Correlation of Histological Changes in Cardiac Muscle with the Severity of Impact

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I. INTRODUCTION

Vehicular crashes are a common cause of blunt cardiac injury. Mechanism and severity of impact affect the nature of cardiac injury which ranges from mild contusion to rupture of the cardiac wall [1]. Rupture of the heart is a rare phenomenon and only found in fatal cases through autopsies. Compression to the chest can also result in cardiac injuries without any visible mark of bony fracture [2]. In this work an attempt has been made to invesitgate the microstructural changes in cardiac muscles during compressive injuries and derive an injury risk function using logistic regression based on post impact histopathological observations and determine the extent of microstructural injury / damage correlation of the cardiac muscle in conjunction with impact velocity.

II. METHODS

Compressive impact tests were conducted on 26 isolated goat heart samples at velocity varying from 1 to 3 m/s using an electromechanical impact gun [3] following which the samples were fixed in 10% formalin solution for 48 hours and histopathological analysis done thereafter. Overall dimensions and shape of the samples were noted before and after fixation. A rigid cylindrical impactor was used to induce blunt trauma to the samples. Imapct speed was decided based on the range of speed experienced by the heart in impact simulations using the GHBMC model. A visual examination was performed for presense of superfical damage such as discoloration of tissue, accumulation of blood in tissue etc.

Three rectangular sections, one from the impacted area, Left ventricle (IA(LV)), adjacent area NIA(LV) and non impacted area of the Right Ventricle (NIA(RV)), were extracted from the sample for microscopy. Physical and chemical state of sectioned tissue was preserved using 10% formalin. After this fixation process, tissue was submerged into methanol of varying concentrations for dehydration. Dehydrating fluid was replaced using xylene, as a clearing agent. Molten paraffin wax was poured into a mould to surround a clear section of tissue. After solidification, the wax provides a support matrix that allows thin sectioning of the order two microns in a microtome. The sectioned tissue ribbon was transferred into warm water where the tissue was scooped up onto a glass slide. Since most cells are transparent and colourless, hematoxylin and eosin were added to stain the micro slide for microscopic analysis Figure 1 (a) & (b).

III. INITIAL FINDINGS

Superficial damage was identified by visual examination and underlying deep damage was subsequently investigated by histopathology. As samples were obtained post-mortem and did not contain any blood, post impact external / superficial changes such as damage to the blood vessel, contusion of cardiac muscle, etc., were not discernable in the surface examination. Penetration type of damage such as lacerations were also absent, as organs had been tested in isolation and did not have any bony parts.

The damage considered for cardiac muscle through histopathology is tabulated in Table 1. The damage was classified as normal- 'score 0', mild- 'score 1' and 'score 2', moderate- 'score 3' and severe-'score 4'. Scores were assigned to every case based on the histological findings. Multiple damage instances were also seen in some cases and correspondingly multiple scores have been assigned to these cases. The frequencies of occurrence of score 0, 1, 2 are high in in IA(LV), NIA(LV) and NIA(RV) as compared to a score of 4 (Figure 1). Using logistic regression analysis [4], the probability of having injury score \geq 1, score \geq 2, score \geq 3 and score \geq 4 were obtained as a function of the velocity of impact. The bar plot in Figure 1 (c) gives the probability of injury of

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different severity levels at different impact speeds. For instance, it can be seen that the probability of having an injury with score \geq 3 and score \geq 4 at 3 m/s is 79% and 46% respectively. This data available is limited by the impact speed being in the range of 1 to 3 m/s. Beyond this range of impact velocity, the injury risk curve has been extrapolated assuming a logistic distribution and shown in Figure 1(d).

			Cooro	Damaga
	S.	Features	Score	Damage
	No.			Classification
		Normal	0	Normal
	1	Myocardial eosinophilic changes	1	Mild
	2a	Cell coalescence with maintenance of cell borders,	2	Mild
Due to impact		Approximation, nuclei approximation without overlapping		
	2b	Cell coalescence with overlapping of cells, nuclei overcrowding	3	Moderate
	3	Myocardial/ pericardial tear, disintegration of peripheral cells	4	Severe
Prior	1	Inflammation –focal mild	1	Mild
Pathological		Moderate	2	Mild
condition		Severe	3	Moderate
	2	Fibrosis	3	Moderate

TABLE I CLASSIFICATION OF DAMAGE IN CARDIAC MUSCLE

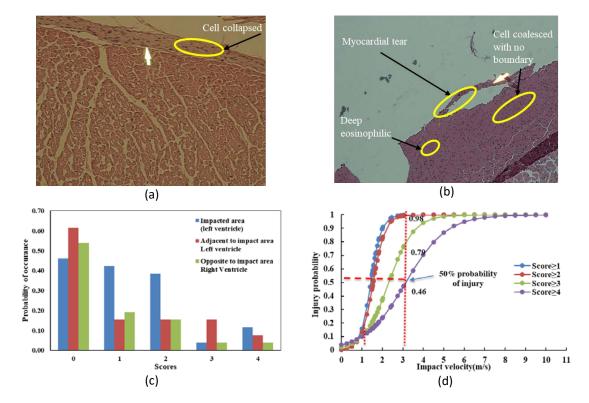


Fig. 1. (a) Stained microscopic slide of collapsed cell, (b) coalesced cell with no boundary, (c) Probability of occurrence vs score, (d) Variation of Probability of injury with impact velocity.

IV. DISCUSSION

A methodology to correlate the microstructural damage with the severity of impact for heart tissues has been proposed. A method to generate injury risk curves, indicating the probability of varying levels of damage, has been developed based on post impact histological assessment. It can be seen (Figure 2) that 50% probability of score \geq 1 and score \geq 2 lies at impact speeds of the order of 1.5 m/s, whereas 50% probability of score \geq 3, and

score \geq 4 lies at speeds of 2.4 m/s and 3.05m/s respectively. As an extension to this work, it is proposed to also develop correlation between injury at microstructural level and stress-strain data from FE simulations.

V. REFERENCES

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