

Acute myocardial infarction with concomitant cardiac contusion: A case report

Hebat Allah A. Amin.^{1,*}, Heba A. Abdelal.², Dalia M. Aboufadel.³

^{1,2}Forensic Pathologist, Pathology Department, Egyptian Forensic Medicine Authority (EFMA), St. Berum El- Tonsi, Zeinuhum, Cairo, Egypt.

³Researcher, Pathology Department, Medical Division, National Research Centre, Cairo, Egypt.

*Corresponding Author:

E-mail: heba_forensicpathology_2912@hotmail.com; hebatallahamedamin@gmail.com

ABSTRACT:

The relative contributions of trauma and disease may become a challenging acute medico-legal problem. Discussed here a case of sudden death due to myocardial infarction, associated with an external chest injury. Routine histopathological as well as immunohistochemical examination of the tissue samples using cTnT, MB, desmin and caspase-3 was made to evaluate and detect the extent of the myonecrosis.

The presented case highlights the importance of detailed history by the medical examiner in cases of sudden death with associated injuries.

Keywords: Acute myocardial infarct; blunt chest trauma; cardiac contusion; Caspase-3; Desmin; Myoglobin; Troponins.

Access this article online	
Quick Response Code:	Website: www.innovativepublication.com
	DOI: 10.5958/2394-6776.2015.00012.0

INTRODUCTION

Cardiac contusion is the cellular damage resulting from a non-penetrating chest or abdominal trauma. Even an apparently mild, non-penetrating trauma can result in fatal myocardial contusion [1]. A blow or injury to the chest may precipitate myocardial infarction [2], or arrhythmia leading to death. Interpretation of these types of injuries is very crucial in determining the manner of death [3-4]. Cardiac injuries may result from one of these main mechanisms: a direct blow to the chest, in which the heart is compressed between the sternum and the thoracic vertebrae, sudden deceleration, striking compression of the abdomen, or secondarily from sternal or rib fractures [5]. Blunt chest trauma may result in various cardiac injuries including pericardial injury, valvular injury, great vessel injury, cardiac conduction system injury, cardiac rhythm disturbances, coronary artery injury, cardiac concussion cardiac contusion, cardiac rupture and acute myocardial infarction (AMI) [6-7-8].

Immunohistochemical staining for the markers cTnT, MB, desmin and caspase-3 can be considered reliable indicator of acute myocardial damage. Immunohistochemistry proved valuable in evaluating the extent of myonecrosis in cases of

cardiac contusion and AMI not detected by the conventional H&E staining [9-10].

CASE REPORT

The presented case belonged to a 58 years old male, with a history of verbal and physical conflict, and trauma. He was hit on his head, lost consciousness, and this was followed by death one hour after hospitalization. Gross autopsy findings included:

-Scalp: cut injury and bruises.

-Thorax: fracture 5th and 6th ribs.

-Brain: weighed 1300 gm. Cerebral hemispheres measured 17 x 14 x 10 cm while cerebellar hemispheres measured 11.5x 6 x 4.5 cm. Severe congestion was seen. White matter showed diffuse petechial hemorrhages. Middle cerebellar peduncle showed a haemorrhagic area 0.5x 0.5 cm [A gross picture suspicious for diffuse axonal injury (DAI)] [11].

-Heart: The heart grossly had a normal size (11 cm x 10 cm x 5 cm) and weight (227 g). The left main coronary artery arose normally. The left anterior descending and the left circumflex coronary arteries showed calcifications and severe stenosis. The atrio-ventricular and semilunar valves were normal.

The outer surface of the heart showed coup contre-coup injury, with minor superficial pericardial hemorrhages on both, the anterior and the facing posterior surfaces opposing the fractured ribs. The anterior half of the septum showed hemorrhages, its thickness was 0.5cm. The left ventricle was 1.4 cm thick while the right ventricle was 0, 4 cm thick.

HISTOLOGIC EVALUATION

The histological examination of the brain revealed arteriolosclerosis, petechial hemorrhages (old

and recent) and middle cerebellar peduncle capillary telangiectasia with surrounding old and recent hemorrhages, associated with a micro-infarction. Therefore, the grossly noticed hemorrhages were attributed to hypertensive etiology. Brain edema was not noticed. Thus the grossly suspected DAI was not supported microscopically (Bearing in mind that the trauma – death interval was less than 6 hours).

