—Case Reports—

Visceral Ischemia Caused by Acute Aortic Dissection Following Blunt Aortic Injury: Report of a Case

Shigeki Kushimoto[1], Shin-ichiro Shiraishi[2], Masato Miyauchi[2], Reo Fukuda[2], Tomohiko Masuno[2] and Hiroyuki Yokota[2]

[1]Division of Emergency Medicine, Tohoku University Graduate School of Medicine
[2]Department of Emergency and Critical Care Medicine, Graduate School of Medicine, Nippon Medical School

Abstract

The diagnosis and management of blunt thoracic aortic injuries have seen significant changes over the last decade. The purposeful delay of definitive repair by aggressive blood pressure management has been suggested to be safe, and deliberate nonoperative management may be a reasonable alternative in selected patients with minimal aortic injuries detected with contrast-enhanced computed tomography. We report a case of acute aortic dissection due to blunt thoracic aortic injury resulting in splanchnic ischemia, for which we selected primarily nonoperative management, because the aortic tear was small but was complicated by multiple trauma. Although acute aortic dissection is an uncommon presentation of blunt injuries of the thoracic aorta and the poor prognosis has not been adequately emphasized, close observation, to evaluate the dissection and to determine whether splanchnic ischemia has developed, is recommended for patients for whom delayed definitive repair or nonoperative management has been selected.
(J Nippon Med Sch 2011; 78: 110–115)

Key words: acute aortic dissection, blunt thoracic aortic injuries, blunt chest trauma

Introduction

The diagnosis and management of blunt injuries of the thoracic aorta have seen significant changes over the last decade[1,2], although such injuries are traditionally considered surgical emergencies, on the basis of a 1958 study by Parmley et al[3]. Chest radiography for screening and diagnostic angiography have been replaced by computed tomography (CT) for the diagnosis of this injury. Endovascular repair has also largely replaced open repair and has significantly reduced mortality and morbidity[1,4]. On the other hand, purposeful delay of definitive repair by aggressive blood pressure management using beta blockers has been reported to be safe and is associated with reduced mortality in patients who are not candidates for immediate open repair or endovascular treatment. Moreover, deliberate nonoperative management has been suggested as a reasonable alternative for selected patients with minimal aortic injuries[5–9].

Herein, we report a case of acute aortic dissection due to blunt injury of the thoracic aorta that resulted in splanchnic ischemia, for which we selected primarily nonoperative management, because the aortic tear was small and was complicated by multiple trauma, including lung

Correspondence to Shigeki Kushimoto, MD, Division of Emergency Medicine, Tohoku University Graduate School of Medicine, 1–1 Seiryo-machi Aoba-ku, Sendai 980–8574, Japan
E-mail: kussie@emergency-medicine.med.tohoku.ac.jp
Journal Website (http://www.nms.ac.jp/jnms/)

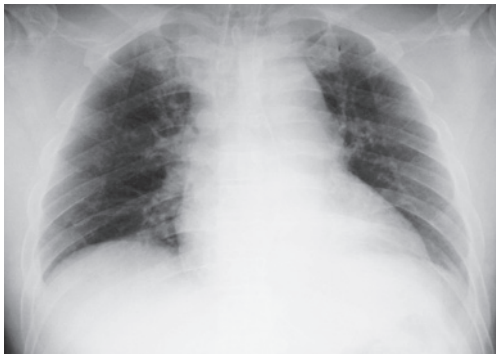

Fig. 1 Chest X-ray on admission shows mediastinal widening and an obscure aortic knob suggestive of thoracic aortic injury, without marked deviation of the trachea or the nasogastric tube. No rib fractures or hemopneumothorax is visualized.

contusion and pelvic fracture requiring surgical fixation. Although acute aortic dissection is an uncommon presentation of blunt injuries of the thoracic aorta and the poor prognosis has not been adequately emphasized[10], close observation, to evaluate the dissection and to determine whether splanchnic ischemia has developed, is recommended for patients for whom delayed definitive repair or nonoperative management has been selected.

