majority of NRK dispensed. Given the interdisciplinary approach and support from many professional and governmental agencies for improved access to this life-saving antidote, very few challenges exist that should restrict adoption of this program.

## Source of support

None.

## Prior presentations

Poster Presentation. American College of Clinical Pharmacists (ACCP) Annual Meeting 2017. Phoenix, AZ.

Kevin A. Kaucher, PharmD
*Department of Pharmacy, Denver Health Medical Center, 777 Bannock St. MC 0056, Denver, CO 80204, United States*
Corresponding author.
*E-mail address:* kevin.kaucher@dhha.org (K.A. Kaucher).

Nicole M. Acquisto, PharmD
*Department of Pharmacy, University of Rochester Medical Center, 601 Elmwood Ave., Box 655, Rochester, NY 14642, United States*
*Department of Emergency Medicine, University of Rochester Medical Center, 601 Elmwood Ave., Box 655, Rochester, NY 14642, United States*

Kerry B. Broderick, M.D.
*Department of Emergency Medicine, University of Colorado, 12401 E. 17th Avenue, Aurora, CO 80045, United States*
*Denver Health Medical Center, 777 Bannock Street, MC #0108, Denver, CO 80204, United States*

5 December 2017

https://doi.org/10.1016/j.ajem.2017.12.020

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## A novel biochemical marker for predicting the severity of ACS with unstable angina pectoris: Asprosin

Ischemic Heart Disease (IHD) is the leading cause of death worldwide, accounting for 12.7% of global mortality [1]. Acute Coronary Syndrome (ACS) covers a wide spectrum of clinical conditions ranging from unstable angina to Non-ST Elevation Myocardial Infarction (N-STEMI) and ST Elevation Myocardial Infarction (STEMI). New treatments and management guidelines emerge for the care of patients with ACS; risk stratification is the cornerstone in the initial evaluation of these patients [2]. That inflammatory mediators play a role during the evolution of ACS is indicated by the widespread coronary inflammation found during unstable angina pectoris (UAP), throughout the entire coronary artery bed, and in the extent that ACS outcome is related to a concurrent inflammatory response [3,4]. Circulating asprosin, a protein hormone, responds to low dietary glucose by triggering the release of liver glucose stores, and the reduction of asprosin protects against the hyperinsulinism associated with metabolic syndrome [5].

Asprosin has been identified as a novel hormone enriched in white adipose tissue and is pathologically increased in insulin-resistant mice and humans and asprosin concentrations have been shown to be increased in adults with Type 2 Diabetes Mellitus (T2DM).The previous study suggests that asprosin might serve as a risk factor associated with the pathogenesis of T2DM [6]. The aim of the present study was to compare the asprosin level and syntax scores in the prediction of the severity of ACS with UAP at the Emergency Department (ED).

The study group was formed of patients that were over 18 years of age, with a diagnosis of UAP who presented in ED and adult patients who experienced UAP in ED. The patients included in the study were all diagnosed with unstable angina pectoris. Coronary angiography were applied to patients with suspected ACS. Blood samples were taken from brachial veins of both the study and control groups into empty vacuum tubes to measure asprosin levels. Serum samples were obtained after centrifugation and the samples were stored at −80 °C until the day of serum asprosin analysis. Blood samples were collected in 2 ml EDTA tubes and analyzed using an automated hematology analyzer (XT-2000İ; Symex, Osaka, Japan) on the first day of presentation for both the study and control groups. To measure the asprosin levels, the blood samples of the study and control groups were collected in empty vacuum tubes which were initially covered with gel. After the blood coagulated in the tubes, they were centrifuged at 1200 g and 3000 rpm for 10 min, and then the upper remaining serum parts were collected into Eppendorf micro centrifuge tubes (Eppendorf AG, Hamburg, Germany) and stored at −80 °C. When patient enrollment for the study was completed, asprosin levels were measured with the micro ELIZA method as described by the manufacturer (Abbexa Co., Ltd., UK). Statistical analyses were made using SPSS 23.0 software (SPSS Inc., Chicago, IL, USA). The Student's *t*-test was used to compare mean values and the Spearman and Pearson correlation tests were applied for correlation analyses. Simple correlation analyses were performed to investigate the association of serum asprosin levels with the Syntax Score. A value of $p < 0.05$ was considered statistically significant.