The histological examination of the coronary arteries sections' stained with routine hematoxylin-eosin stains revealed advanced atherosclerosis with calcifications, associated with severe stenosis involving the anterior descending and circumflex branches of the left coronary artery.

Examination of the anterior and posterior left ventricular wall samples with gross superficial hemorrhages, showed sub-epicardial adipose hemorrhages and localized foci of necrosis, with limited extension to the sub-epicardial region [fig 1a and 1b].

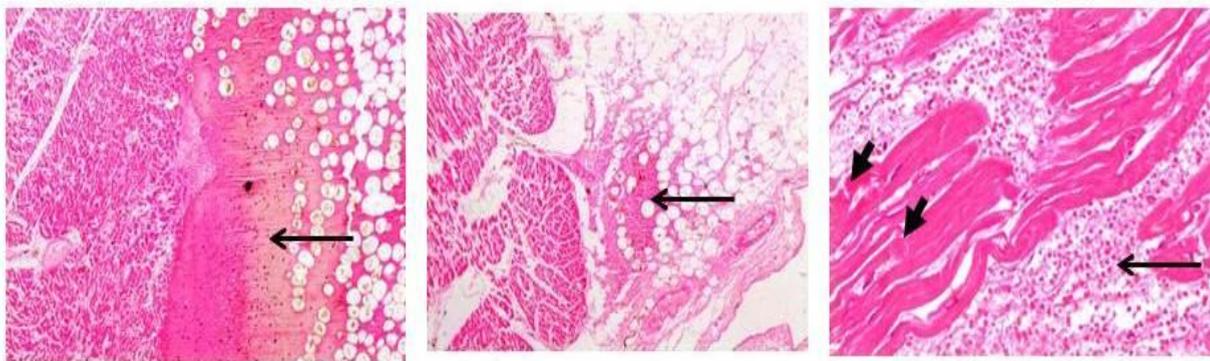
Examination of the anterior half of the inter-ventricular septum revealed wide contraction band necrosis (reperfusion necrosis), fragmentation of the

myocardial cells and diffuse interstitial hemorrhages [fig 1c]. No signs of old ischemic changes were noted - no foci of fibrosis.

Immunohistochemical examination of the left ventricular and septal samples using the four antibodies (cTnT, MB, desmin and caspase-3) was made to detect and evaluate the extent of the myonecrosis.

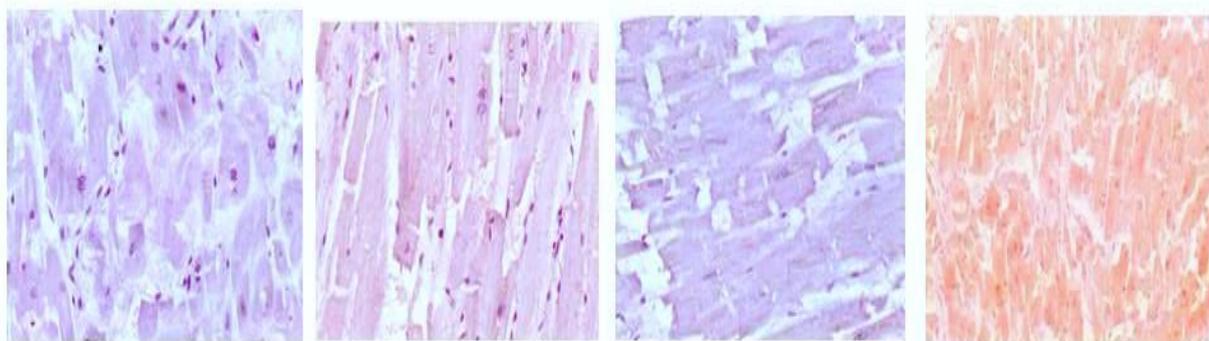
The results were as follows:

- Sections from the anterior left ventricle with pericardial hemorrhages showed moderate cTnT depletion and diffuse MB and desmin depletion, together with weak expression of caspase 3.
- Sections from the posterior left ventricle, with pericardial hemorrhages (contre coup) showed total, diffuse uniform depletion of cTnT, MB and desmin and moderate caspase 3 expression [fig 2a, b, c and d].
- Sections from the hemorrhagic anterior septal area revealed diffuse uniform depletion of cTnT, MB and desmin and moderate caspase 3 expression [fig 3a, b, c and d].



