Case Report

Simultaneous "traumatic Gerbode" and aortic rupture due to blunt chest trauma

Hector Anninos, Nikolaos G. Baikoussis[1], Panagiotis Dedeilias[1], Michalis Argiriou[1], Panagiotis Politis, Pantelis Gounopoulos, Apostolos Koroneos, Christos Charitos[1]

Departments of Intensive Care Medicine, and [1]Cardiovascular and Thoracic Surgery, "Evangelismos" General Hospital of Athens, Athens, Greece

ABSTRACT

The Gerbode defect is characterized by a perimembranous ventricular septal defect between the left ventricle and the right atrium. This intracardiac shunt is a congenital defect but may be iatrogenic after valve surgery or atrioventricular node ablation, may be the result of endocarditis or may be traumatic. It is really rarely encountered as sequelae of non-penetrating heart trauma, and their clinical manifestations may often be unrecognized in the multi-injured patient. However, they are serious complications, and their diagnostic approach is not always feasible. We hereby present a case of a young man with the left ventricle to the right atrium communication after blunt thoracic trauma due to a car accident and concomitant rupture of the thoracic aorta. We present also the case and the ways of treatment according to the international bibliography.

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INTRODUCTION

The Gerbode defect is characterized by a perimembranous ventricular septal defect (VSD) between the left ventricle and the right atrium. It is a really rare defect representing <1% of the whole congenital cardiac defects.[1] Acquired cases have been described, most often due to infective endocarditis, but also secondary to valvular surgery, thoracic trauma, and ischemic heart disease. Frank Gerbode was the first surgeon to report a successful series of patients underwent on intervention for left ventricular to right atrium shunt in 1958.[2] Gerbode has described two types of the defect; type A and type B. In type A form, the concurrent presence of a perimembranous VSD plus the tricuspid valve defect are visible. The shunt starts from the left ventricle to the right ventricle and through the tricuspid valve into the right atrium. This is referred to as an indirect left ventricle to right atrium shunt. The type B form is characterized by a left ventricle to the right atrium shunt. Gerbode *et al.* described two routes for blood to travel from the left ventricle to the right atrium. In a patient with a perimembranous VSD and in addition a defect in the tricuspid valve, the shunt is from left ventricle to right ventricle, through the tricuspid valve into the right atrium. This is also referred to as an indirect left ventricular-to-right atrial shunt.[3,4] The first description of direct communication between the left ventricle and the right atrium was reported by Buhl in 1857. The first successful surgical closure of such a defect was first reported by Kirby (using hypothermia

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Address for correspondence: Dr. Nikolaos G. Baikoussis, Department of Cardiovascular and Thoracic Surgery, "Evangelismos" General Hospital of Athens, Ipsilantou 45-57 Kolonaki, Athens, Greece. E-mail: nikolaos.baikoussis@gmail.com

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and inflow occlusion) at Hospital of the University of Pennsylvania in 1956. The first successful series of patients operated on with a left ventricular-to-right atrial shunt was reported by Frank Gerbode; surgeon at Stanford University.

We present a case of an adult man with a recent history of blunt thoracic trauma and concomitant traumatic Gerbode defect and aortic rupture. The best to our knowledge this is the first such case to be published.

