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Original Contribution

The diagnostic value of serum pentraxin 3 levels in pulmonary contusion

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ABSTRACT

Purpose: To investigate the difference in pentraxin 3 (PTX 3) levels between patients with pulmonary contusion and healthy volunteers.
Materials and methods: This study was conducted with a group of 20 trauma patients diagnosed with pulmonary contusion and 30 healthy individuals enrolled as a control group in a tertiary university hospital.
Results: Median PTX 3 levels were 7.05 (3.29–13.1), ng/ml in the contusion group and 1.03 (0.7–1.58) ng/ml in the control group. PTX 3 titers were significantly higher in patients with pulmonary contusion compared to those of the control group ($p < 0.001$). An area under the curve (AUC) value of 0.968 investigated using ROC analysis to determine the diagnostic value of the PTX-3 in pulmonary contusion patients was measured. A PTX-3 cut-off value of 2.06 produced 95.5% sensitivity and 86.7% specificity.
Conclusion: PTX 3 levels in pulmonary contusion increased significantly compared to the healthy control group. If supported by wider series, PTX 3 may be expected to be capable of use as a marker in pulmonary contusion.

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1. Introduction

Pulmonary contusion is a common and significant cause of mortality and morbidity, particularly in cases of blunt thoracic trauma [1]. In addition to local injury in the lung in pulmonary contusion, systemic inflammatory response is also triggered. Oxidative injury also accelerates the destructive process [1-3].

Pentraxins are a multi-functional protein superfamily involved in inflammatory response. Circulating levels of pentraxin 3 (PTX 3), one of the main acute phase reactants, can increase up to 3–5 times above basal values in inflammatory conditions. PTX 3 is produced in the inflammation site and binds immediately to the endothelium. PTX 3 levels are thought to be an independent marker of inflammatory processes [4].

Lung injury associated with pulmonary contusion is frequently observed in severe traumas, particularly chest traumas. Endothelial dysfunction occurs with capillary leakage in the pulmonary parenchyma. Post-traumatic proinflammatory response (such as burns, surgical interventions, soft-tissue damage and thoracic injury) increases alongside a decrease in microcirculation. Various cytokines, such as IL-1, IL-6, IL-8 and TNF-α, are released in association with trauma, tissue injury and cell death, and these trigger PTX 3 release by stimulating endothelial cells, muscle cells, fibroblasts, adipocytes and chondrocytes. PTX 3 further increases the release of cytokines such as IL-1, IL-6, IL-8 and TNF-α by stimulating pro-inflammatory cytokines and complement activation (Fig. 1). Respiratory distress and hypoxemia associated with this develop due to pulmonary contusion-induced pulmonary edema. When pulmonary contusion is accompanied by acute respiratory distress syndrome, mortality levels reach 56–76% [5-8]. Post-traumatic proinflammatory response increases with a decrease in pulmonary microcirculation.

In the light of this information from the literature, acute inflammatory response plays a significant role in the pathophysiology of pulmonary contusion. PTX 3 levels might thus be anticipated to increase, as an inflammatory marker, in pulmonary contusion, an acute inflammatory process. This study was intended to investigate whether or not PTX 3 increases in patients with pulmonary contusion.

2. Materials and methods

This research was performed as a prospective clinical study. Patients presenting to the Karadeniz Technical University (KTU) Medical Faculty

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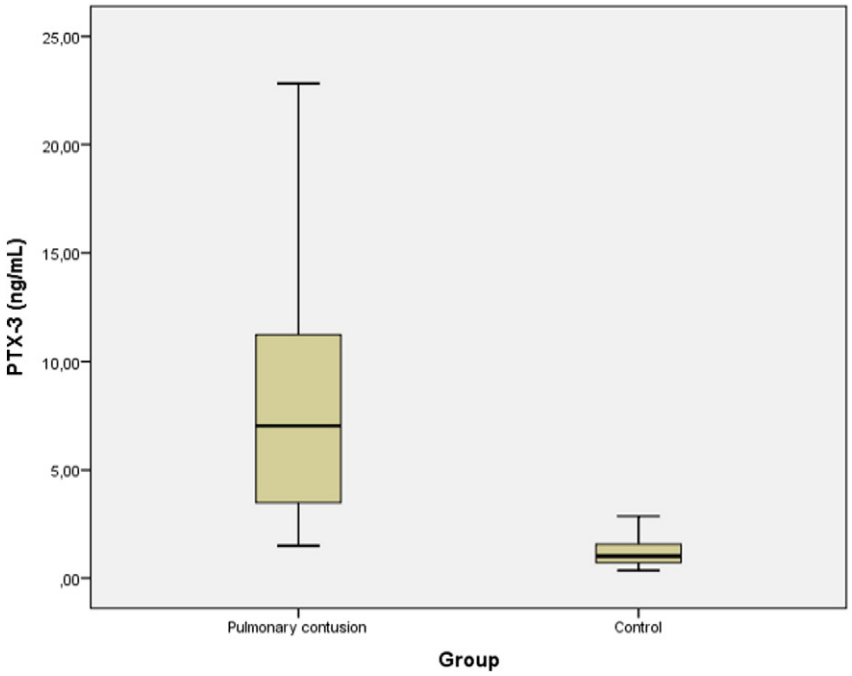

Fig. 1. A comparison of serum PTX 3 levels in the pulmonary contusion and control groups.