Case Report

An inebriated 57-year-old man was struck by a car moving at moderate speed. He was transported to our hospital within 30 minutes of the accident. On arrival in the emergency department, the patient had a blood pressure of 108/68 mm Hg, a heart rate of 78/minute, a respiratory rate of 10/minute, and a Glasgow Coma Scale score of 7 (E1; V1; M5). He had contusions and excoriations on his face and in the right flank to the iliac region. The right elbow and thigh showed large contusions. No abnormal physical findings were evident on chest examination. Focused assessment with sonography for trauma (FAST) was negative. The hemoglobin concentration was 12.2 g/dL, the base excess was -3.1, and the serum lactate level was 40 mg/dL. After endotracheal intubation because of consciousness disturbance (Glasgow Coma Scale score of 7), plain X-ray were obtained, which showed pelvic fracture, right tibia plateau/fibula fracture, and mediastinal widening suggestive of thoracic aortic injury without marked deviation of the trachea or the nasogastric tube (**Fig. 1**). CT scans of the head and neck without contrast enhancement and of the chest to the pelvis with intravenous contrast enhancement confirmed nasal bone fracture, traumatic subarachnoid hemorrhage, lung contusion, multiple pelvic fractures, including of the right acetabulum, and a retroperitoneal hematoma. There was a small pseudoaneurysm in the lesser curvature of the distal aortic arch with a small mediastinal hematoma, indicative of thoracic aortic injury (**Fig. 2**). No findings suggesting visceral ischemia were apparent on this study.

The patient was admitted to the intensive care unit and received mechanical ventilatory support, and direct traction of the right leg was performed for the pelvic acetabular fracture. While open surgical repair and endovascular stent grafting were considered, primarily nonoperative management of the thoracic aortic injury under blood pressure control was selected because of 1) the relatively small aortic tear with a small mediastinal hematoma, 2) the unstable respiratory status that necessitated mechanical ventilation, 3) the need to avoid systemic heparinization for the control of pelvic bleeding, and 4) the need to avoid, because of the acetabular fracture, the right lateral position needed during surgery.

On day 1 after admission, the patient's hemodynamic and respiratory conditions remained stable despite a decrease in the serum hemoglobin level to 9.3 g/dL in the absence of blood transfusion. However, the serum lactate level increased from 15 to 74 mg/dL. On day 2, the serum lactate level increased to 131 mg/dL with metabolic acidosis despite the absence of any apparent hemodynamic deterioration or further decrease in the hemoglobin level. Furthermore, the serum levels of aspartate aminotransferase (AST), alanine aminotransferase (ALT), lactate dehydrogenase (LDH), and creatinine phosphokinase (CK) increased from near the normal ranges to 15,726 IU/L, 4,255 IU/L, 18,558 IU/L, and 2,066 IU/L, respectively. CT scans from the chest to the pelvis obtained after intravenous contrast enhancement to re-evaluate the thoracic aortic injury and the splanchnic perfusion revealed acute aortic dissection from the distal arch to the infrarenal abdominal aorta, with no evidence of aortic rupture (**Fig. 3**). The CT images also showed 1) the celiac and superior mesenteric arteries were