CASE REPORT

A 26-year-old man with insignificant personal and family history, was admitted to hospital after suffering a car accident. On admission, he was fully awake and aware, with a Glasgow Coma Scale 15/15. He was slightly tachypneic; he had a heart rate of 110 beats/min and a blood pressure 80/50 mmHg. On auscultation, there was a decrease in vesicular breath sounds in the lower areas of the lung and a systolic murmur 3/6 heard diffusely in the precordial. The electrocardiogram revealed incomplete right bundle branch block with left posterior hemiblock [Figure 1]. The white blood cell count reached 22,000/mm^3 and the hemoglobin (Hg) was 14.5 g/dl. The rest of the blood analysis did not show any abnormal values during the admission. The patient underwent computed tomography (CT) scan which displayed bilateral pleural effusions with compression atelectasis, fractures of the 3rd, 4th, and 5th right costal ribs, contusion in the right lung, and rupture of the aortic isthmus with the presence of pseudoaneurysm and mediastinal hematoma [Figure 2]. The abdominal CT scan was normal. The patient was transferred to the catheterization laboratory and aortography was performed which confirmed the CT findings. No other site of blood extravasation was found [Figure 3]. A stentgraft was inserted to the descending aorta, at the isthmus level [Figure 4]. Subsequently, he was admitted in the Intensive Care Unit (ICU) to receive close monitoring. He was hemodynamically stable at the time (heart rate 100 beats/min, blood pressure 105/65 mmHg) and his arterial blood gases were 7.42/30/132/22 on FiO_2 60%. The oxygen saturation in blood drawn from the superior vena cava ($ScvO_2$) was measured 60%. Due to this borderline value and the systolic murmur aforementioned, a transthoracic cardiac ultrasound was performed, which disclosed an abnormal blood flow between the left ventricle and the right atrium without any remarkable findings regarding the right chambers [Figure 5]. A traumatic rupture in the aortic root creating a shunt toward the right atrium was presumed. A right heart catheterization followed demonstrating O_2 step-up at right atrium level (Hg oxygen saturation in superior vena cava, right atrium, right ventricle, and pulmonary artery were 46%, 80%, 85%, and 85% respectively) [Table 1]. This finding is equal of a left-right communication. The patient displayed elevated hepatic liver enzymes. Aspartate aminotransferase 455 IU, alanine aminotransferase (ALT)

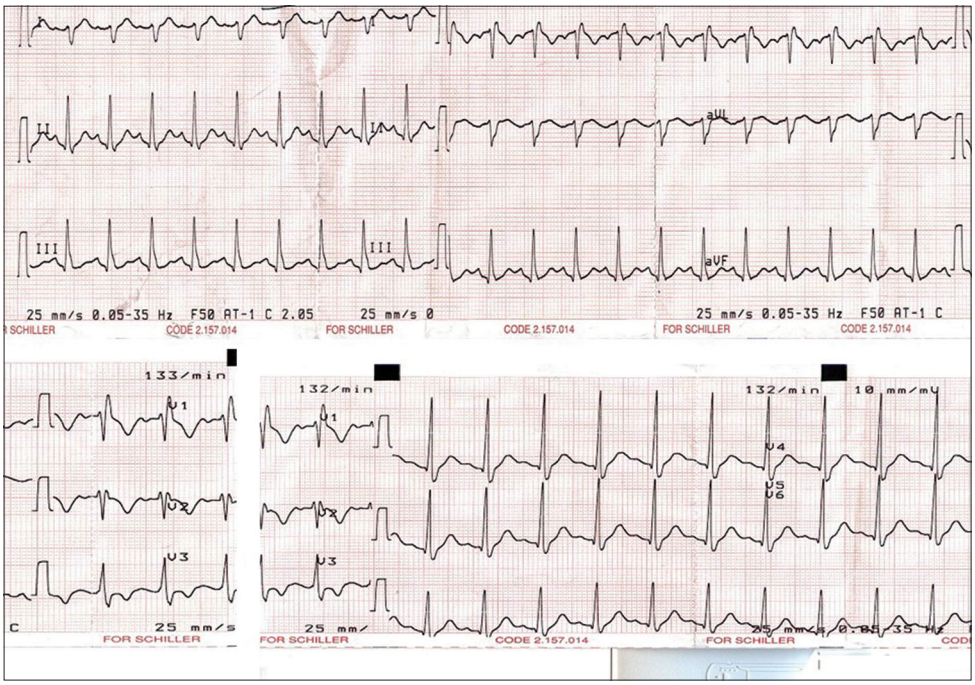

Figure 1: The electrocardiogram revealed incomplete right bundle branch block with left posterior hemiblock

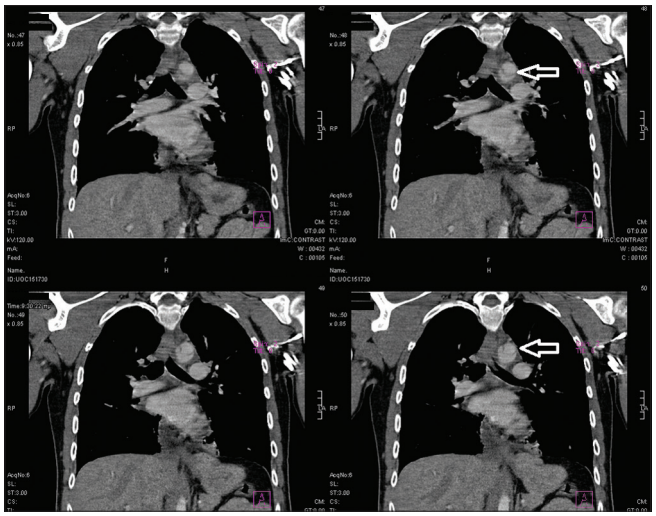

Figure 2: Computed tomography showing the rupture of the aortic isthmus (arrow) with the presence of pseudoaneurysm and mediastinal hematoma

