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Case Presentation

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Intrathoracic Liver Due to Trauma: Uncommon Phenomenon, Doubtful Origin

David Sutori[1,2*], Tibor Kovacs[3], Attila Olah[3] and Tamas F Molnar[1,4]

[1]St. Sebastian Thoracic Surgery Unit, Chair of Surgery, University of Pécs, Petz Aladár University Teaching Hospital, Győr, Hungary

[2]Department of Trauma Surgery Unit, Petz Aladár University Teaching Hospital, Győr, Hungary

[3]General Surgery Unit, Chair of Surgery, University of Pécs, Petz Aladár University Teaching Hospital, Győr, Hungary

[4]Department of Operational Medicine, Faculty of Medicine, University of Pécs, Hungary

[*]**Corresponding author**: David Sutori, St. Sebastian Thoracic Surgery Unit, Chair of Surgery, University of Pécs, Petz Aladár University Teaching Hospital, 9172 Gyorzamoly, Buzavirag str. 26, Hungary, Tel: 36204762942; E-mail: david.sutori@gmail.com

Abstract

Traumatic diaphragmatic injuries (TDI) are relatively uncommon of all blunt thoraco-abdominal trauma. We describe the case of 57-year-old male with a history of a motor vehicle accident who presented with muted abdominal symptomatology and was found to have a right diaphragmatic hernia with the right hemiliver, gallbladder and part of the ascending colon with the hepatic flexure.

Keywords: Blunt thoracoabdominal trauma; Diaphragmatic injury; Chest trauma

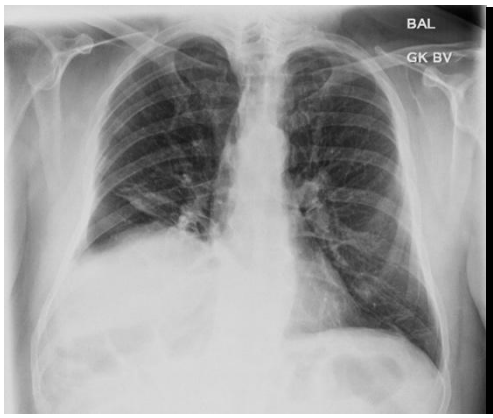
Background

Traumatic Diaphragmatic Injuries (TDI) are relatively uncommon of all blunt thoracoabdominal trauma **[1,2].** Around 20% of TDI occur on the right side **[3].** Diaphragmatic injuries on the right side are often asymptomatic **[4].** Suspicion may arise with chest X-ray, but three dimensional imaging, such as Computed Tomography (CT) or Magnetic Resosnace Imaging (MRI) examination is essential for accurate diagnosis **[5].** The injury may also encompass visceral herniation with, or without symptoms, where as upto 30% of blunt TDI present late [6].

Case Presentation

A muscular type 57-year-old male presented to the Emergency Department (ED) after a motor vehicle accident resulting in blunt trauma to the chest and left wrist. On primary survey, the patient was wake, hemodinamically stable, with normal vital parameters and oxygen saturation, without chest pain or any shortness of breath. No

abdominal complaints or pathological signs were noted. X-ray examinations showed left radial fracture and a significant elevation of the right hemidiaphragm, with slight shift of the mediastinal compartment to the left. Thoraco-abdominal CT **(Figure 1 and 2)** revealed diaphragmatic rupture and intrathoracic right hemiliver due to TDI. Free fluid in the chest and the abdomen was absent, whereas the rib cage was intact. Liver enzymes, as in transaminases, as well as static liver functional serum markers, suchas serum- and direct bilirubine and prothrombin time/International Normalized Ratio (INR) were also in the normal range **(Table 1).** The last chest X-ray prior to injury was performed four years prior to the present injury, and showed normal diaphragm. Patient reported occasional blunt chest trauma shaving worked with large animals, but didnot receive medical attention, as he was complaint free.