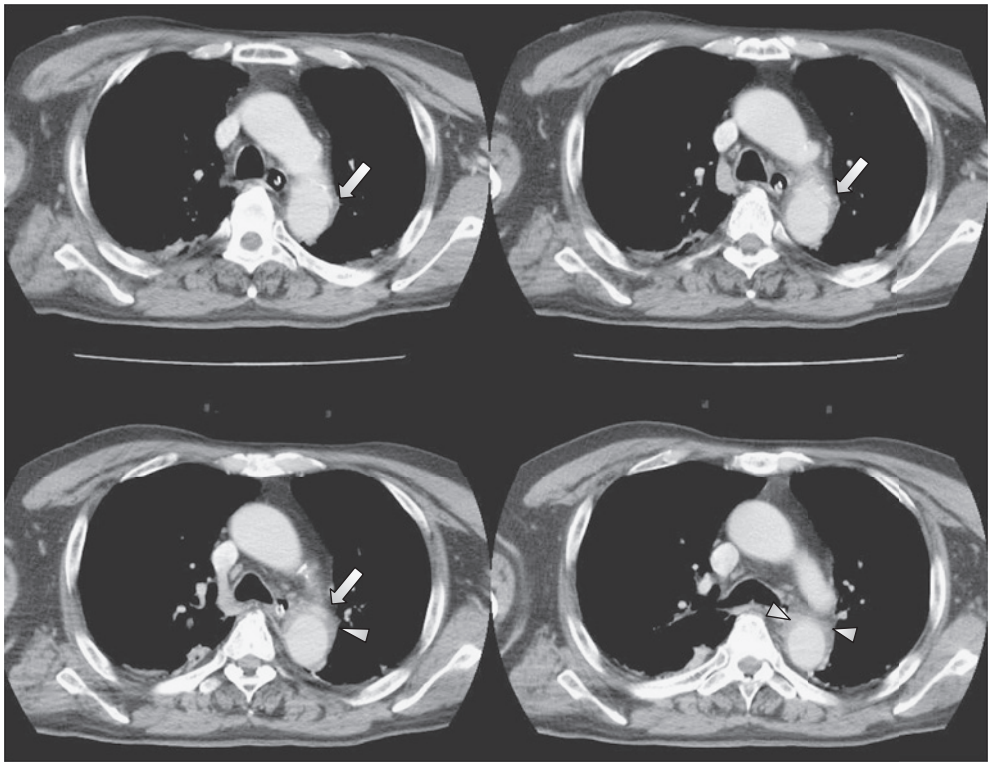

Fig. 2 Initial contrast-enhanced CT of the chest shows a small pseudoaneurysm in the lesser curvature of the distal aortic arch **(arrows)**, with a small mediastinal hematoma **(arrowheads)**, indicative of thoracic aortic injury.

supplied from the false lumen, associated with thrombotic obstruction within several centimeters from their origins, 2) marked compression of the true lumen by the false lumen due to the aortic dissection, and 3) patchy contrast enhancement of the hepatic parenchyma and poor-to-no enhancement of the small intestinal wall with narrowing superior mesenteric venous caliber, suggestive of hypoperfusion of these organs **(Fig. 3)**.

Because splanchnic ischemia, including ischemia of the liver and, especially, the small bowel, was strongly suggested by the CT findings and laboratory data, we performed exploratory laparotomy to evaluate the mesenteric perfusion and to remove the ischemic bowel, if the ischemia was found to be irreversible. On exploration of the peritoneal cavity, the hepatic parenchyma appeared mottled because of the ischemic damage, and the small bowel was also ischemic from the jejunum to the terminal ileum, with irreversible changes of the ileum **(Fig. 4)**. Although resection of the small bowel showing irreversible ischemic changes was performed without reconstruction, progressive multiple organ failure developed, and the patient died on postoperative day 2.

Discussion

The diagnosis and management of blunt injuries of the thoracic aorta have undergone significant changes over the last decade[1,4]. The most impressive changes in diagnosis during the past decade are: 1) the nearly complete replacement of diagnostic aortography (classical diagnostic "gold standard") by CT and the absolute elimination of transesophageal echocardiography as a method for definitive diagnosis[1], and 2) the establishment of CT as a first-line screening modality, which has resulted in the earlier and more frequent diagnosis of thoracic aortic injuries, even of minimal aortic tears with small mediastinal hematomas, as negative X-ray findings were reported in 7% of patients with aortic injuries in the first prospective study performed by the American Association for the Surgery of Trauma[4]. Along with the liberal use of helical CT with multiplaner reconstruction, which represents a great advance in CT technology, a multicenter prospective study by the American Association for the Surgery of Trauma[1] revealed that the use of diagnostic aortography has declined from 87% of