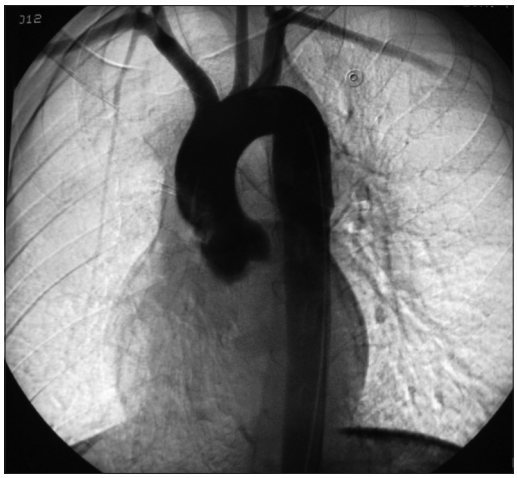

Figure 3: The aortography performed, confirmed the computed tomography findings

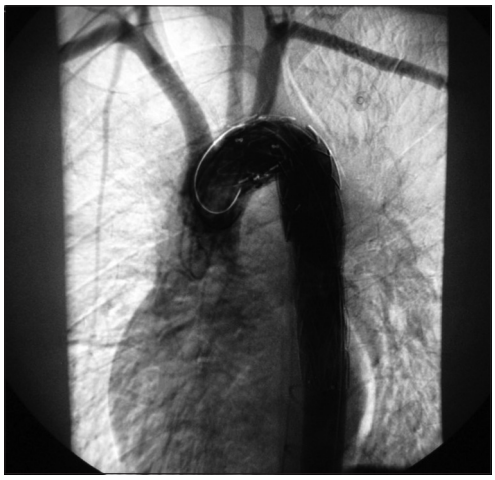

Figure 4: A stentgraft was inserted to the descending aorta, at the isthmus level

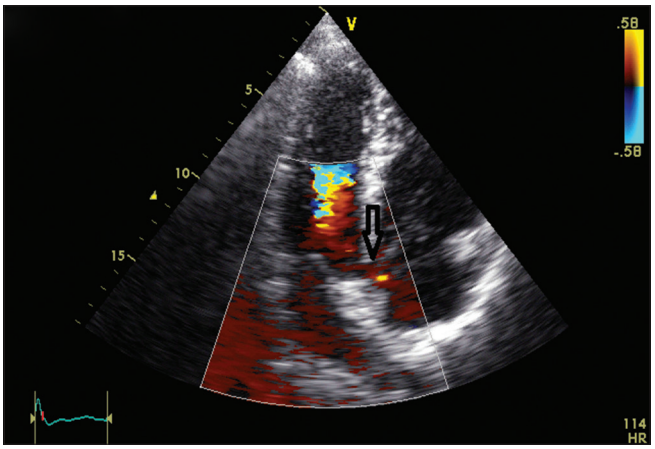

Figure 5: The transthoracic echo cardio performed show an abnormal blood flow between the left ventricle and the right atrium (arrow)

543 IU, normal <40 IU), creatine phosphokinase (CPK) 1388 (normal <190), CPK-MB 71 (normal <18), troponin (hsTpn) 245 ng/ml (normal <14, grey zone 14–56) [Figures 6-8]. The elevation of the hepatic enzymes show that this defect is recent and traumatic and not congenital. We would like here explain the treatment strategy.

