasound and CT images (Fig. 6) obtained 21 hours after injury (Fig. 2-(5)). At that time, fluid accumulation was noted in the pleural cavities bilaterally and in the anterior mediastinum. Drainage of the left thoracic cavity was performed, with the removal of about 700 ml of bloody fluid. Thereafter, the volume of drainage from the intercostals drain did not increase, the heart rate and blood pressure remained stable, and the symptoms resolved completely. It was thus surmised that the cardiac tamponade had subsided following drainage of the pericardial fluid into the thoracic cavity. Considering that the patient showed no sign of pericardial effusion at arrival and that relatively slow pericardial fluid accumulation occurred after arrival, we judged it unlikely that the patient had cardiac rupture. For this reason, surgery was skipped and the patient was followed up conservatively.

In the subsequent course, the pericardial effusion did not recur, and the fluid within the right thoracic cavity disappeared completely. The ST level on the electrocardiogram gradually returned to normal. The serum myocardial troponin I level decreased. No further abnormalities were detected by echocardiography or ECG. On the 18th hospital day, the patient underwent

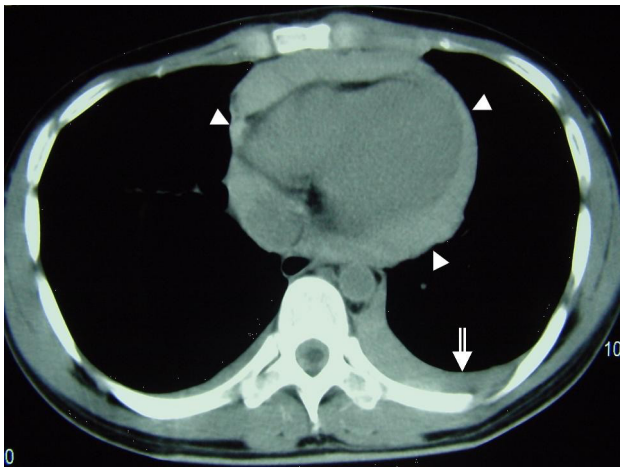

Fig. 4 Chest CT at 6 hours after the injury. Slight pericardial effusion is noted (▶). Slight fluid accumulation also noted in the left thoracic cavity (⇒).

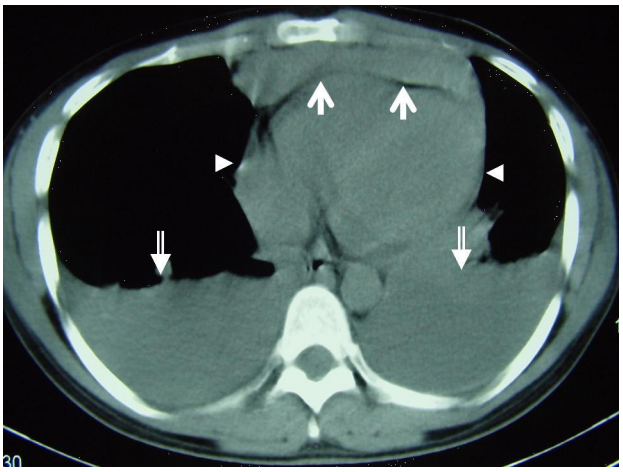

Fig. 6 Chest CT at about 21 hours after the injury. The pericardial fluid has disappeared (▶), the fluid in the thoracic cavity has increased (⇒) and a hematoma is visible in the anterior mediastinum (→).

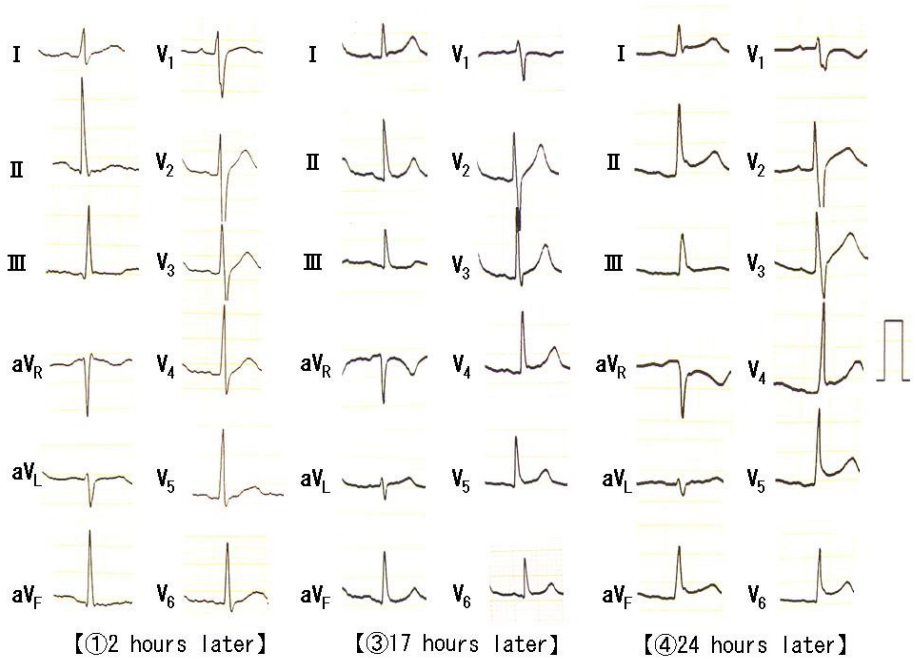

Fig. 5 12-lead ECG. (1) At 2 hours after the injury, ST depression was noted in leads II, III and aVF as compared with other ECGs. (2) At 18 hours after the injury, the amplitude of the waves was lower in all leads as compared with that in the previous ECG recorded at admission. (3) The low amplitude has alleviated, but ST elevation is still noted in all leads.

posterolateral fixation of the spine at the Department of Orthopaedic Surgery. His postoperative course was uneventful, therefore, the patient was referred to another hospital for rehabilitation on the 66th hospital day. His disease-free survival until at least about a year after the injury has been confirmed.