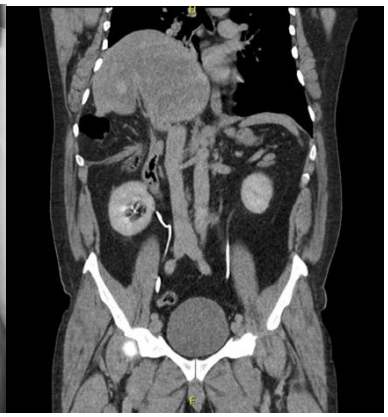


Figure 1 **Figure 2**

Table 1: Preoperative.

	current case	normal range	
total bilirubin	**31**	2 - 18	umol/l
GOT	**44**	6 - 37	U/L
GPT	**53**	6 - 40	U/L
Prothrombin time	**9.58**	7.7 – 9.7	sec
INR	**1.07**	0.85 - 1.15	
CRP	**81.4**	0 - 5	mg/L

On the fourth post trauma day a right postero-lateral thoracotomy was performed, with unilateral lung ventilation. The right lobe of the liver, the gallbladder and a part of the colon occupied a large part of the right chest cavity pushing up and compressing the entire right lung upto the level of the pulmonary veins **(Figure 3).** Thin adhesions connected the edges of the partly vanished centrum tendineum to the Glisson capsule of liver, indicating a sort of an already under taken organizing process. The right lobe of the liver, located intrathoracically gave an impression of being swollen but hypothrophic in spite of a stagnant appearance of the

circulation, while the left lobe partially below the hernial rim seemed to be hyperthrophic. Lack of blood, or reactive fluid questioned the theory of a completely fresh event. The parenchymal circulatory redistribution following release of strangulation was noted. Circular dissection of the adhesions made repositioning of the liver-bowel-omentum complex into the abdomenonly partially possible. An additional phrenotomy made the hernial gate sufficiently large enough, and all organs were repositioned into the abdominal cavity with a help of Fowler's position of the surgical table. The diaphragm was reconstructed using a dual (- layered) mesh, fixed with interrupted non-absorbable sutures **[7] (Figure 4 and 5).**

Figure 3

Figure 4

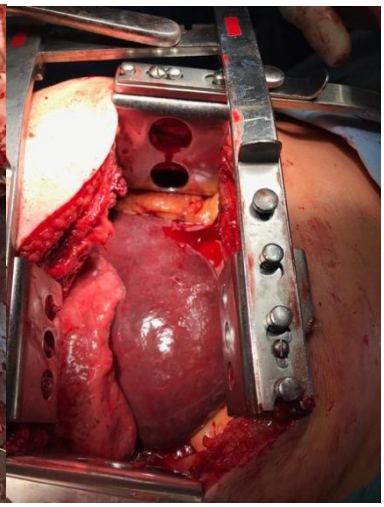


Figure 5

An uneventful recovery followed **(Figure 6)** and the patient was discharged on the 9th postoperative day. Laboratory chemistry tests on the first postoperative day have shown elevated liver enzymesas compared to preoperative values, normalizing by the day of discharge, one week later **(Table 2 and 3).**

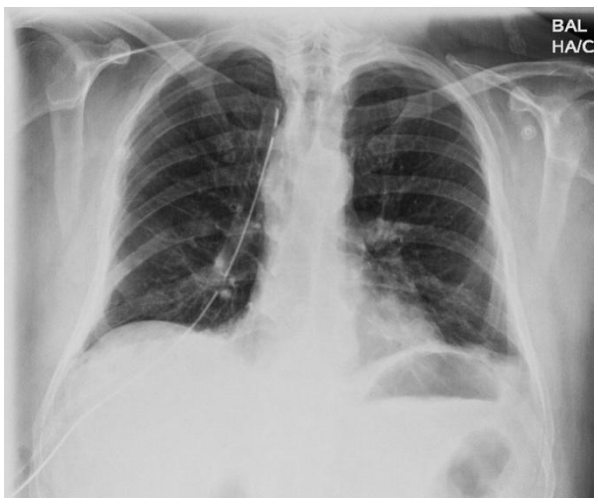


Figure 6

Table 2: Postoperative 1st day.