After the interventional establishment of intracardiac shunt, an operation was undertaken by the cardiac surgeons who sealed the shunt with the placement of a synthetic patch. The patient underwent on median sternotomy and the connection with the cardio-pulmonary machine via a bicaval cannulation. Furthermore, a catheter for antegrade cardioplegia (blood-cold) was placed in aortic root and the catheter for left ventricle venting was placed in the superior right pulmonary vein. A right atriotomy was performed after total cardiopulmonary bypass (CPB) establishement and the defect was immediately detected [Figure 9]. A pericardial patch was sutured in order to repair this shunt with horizontal mattress stiches [Figure 10]. Heart rhythm was stablished and a weaning from CPB was able without inotropic support. The patient was transferred to the ICU. He did not require any special anesthesiology treatment postoperatively. He had an uneventful postoperative course except his atrioventricular block which was treated with a permanent pace maker implantation. The left pleural effusion was treated before discharge through a chest tube insertion. The patient discharged after 28 days of hospitalization and during his follow–up, he is in optimal health status. His long hospital stay was due to delay of the correct diagnosis and due to multi-injuries.

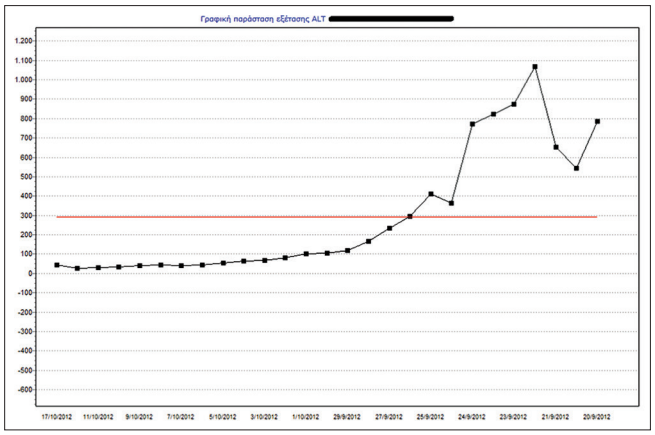

Figure 6: Diagram showing the alanine aminotransferase course

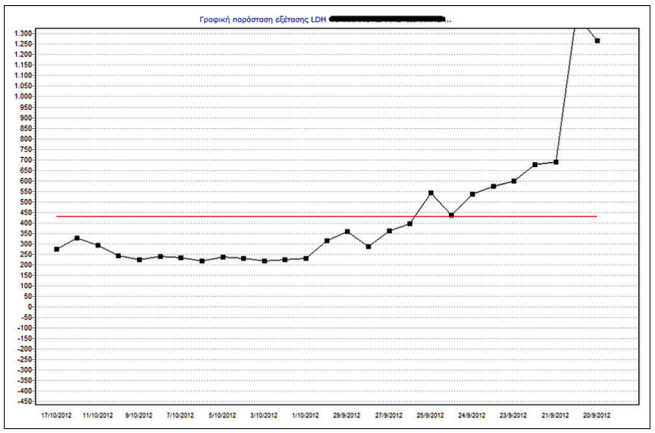

Figure 7: Diagram showing the lactate dehydrogenase course

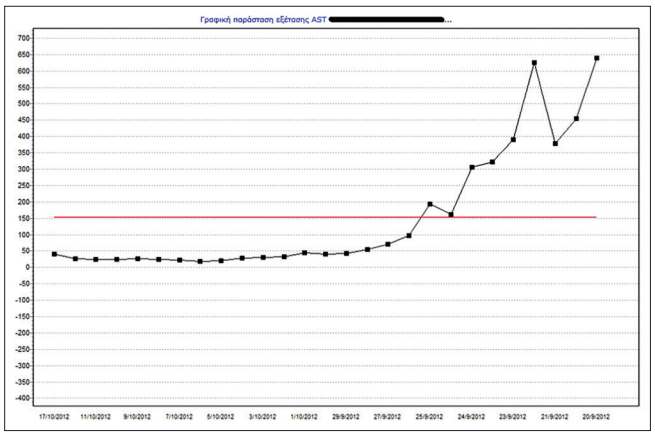

Figure 8: Diagram showing the aspartate aminotransferase course

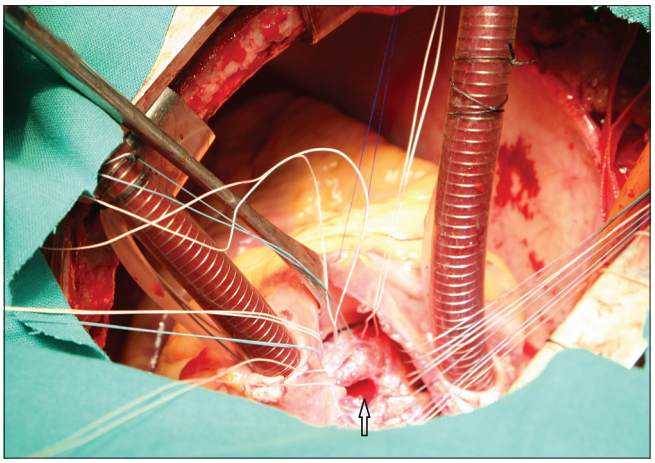

Figure 9: In this surgical field the defect was detected (arrow)