DISCUSSION

Bloody pericardial effusion caused by traumatic myocardial injury has been reported to occur in 5.9% of all cases of chest injury [3]. Traumatic cardiac tamponade often develops soon after the injury and requires immediate drainage and emergency surgery involving thoracotomy [1, 2]. In the case presented in this paper, while no sign of pericardial effusion was detected at the time of arrival of the patient to the hospital within a few hours of the injury, cardiac tamponade developed 18 hours after the injury. However, the patient was hemodynamically stable at this time and continued to show rapid and spontaneous improvement. Subsequently, at a time when removal of the fluid was planned because of worsening hemodynamics, the pericardial fluid suddenly disappeared from the diagnostic images, accompanied by fluid accumulation in the thoracic cavity and mediastinum. These findings suggest that in this case, blood gradually pooled in the pericardium after the injury, resulting in the onset of cardiac tamponade, but that the pericardial fluid then drained spontaneously through the fragile or injured parts of the pericardium into the thoracic cavity. This can be viewed as a very rare case, both from the point of view that the cardiac tamponade developed some time after the injury (delayed cardiac tamponade) and from the standpoint that it subsided by spontaneous drainage into the thoracic cavity before pericardiocentesis could be executed.

Delayed cardiac tamponade following blunt injury was first reported in 1920 by MacQuot et al. [4]. Few cases have been reported thereafter. The condition can therefore be viewed as rare. Cardiac tamponade is usually labeled as delayed cardiac tamponade if no sign of cardiac tamponade is detected at the fist evaluation and the fluid accumulates during the follow-up period. The length of time from the injury to the onset of delayed cardiac tamponade varies among reports, and ranges from several hours to several months [5-8]. In the present case, no sign of pericardial effusion was detected at arrival, with pericardial fluid accumulating gradually thereafter, eventually leading to cardiac tamponade; thus, a diagnosis of delayed cardiac tamponade was made. The causes of delayed cardiac tamponade reported to date include re-bleeding from a ruptured heart after temporary hemostasis [9], continued bleeding due to collapse of small blood vessels due to injury to the epicardium or coronary arteries [10, 11], and reactive or inflammatory pericardial effusion (Dressler's syndrome) [12, 13]. In cases of delayed cardiac tamponade developing relatively soon after the injury, exploratory thoracotomy often needs to be carried out, because of the possibility of cardiac rupture. On the other hand, cases of cardiac tamponade showing spontaneous recovery following drainage alone have also been reported [12]. Among others, the majority of cases with inflammatory pericardial effusion attributed to the possible development of autoantibodies directed against the injured myocardium (i.e., post-cardiac injury syndrome [12, 13]) follow a favorable courses after treatment by drainage alone. In the present case, pericardial fluid, which was absent at the time of arrival of the patient to the hospital, was first detected 6 hours after the injury and began to slowly influence the hemodynamics adversely by 8 hours after the injury. Clinical signs of cardiac tamponade were noted 17 hours after the injury. After spontaneous recovery from the cardiac tamponade, this patient was treated conservatively for two reasons: (1) it seemed likely that some factor other than cardiac rupture had caused the delayed cardiac tamponade, and (2) emergency surgery involved a high risk in this patient with polytrauma. Considering that this patient showed evident signs of myocardial contusion (i.e., changes in the ECG and elevation of the serum myocardial troponin level), exploratory thoracotomy at an appropriate time was one of the options considered.

Regarding spontaneous drainage observed in cases of cardiac tamponade, Paul et al. reported a case in 1983 where traumatic cardiac tamponade after blunt injury subsided as a result of spontaneous drainage into the thoracic cavity [3]. Our literature search identified no other cases with a similar outcome. The present case therefore seems to be a very rare case indeed. In the case reported by Paul et al., cardiac tamponade suddenly subsided immediately before the surgery scheduled to treat perforating cardiac injury, and this change was accompanied by increased discharge via a thoracic drain. When they opened the chest in their patient, a full-thickness injury (15-20 mm in length) was found in the right ventricular wall, and a slight accumulation of bloody fluid, free of aggregates, was found in the pericardial cavity. Based on these findings, Paul et al. judged that their patient had developed cardiac tamponade due to a perforating injury of the right ventricle, and that the tamponade subsided by spontaneous drainage from the wound in the pericardium into the thoracic cavity following elevation of the right ventricular and pericardial pressure due to I.V. fluid infusion, etc. We surmise that in the present case also, spontaneous drainage of the pericardial fluid into the thoracic cavity occurred by a mechanism similar to that reported by Paul et al., but in the presence of less serious injuries. In any event, this seems to be a very rare case.

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