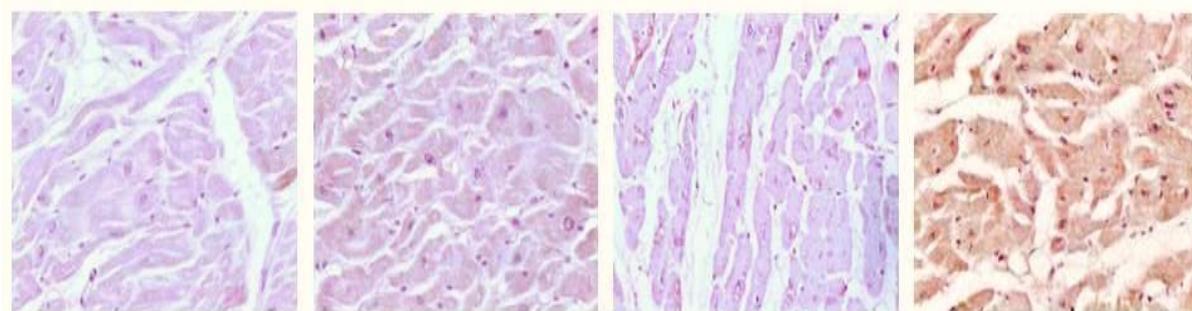
a) Pericardial hemorrhage -coup injury, (long thin arrow). (x100). **b)** Pericardial hemorrhage - contre coup, (long thin arrow), (x100). **c)** Anterior half of the inter-ventricular septum revealed wide contraction band necrosis) short thick arrow) and diffuse interstitial haemorrhages (long thin arrow), (x200).

Fig. 1, (Hematoxylin and eosin stain)



a) Diffuse depletion of cTnT. **b)** Diffuse depletion of MB. **c)** Diffuse depletion of desmin. **d)** moderate caspase 3 expression.

Fig. 2: Sections from the posterior left ventricle (contre -coup) - (cTnT, MB, Desmin and caspase 3 with Hematoxylin counterstain, x 200).



a) Diffuse depletion of cTnT. b) Diffuse depletion of MB. c) Diffuse depletion of desmin. d) moderate caspase 3 expression .

Fig. 3: Sections from the anterior septal area - (cTnT, MB, Desmin and caspase 3 with Hematoxylin counterstain, x 200)

DISCUSSION

One of the major dilemmas we face in the forensic practice occurs when a person with a pre-existing natural disease and a history of recent trauma decease. As the cause of death could be attributed entirely to the disease irrespective of the injury or could be caused entirely by the injury irrespective of the disease or maybe a combination of trauma and disease [12].

In the above mentioned case, the weight of the brain and the absence of evidences for edema exclude traumatic brain injury (TBI)[11]. Moreover, the grossly detected petechial hemorrhages were proven microscopically to be old and recent, altogether with the arteriolosclerosis, suggest a hypertensive etiology. The history of trauma and gross, and histological examination of the heart [13-14] led us to suspect cardiac contusion; However, the presence of advanced coronary artery disease and anterior septal hemorrhagic infarction with wide contraction band necrosis and diffuse interstitial hemorrhages, point to AMI [4].

The Contraction bands and waving detected indicate a sudden cardiac death [15]. Yoshida et al., in 1992 stated that myocardial contraction bands are markers for hyper-contraction and sympathetic nerve activation. However, the causality between extrinsic factors (e.g. violence, restraint, over-work) and sudden death should be judged [16].

Despite the advanced coronary disease, the case was compensated with no signs of old ischemia. However, after exposure to stress, conflict and apparently trivial trauma, decompensation occurred, in the form of AMI. Therefore, death in the case could be attributed to interaction between trauma and disease, with the conflict precipitating decompensation in an otherwise compensated person.