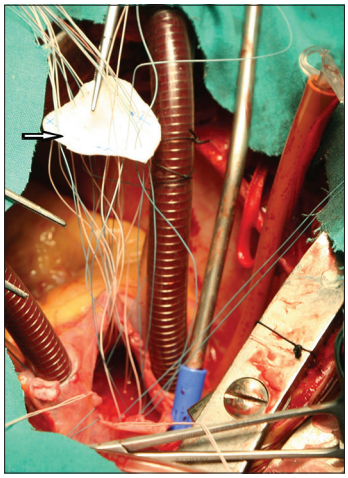

Figure 10: A pericardial patch (arrow) was sutured in order to repair this shunt

According the international bibliography the Gerbode defect must be corrected in the way above described. In the era of endovascular surgery and interventional cardiology, a percutaneous closure may be applied. This is not yet an established treatment because only some case reports have been published.[5-7]

Table 1: Right heart catheterization results

	SO_2%	PO_2 (mmHg)
Radial Artery	97	95,6
Superior Vena Cava	46	46
Right Atrium	78,6	47,8
	80	48,8
	86	55,4
	79	49
Right Ventricle	87	57
	88	58,4
	84,5	54
Pulmonary Artery	85	54
	82	51,5

DISCUSSION

Acquired left ventricular-to-right atrial communications are relatively uncommon complications, most often attributable to surgical procedures, trauma, endocarditis,

myocardial infarction or even endomyocardial biopsy.[8-10] Most cases represent postoperative complications. Blunt chest trauma is scarcely reported in the literature as a cause for such an injury.[11-13]

Usually myocardial contusion dominates the picture in nonpenetrating heart traumas although ventricular septal ruptures have been reported. Unfortunately, even in cases of intracardiac rupture the clinical presentation is nonspecific and can be underestimated in the context of more predominated injuries. The electrocardiogram shows findings that can be attributed to electrolyte disturbances, acid-base imbalance, and hypoxia or hypovolaemia, which are all relatively common in thoracic injuries. Atrio-ventricular conduction disturbances have been reported, although this was not the case in our patient.[14,15]

Though nondiagnostic, electrocardiographic findings can provoke a high degree of suspicion and initiate further evaluation by ultrasonography which can demonstrate a pathological flow and set the diagnosis. The echocardiographic study should be detailed to discriminate tricuspid regurgitation jet from the left ventricle to right atrium shunt. However, transthoracic examination may be flawed due to suboptimal visualization, especially in multi-trauma patients and in that case transesophageal approach can be helpful, after the stenting procedure in case of aortic trauma.[16-19] In selected cases contrast enhanced CT can be of diagnostic value.[20,21]

If the shunt is significant, hamodynamic deterioration may occur and this should prompt rapid surgical closure of the communication. According to the international bibliography the Gerbode defect must be corrected in the way above described.

In the era of endovascular surgery and interventional cardiology, a percutaneous closure may be applied.[5,6] It may be closed using the Amplatzer duct occluder.[7] In our case, the initial hamodynamic instability was attributed to the aortic trauma and after the stent was positioned, the patient indeed recovered from hypotension and shock. The intracardiac shunt was presumed mostly on the basis of the physical findings.

CONCLUSION

Nonpenetrating cardiac trauma can sometimes cause significant intracardiac damage leading to rupture of cardiac structures and producing pathological shunts. With respect to their size and location, these lesions can produce a variety of clinical features ranging from complete absence of symptoms to rapid hemodynamic deterioration and shock. After blunt trauma, one should always be alert for the presence of such complications and regular physical examination is necessary to detect new cardiac murmurs which will orientate further evaluation. Transthoracic ultrasound is a keystone examination but transesophageal views or CT may be needed in some cases.

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Conflicts of interest

There are no conflicts of interest.

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