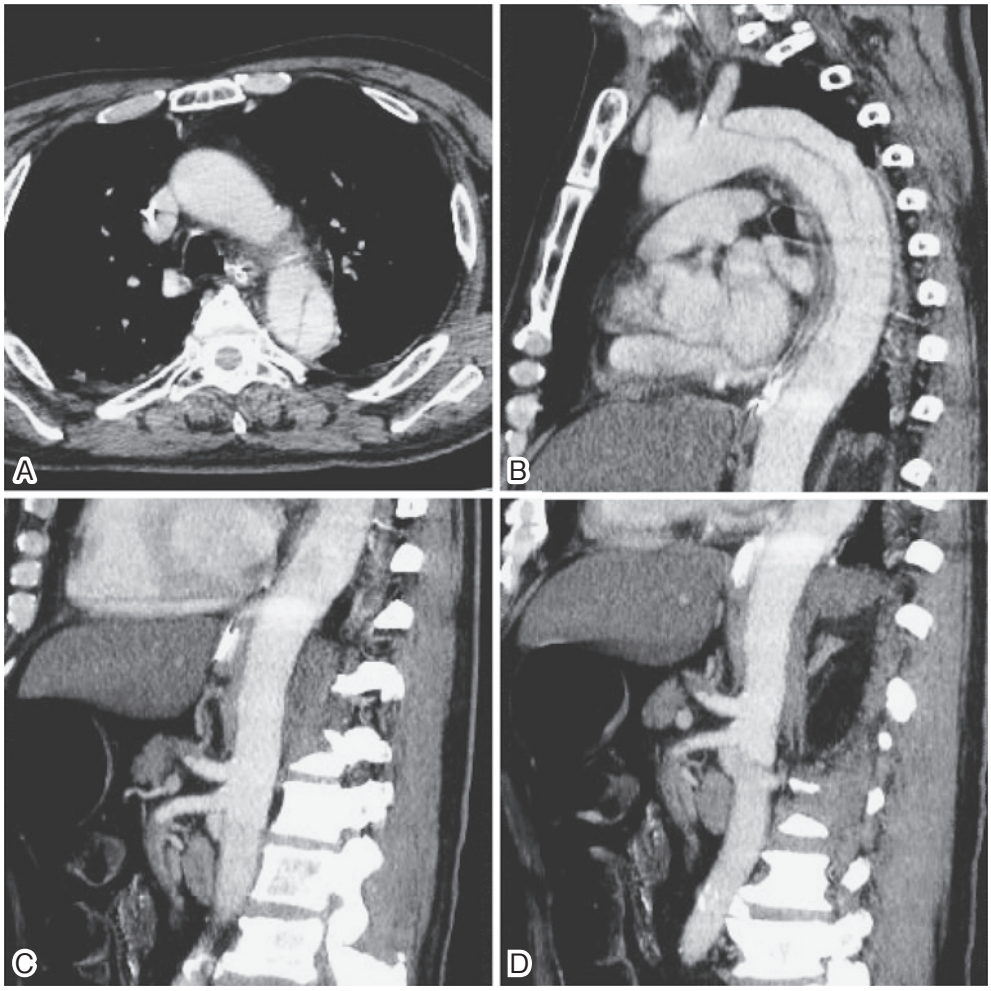

Fig. 3 Follow-up contrast-enhanced CT demonstrates acute aortic dissection from the distal arch to the infrarenal abdominal aorta, with no evidence of aortic rupture (**arrows** indicating the dissection and false lumen). CT also shows: 1) the celiac and superior mesenteric arteries being supplied from the false lumen, with thrombotic obstruction within several centimeters from their origins (**arrowheads**), and 2) marked compression of the true lumen by the false lumen due to dissection.

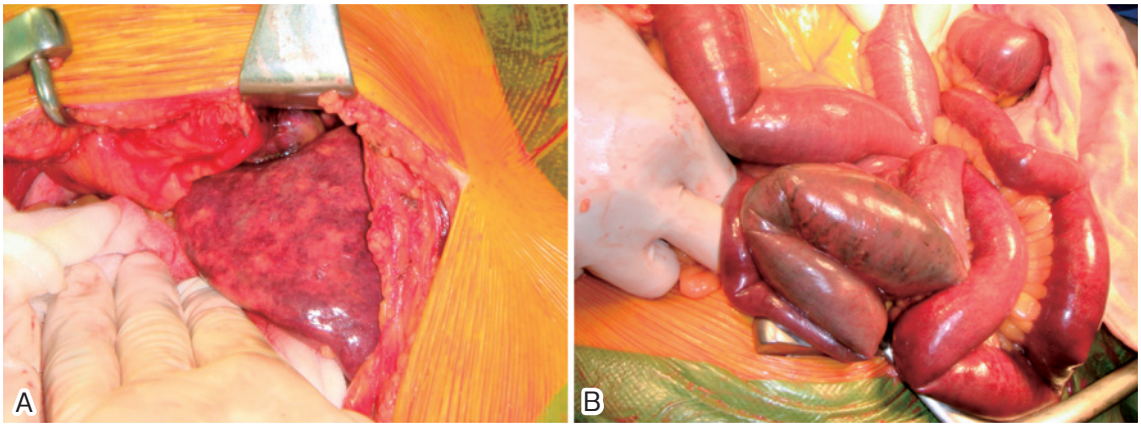

Fig. 4 Intraoperative findings show a mottled appearance of the hepatic parenchyma caused by the ischemic damage (A), and the small bowel was ischemic from the jejunum to the terminal ileum, with irreversible ischemic changes of the ileum (B).

cases in 1997 to only 8% of cases in 2007.

