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Microspical Picture of Microcirculatory Disturbances in Crush Syndrome Experimental Modelling

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Abstract

The aim of the study was to research adrenoreceptors sensitivity and microvascular diameter changes in several severity of crush syndrome.Experiments were administered on 50 white rats (200- 250 gr) with the utilization of crush syndrome modeling classical methods in various periods of compression (3 hours, 6 hours) and decompression (1 hour, 6 hours). Microhemocirculation and microvascular adrenoreactivity were studied in rats' intestine mesenteric arterioles by biomicroscope "Nikkon Labopot". Microvascular adrenoreactivity was studied by pharmacological analysis, concretely, with the utilization of epinephrine on the idea of α- or β-adrenal receptors blockers action.Numerous microhemocirculatory disturbances were revealed in mesenteric arterioles It is evident due to following changes in microhemocirculation: microvessels diameter changes, adrenoreactive structures dysfunction. Crush syndrome causes suppression of adrenoceptor sensitivity of mesenteric arterioles towards adrenaline which may be explained by stress induced hypercatecholaminemia, which is extremely apparent in decompression period. Vascular tone practically remains without adrenal control, which negatively affects the regional blood flow to the organs We can assume that severity of the changes correlates with compression and immensely on decompression period duration.

Objective:

To explore the diagnostic value of quantitative contrast-enhanced (CE) ultrasonography for crush injury in the hind limb muscles of rabbits.

Methods:

A complete of 120 New Zealand white rabbits were randomized to receive compression on the left limb for either 2 h (n = 56) or 4 h (n = 56) to induce muscle crush injury. Another eight animals weren't injured and served as normal controls. CE ultrasonography parameters like peak intensity (PI), ascending slop, descending slop and area under curve (AUC) were measured at 0.5, 2, 6 and 24 h and three , 7 and 14 days after decompression.

The rabbits were anesthetized with pentobarbital 30 mg kg−1 injected into the outer ear vein. the overall anaesthesia was maintained by injecting 10 mg kg−1 of pentobarbital every 2 h throughout the compression process. The left hind limb was shaved and bound up with a cotton pad (5 × 25 cm), then covered by a balloon cuff. The balloon cuff was connected to a pressure meter and aired to take care of a pressure of 40 kPa for two or 4 h within the two experiment groups. Two ear vein catheters were placed for CE ultrasonography and blood collection, respectively.

Results:

Compared with the uninjured muscles, reperfusion of the injured muscles showed early and high enhancement in CE ultrasonography images. The time-intensity curve showed a trend of rapid lift and gradual drop. The PI and AUC values differed significantly among the three groups and were positively correlated with serum and tissue biomarkers. Rabbits of the 4-h compression group showed significantly higher PI and AUC values, and serum and tissue parameters than the 2-h compression group at each time points. Crush injuries were successfully created in the two compression groups. Among the 112 rabbits that underwent crush injury, 8 died from overdose of anaesthetics or complications of muscle injury within 14 days and 104 survived until their sacrifice. The eight rabbits that died were not included in the statistical analysis.

Conclusion:

CE ultrasonography can effectively detect muscle crush injury and monitor dynamic changes of the injured muscles in rabbits. PI and AUC are promising diagnostic parameters for this disease. during this study, higher enhancement and better intensities were found by CE ultrasonography within the injured muscles than within the normal muscles. The microbubbles vanished slower within the injured muscles than within the normal muscles. The time–intensity curve showed a trend of rapid lift and gradual drop. it had been suggested that the blood volume was increased within the injured muscles compared with the traditional muscles. and therefore the washout of microbubbles from the injured muscles was slower than that from normal muscles.

There are several possible reasons for the CE ultrasonography findings during this study. First, the prolonged ischaemic injury led to metabolic product accumulation within the injured muscles. This resulted in metabolic regulation and opening of microvessels to clear off these products Second, the venules were damaged and occluded by the accumulated blood cells and microthrombi within the injured muscles. But the arteries were less affected. This resulted in obstructed blood flow and

fewer affected blood perfusion, resulting in congested microcirculation within the injured muscles. Third, damaged endothelial cells and increased vascular permeability caused the microbubbles to exudate into the tissue space. There are limitations during this study. First, the rabbit limb muscle accounts for a way lower proportion of the entire body than that of humans. Hence, caution should be used when extrapolating these

results to clinical applications. Second, the severity of muscle injury during this study wasn't adequate for observing muscle perfusion. Third, just one crush injury was created in each animal. This doesn't reflect the truth in natural disasters that crush injuries are commonly combined with fracture and arterial injuries.

In conclusion, CE ultrasonography can effectively detect muscle crush injury and monitor the dynamic changes of this disease process. PI and AUC are promising diagnostic CE ultrasonography parameters. CE ultrasonography might play an important role in the pre-hospital and bedside settings for the diagnosis of muscle crush injury.

Compared with a previous study by Lv et al, this study examined the effects of two different compressions on muscles. The observation time was 14 days, which was much longer than the previous 72 h. In addition, we examined more biochemical and CE ultrasonography parameters and therefore obtained a better understanding of the pathology of muscle crush injury.

Biography

Natalia Pavliashvili has completed his PhD at the age of 29 years from Tbilisi State Medical University (TSMU). She is Associate Professor of Pathophysiology Department of TSMU and . She has published more than 80 papers in reputed journals, is an author of 3 elective programs and 2 textbooks in Pathophysiology. She is head of Educational Programs Management and Assessment Department. She is a member of British Microcirculation Society. She has supervised 7 PhD Candidates and has been Scientific Secretary for more than 20 PhD thesis defense. She is a peer reviewer more than 15 PhD research programs.

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