CONCLUSION

This case highlights the importance of detailed history taking by the medical examiners in such incidents and calls for open mindedness in the interpretation of circumstances of each single case. Accordingly, it is essential to consider all the associated circumstances before the examiners formalize their conclusions.

REFERENCES:

1. Irene Riezzo, Cristoforo Pomara, Margherita Neri, Giuseppina Rossi, Vittorio Fineschim. Cardiac contusion: Ending myocardial confusion in this capricious syndrome. *International Journal of Cardiology* 128 (2008), e107–e110
2. Mugadlimath, Mandar Sane, K. Yoganarasimha, Girish Kamath, Vishal Koulapur, K.U. Zine. Myocardial infarction and concurrent chest injuries: Two case reports. *Anand. Forensic Science International* 217 (2012) e1–e3.
3. Maron BJ, Poliac LC, Kaplan JA, Mueller FO.. Blunt impact to the chest leading to sudden death from cardiac arrest during sports activities. *N EnglJMed*(1995) 333:337-42.
4. Fineschi V, Baroldi G and Silver MD. Pathology of the heart and sudden death in forensic medicine. Boca Raton, FL: CRC Press (2006); p. 309–10.
5. Sato Y, Matsumoto N and Komatsu S. Coronary artery dissection after blunt chest trauma: Depiction at multi detector-row computed tomography, (2007). *Int J Cardiol.* 118:108–10.
6. Vougiouklakis T, Peschos D, Doulis A, Batistatou A, Mitselou A and Agnantis NJ. Sudden death from contusion of the right atrium after blunt chest trauma: case report and review of the literature, (2005). *Injury*; 36:213–7.
7. Darok M, Beham-Schmid C, Gatterinig R, Roll P. Sudden death from myocardial contusion following an isolated blunt force trauma to the chest, (2002). *Int J Leg Med.* 115:85–9.
8. Maenza RL, Seaberg D and D'Amico F . A meta-analysis of blunt cardiac trauma: ending myocardial confusion, (1996). *Am J Emerg Med*; 14:237- 41.
9. Hebat Allah A. Amin, Ahmed M. Y. El-Hennawy, Gina A. A. Nakhla b, Sahar A. Tabak, Hanan H. Hassan.

- “Immunohistochemistry in the Detection of Early Myocardial Infarction” (A Post-Mortem Study), *Egyptian Journal of Forensic Sciences*, (2011). 1, 5–12; DOI:10.1016/j.ejfs.2011.04.003
10. Hebat Allah A. Amin, Heba A. Abdelal , Amany A. Shabaiek, Ghada S. Osman, Amany A. Mousa. “A Comparative Study of Immuno-Histochemical Markers in the Detection of Early Myocardial Infarction (Autopsy Study)”. *Egyptian journal of pathology*. July, 2015, vol.35, 1: 76-80. DOI: 10.1097/01.XEJ.0000465414.47169.95
 11. Hebat Allah A. Amin. “Neuropathological Examination in forensic Autopsy. Edition: First, Publisher: Faculty of Commerce – Males, Cairo (2007). Chapter 3, p.63-67. DOI: 10.13140/RG.2.1.1009.7361
 12. Saukko P. and Knight B. “Knight’s forensic pathology, third edition; Arnold, London, 2004. Chapter 25, p.495.
 13. Peter J, Kirchner A, Kuhlisch E, Menschikowski M, Neef B, Dressler J. The relevance of the detection of troponins to the forensic diagnosis of cardiac contusion. *Forensic Sci Int* 2006; 160:127–33, e110 I.
 14. Mahajan N, Mehta Y, Rose M, Shani J and Lichstein E. Elevated troponin level is not synonymous with myocardial infarction, 2006. *Int J Cardiol*; 111:442–9.
 15. Yoshida K, Ogura Y and Wakasugi C., 1992. Myocardial lesions induced after trauma and treatment. *Forensic Sci Int*. 54:181–189.
 16. Baum VC. The patient with cardiac trauma, 2000. *J Cardiothorac Vasc Anesth*. 14:71–81.