The most striking changes in the definitive management of thoracic aortic injuries are the major shift from open surgical repair to endovascular stent graft repair, with significant reductions in the rates of overall mortality and procedure-related paraplegia in the second study period[1,2]. Although endovascular stent graft placement was initially considered the procedure of choice for the definitive management of thoracic aortic injuries in high-risk patients with multiple trauma or elderly patients, it has become the procedure of first choice even for low-risk and young patients at many trauma centers in the United States[2].

The third important change in the management of traumatic aortic injury is that the introduction of strict blood pressure control using antihypertensive drugs, such as beta blockers, has reduced the risk of in-hospital free rupture[7,11]. In selected cases, the delay of definitive repair to when more optimal conditions were present reduced mortality[8,12–14]. Moreover, the concept of nonoperative management in selected high-risk, elderly patients with small aortic tears has shown encouraging results in early exploratory studies[5,15] and has been suggested to be a reasonable alternative treatment in carefully selected patients. Although nonoperative management by means of blood pressure control was selected in our case because of the small aortic tear with a small mediastinal hematoma and other conditions, immediate surgical or endovascular treatment should be considered when the aortic dissection is evident even if it is minor and localized.

Acute dissection of the aorta is an uncommon presentation in patients with blunt chest trauma[10,16–18], and trauma has been reported as an infrequent cause of this condition[19]. Few case series and case reports have been published[16–18,20], and "acute dissection" has not been classified as a type of injury in the Abbreviated Injury Scale, 1990 revision[21]. While the outcome of aortic dissection due to blunt chest trauma is not always favorable, the poor prognosis associated with extensive dissection resulting in splanchnic ischemia has not been adequately emphasized.

The mechanisms underlying the development of blunt injuries of the thoracic aorta have not been defined. It has been suggested, however, that the aorta is subjected to 2 principal forces: torsion and shearing. Torsion caused by rapid deceleration produces a pressure wave of blood through the aorta. Shearing associated with sudden impact is transmitted in a radial direction. These forces are concentrated on the segment between the relatively mobile portion and fixed portion of the thoracic aorta, resulting in transverse injury to the aortic isthmus, which is the most vulnerable anatomic site to this injury[10,22]. Although rupture or pseudoaneurysm formation by the adventitia and surrounding mediastinal tissues due to transverse laceration at the level of the isthmus is the most common presentation and lesion following blunt chest trauma, the aortic injuries may be partial thickness, histologically an initial tear of the intima, and similar to that in nontraumatic aortic dissection, possibly extending to the media and the adventitia[10]. Furthermore, aortic injuries at the level of the isthmus may be associated with dissection, and localized subadventitial and medial dissection can occur[20]. During open surgical repair of this injury, transverse laceration of the intima/media is larger than the adventitial laceration and is almost always accompanied by local dissection of the intima/media proximally and distally from the site of injury. It has been recommended that the proximal and distal ends of the aorta should be completely transected and placed away from the esophagus during open surgical repair; this maneuver allows full-thickness suturing[10]. According to these findings and recommendations, aortic dissection may occur in patients with blunt aortic injuries treated nonoperatively or awaiting delayed definitive repair, although the risk of extensive dissection accompanied by splanchnic ischemia may not be high.

Although early diagnosis and immediate intervention are imperative in patients with visceral ischemia due to aortic dissection, reliable diagnostic markers or findings to predict the condition have not been proposed. Molecular markers, such as intestinal fatty acid binding protein[23], and dynamic CT may have a role in early diagnosis, although their sensitivity and reliability have not been established.

In conclusion, although minimal injuries with small mediastinal hematomas can be diagnosed with the liberal use of CT, and delayed definitive repair under strict blood pressure control or nonoperative

management can be performed safely in carefully selected patients, acute aortic dissection can occur owing to blunt thoracic aortic injury in patients selected for nonoperative management. Acute aortic dissection is an uncommon presentation of blunt injuries of the thoracic aorta, and the poor prognosis has not been adequately emphasized. When aortic dissection, even if minor or localized, has been diagnosed with CT, it is important to bear in mind that it can become complicated by not only exanguinating rupture but also by splanchnic ischemia, as can nontraumatic dissection. Close observation to evaluate the dissection leading to splanchnic ischemia is recommended in patients selected for delayed definitive repair or nonoperative management.

Conflict of Interest Statement: All authors have no conflict of interest.