Emergency Medicine Department and diagnosed with pulmonary contusion within 6 months of receipt of KTU Medical Faculty Ethical Committee approval (Approval no:2016-122) were included in the study. Twenty patients diagnosed with pulmonary contusion in the emergency department and 30 healthy volunteers were enrolled.

Patients presenting to the emergency department with isolated thoracic trauma and aged over 18 were included. Subjects with non-thoracic trauma, sepsis, vasculitis, burns or a recent history of surgery or refusing to participate were excluded. Various clinical and demographic characteristics of the patients enrolled, such as symptoms, physical examination findings, X-ray and computerized tomography (CT) findings were recorded onto study forms.

As much serum specimen as the vacuum would permit was placed into CBC tubes containing EDTA in order to measure PTX 3 levels at time of presentation. Following centrifugation for 10 min at 3000 rpm, plasma was separated and stored at −80 °C.

2.1. Determination of plasma pentraxin 3 levels

PTX 3/TNF-inducible gene 14 protein (TSG-14) levels in human plasma were determined using enzyme-linked immunosorbent assay (ELISA) kits (R&D Systems, Cat No: DPTX30, Lot: 334734, Minneapolis, USA) in line with the manufacturer's recommendations. Plasma samples stored at −80 °C were brought to room temperature. Into all streptavidin-coated wells was added 200 mL PTX 3 biotinylated antibody. This was then incubated for 60 min in a microplate shaker. At the end of this period, in order to ready the plates for use, each plate was washed with 300 mL washing buffer to remove unbound antibodies. PTX 3/TSG-14 standards were prepared in line with the kit procedures. Standards, controls and specimens were activated with 30-min pretreatment with D solution. Into each plate well was added 100 mL Assay Diluent RD1-56 solution. Next, 20 mL from the pre-treated standards, controls and specimens was added to this solution and incubated for 120 min at room temperature in a microplate shaker. Following incubation, the plate was washed four times with plate buffer with a plate washer. Next, 200 μL PTX 3 conjugate was added to each well and incubated for 120 min at room temperature in a microplate shaker. Following incubation, the plate was washed four times with plate buffer with a plate washer. Subsequently, 200 μL of TMB substrate solutions was added to each well for coloring and left to incubate for 30 min at room temperature. At the end of 30 min, 50 μL of stop solution was added to each well, and the color of the specimens was observed to turn to yellow. Absorbances of specimens were measured at a wavelength of 450 nm on a VERSA (designed by Molecular Devices in California, USA) microplate reader. A standard chart was prepared using absorbance values obtained against standard concentrations (Fig. 2). PTX 3 in specimens were calculated as ng/mL using this

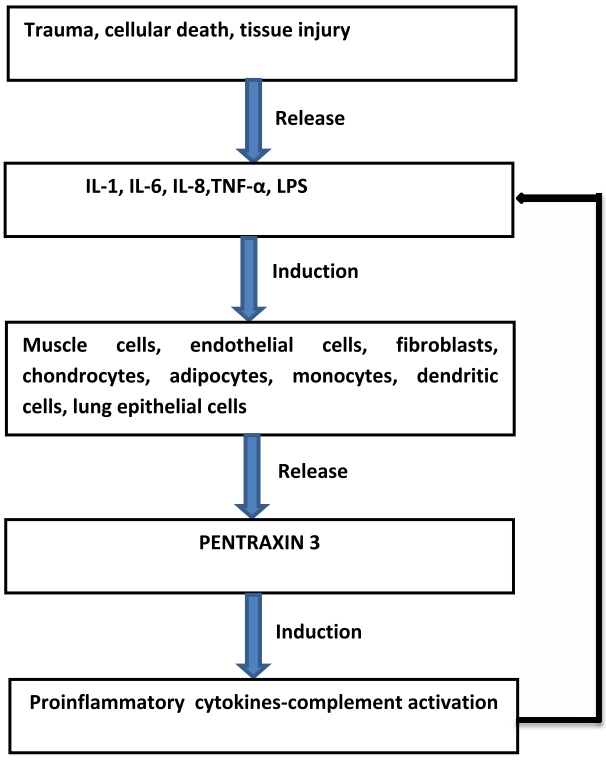

Fig. 2. Chart showing PTX 3 interaction in trauma. IL = interleukin, TNF = tumor necrosis factor, LPS = lipopolysaccharide.

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