	current case	normal range	
total bilirubin	**17**	2 - 18	umol/l
GOT	**413**	6 - 37	U/L
GPT	**146**	6 - 40	U/L
Prothrombin time	**9.67**	7.7 – 9.7	sec
INR	**1.13**	0.85 - 1.15	
CRP	**249.4**	0 - 5	mg/L

Table 3: Postoperative 8th day.

	current case	normal range	
total bilirubin	**8**	2 - 18	umol/l
GOT	**41**	6 - 37	U/L
GPT	**61**	6 - 40	U/L
Prothrombin time	**9.43**	7.7 – 9.7	sec
INR	**1.01**	0.85 - 1.15	
CRP	**25.6**	0 - 5	mg/L

Discussion

This is a presentation of a clinically asymptomatic liver herniation/transposition into the chest cavity due to an undefined previous event, completed by a relatively minor blunt trauma, resulting in a partial strangulation and/or perhaps, portal circulatory redistribution of the liver. The be wondering, still controversial clinical picture is dominated by a hypertrophic liver parenchyma, possibly compensated by a compensatory hyperthrophic in the other part **[8,9].** Accordingly, the pathogenetic background of these volume changes remains of prime interest. On one hand, circulatory congestion of the right liver, secondary to a relative blockage of venous out flow due to the partial strangulation or rotation of the liver, may have been partially responsive for the noted hypotrophy of the right hemiliver, resulting in a simultaeous hyperflow of its left counter part. On the other hand, it would be interesting, still, very much plausible to hypothesize, that the observed hypertrophy-hypotrophy changes may have – atleast partially – been caused by changes in the portal circulation dynamics of the liver. In general, reduction of portal inflow in part of the liver lobe is known the induce the atrophy of affected liver parts (segments), where as the resultant hyperflow of the remaining segments leads to the irreactive hypertrophy over time (period of weeks to months), with the overall process termed as 'liver regeneration' **[10,11].** Clinically, the phenomenon serves as the background for the maneuver called 'Portal Vein Occlusion (PVO)' which is the deliberate/iatrogenic closure of the portal tributaries of a liver part, seeking to cause ipsilateral atrophy (of tumour-bearing liver), and contralateral hypertrophy (of tumour-free liver). PVO is routinely applied in modern hepatic cancer surgery to manipulate liver volumes in order to treat – and ultimately resect – primarily unresectable liver tumours **[12].** In the present case, a sort of 'spontanaeous PVO' may have also taken part in

the development of bilateral liver volume changes. Its interpretation is, that the hypotrophy of the right hemiliver may have partially been the result of a compromised portal flow secondary to a relative stenosis or an anatomical “twist” of the right portal vein, caused by the massive transposition of the right hemiliver to the right chest cavity. Meanwhile, the left, intraabdominally located hemiliver may have undergone a reactive hypertrophy secondary to a sequentially increased left portal inflow. However hard to prove, this hypothesis would underline the ’non-acute‘, or chronic/’two-stage‘ mechanism of the presen tcase.

An application of a dual-mesh overlap gave good results **[7,13],** as the size didnot allow direct closure. It is of note, that hyperthrophy of the liver due to either partial congestion or portal circulatory redistribution challenges reposition. The reversible part of parenchymal circulatory redistribution needs time and causes mild transitory increases in liver enzymes **[9].**

Conclusion

Timing of reconstruction of non-life-threatening diaphragm ruptures with tissue defect is of critical importance; where in an elective approach was justified. The mechanism of right- sided diaphragmatic hernia remains obscure, in which the role of repeated traumas and ’two- (or multiple) stage‘ ruptures cannot be excluded.

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