References

1. Demetriades D, Velmahos GC, Scalea TM, et al.: Diagnosis and treatment of blunt thoracic aortic injuries: changing perspective. J Trauma 2008; 64: 1415–1419.
2. Demetriades D, Velmahos GC, Scalea TM, et al.: Operative repair or endovascular stent graft in blunt traumatic thoracic aortic injuries: results of an American Association for the Surgery of Trauma Multicenter Study. J Trauma 2008; 64: 561–571.
3. Parmley LF, Mattingly TW, Manion WC, Jahnke EJ: Nonpenetrating traumatic injury of the aorta. Circulation 1958; 17: 1086–1101.
4. Fabian TC, Richardson JD, Croce MA, et al.: Prospective study of blunt aortic injury: Multicenter Trial of the American Association for the Surgery of Trauma. J Trauma 1997; 42: 374–383.
5. Hirose H, Gill IS, Malangoni MA: Nonoperative management of traumatic aortic injury. J Trauma 2006; 60: 597–601.
6. Caffarelli AD, Mallidi HR, Maggio PM, Spain DA, Miller DC, Mitchell RS: Early outcomes of deliberate nonoperative management for blunt thoracic aortic injury in trauma. J Thorac Cardiovasc Surg 2010 Jun 23. [Epub ahead of print]
7. Holmes JH, Bloch RD, Hall RA, Carter YM, Karmy-Jones RC: Natural history of traumatic rupture of the thoracic aorta managed nonoperatively: a longitudinal analysis. Ann Thorac Surg 2002; 73: 1149–1152.
8. Hemmila MR, Arbabi S, Rowe SA, et al.: Delayed repair for blunt thoracic aortic injury: is it really equivalent to early repair? J Trauma 2004; 56: 13–23.
9. Malhotra AK, Fabian TC, Croce MA, Weiman DS, Gavant ML, Pate JW: Minimal aortic injury: a lesion associated with advancing diagnostic techniques. J Trauma 2001; 51: 1042–1048.
10. Mattox KL, Wall MJ Jr, Lemaire S: Thoracic great vessel injury. In Trauma (Feliciano DV, Mattox KL, Moore EE, eds), 6th ed. 2008; pp 589–606, McGraw-Hill.
11. Fabian TC, Davis KA, Gavant ML, et al.: Prospective study of blunt aortic injury and helical CT is diagnostic and antihypertensive therapy reduces rupture. Ann Surg 1998; 227: 666–677.
12. Mattox KL, Wall MJ: Historical review of blunt injury to the thoracic aorta. Chest Surg Clin N Am 2000; 10: 167–182.
13. Maggisano R, Nathens A, Alexandrova NA, et al.: Traumatic rupture of the thoracic aorta: should one always operate immediately? Am Vasc Surg 1995; 9: 44–52.
14. Pate JW, Gavant ML, Weiman DS, Fabian TC: Traumatic rupture of the aortic isthmus: program of selective management. World J Surg 1999; 23: 59–63.
15. Santaniello JM, Miller PR, Croce MA, et al.: Blunt aortic injury with concomitant intra-abdominal solid organ injury: treatment priorities revisited. J Trauma 2002; 53: 442–445.
16. Gammie JS, Katz WE, Swanson ER, Peitzman AB: Acute aortic dissection after blunt trauma. J Trauma 1996; 40: 126–127.
17. Ono M, Yagyu K, Furuse A, Kotsuka Y, Kubota H: A case of Stanford type A acute aortic dissection caused by blunt chest trauma. J Trauma 1998; 44: 543–544.
18. Rogers FB, Osler TM, Shackford SR: Aortic dissection after trauma: case report and review of the literature. J Trauma 1996; 41: 906–908.
19. Wilson SK, Hutchins GM: Aortic dissecting aneurysms: causative factors in 204 subjects. Arch Pathol Lab Med 1982; 106: 175–180.
20. Perchinsky M, Gin K, Mayo JR: Trauma-associated dissection of the thoracic aorta. J Trauma 1998; 45: 626–629.
21. The Abbreviated Injury Scale 1990 revision, Association for the Advancement of Automotive Medicine, Des Plaines, IL.
22. Finkelmeier BA, Mentzer RM, Kaiser DL, Tegtmeyer CJ, Nolan SP: Chronic traumatic thoracic aneurysm: influence of operative treatment on natural history. An analysis of reported cases, 1950-1980. J Thrac Cardiovasc Surg 1982; 84: 257.
23. Evennett NJ, Petrov MS, Mittal A, Windsor JA: Systematic review and pooled estimates for the diagnostic accuracy of serological markers for intestinal ischemia. World J Surg 2009; 33: 1374–1383.

(Received, October 18, 2010)
(Accepted, November 20, 2010)