The syntax scoring system was used to assess angiographic vessel-specific disease severity [7]. The patients ranged in age from 39 to 83 years old and 15 of them were male (Table 1). The Asprosin levels were found to be 7.84 ± 6.57 at the time of admission and 9.21 ± 12.71 at 24 h after coronary angiography. Results of the Paired *t*-test comparison of asprosin levels on admission to ED and at 24 h after angiography showed a significant difference

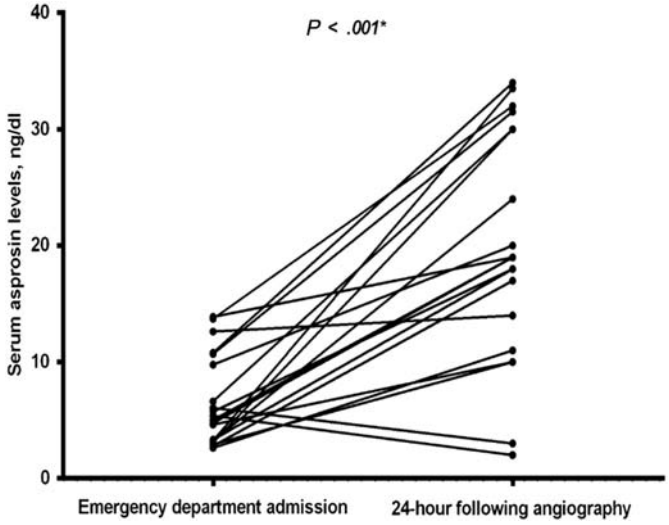


**Fig. 1.** The changes in asprosin levels.

**Table 1**
Demographic and laboratory characteristics of the subjects

| Variables | Unstable angina pectoris $n = 22$ |
|---|---|
| Age, years | 60.27 ± 10.67 |
| Sex, F/M | 7/15 |
| Neutrophil | 4.31 ± 1.57 |
| Lymphocyte | 2.35 ± 0.94 |
| RDW, % | 13.30 (12.80–14.00) |
| Syntax score | 19.00 (13.25–30.37) |

**Table 2**
Correlation coefficient between Δ asprosin and clinical parameters.

| | Δ asprosin | |
|---|---|---|
| | r | *P* |
| Age | −0.264 | 0.236 |
| Neutrophil | −0.127 | 0.573 |
| Lymphocyte | −0.106 | 0.638 |
| RDW | −0.205 | 0.359 |
| Syntax score | 0.486 | 0.022* |

between the measurements ($P < 0.001$) (Fig. 1), r: Spearman's correlation coefficient. A *P* value of <0.05 was considered significant (*). Δ Asprosin: asprosin level 24 h after angiography-ED admission level. Δ Asprosin showed a significantly positive correlation with the syntax score ($r = 0.486$, $P = 0.022$) (Table 2).

The syntax score is a scoring system that estimates the anatomical extent of coronary artery disease (CAD) [7]. Changes in the asprosin levels were compared with the syntax scores of the patients diagnosed with UAP and who underwent percutaneous coronary intervention (PCI). In our study; the alteration in asprosin levels both showed a significantly positive correlation with the syntax score ($r = 0.486$, $P = 0.022$). Asprosin can be a suitable marker for UAP for which there is no clinically useful marker as yet. It could also be used to guide therapeutic decisions and may improve current diagnostic strategies for UAP.

There is currently no marker, such as troponin etc., to predict the severity of coronary pathology in unstable angina pectoris. The results of this study demonstrate that asprosin could be used as a biomarker for UAP in the ED.

Ahmet Cagdas Acara, MD
*Bozyaka Research and Training Hospital, Department of Emergency Medicine, Izmir, Turkey*

Mustafa Bolatkale, MD
*Medipol University Hospital, Department of Emergency Medicine, Istanbul, Turkey*
Corresponding author.
*E-mail address:* dr.mustafa46@hotmail.com.

İlker Kızıloğlu, MD
*Çiğli State Hospital Department of Surgery, Izmir, Turkey*

Ersin İbişoğlu, MD
*Medipol University Hospital, Department of Cardiology, Istanbul, Turkey*

Çağdaş Can, MD
*Merkezefendi State Hospital, Department of Emergency Medicine, Manisa, Turkey*

4 December 2017

https://doi.org/10.1016/j.ajem.2017.12.032

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## Dangerous weapons confiscated after implementation of routine screening across a healthcare system

Healthcare workers are at an increased risk for workplace violence [1-3]. Workplace violence, as defined by the National Institute for Occupational Safety and Health, are "violent acts, including physical assaults and threats of assault, directed toward persons at work or on duty." The highest number of such assaults annually in the US are directed against healthcare workers. In an anonymous survey of 11,000 hospital workers, 39% experienced patient-related violence and 38% of victims reported fear for safety [4]. Under reporting of these events is common [5,6].

Violence within the ED is also well known, and contributes to overall higher risk of violence faced by healthcare workers [2,7]. This violence can impact employee well-being as well as patient and visitor safety. Emergency personnel may be at significant risk [8,9].

The United States Department of Labor Occupation Safety and Health Administration (OSHA) recommends screening for conventional weapons, as well as restricting items that can be used as weapons to prevent workplace violence [1]. Screening for dangerous weapons upon ED entry is important for security, as it is the point of entry for many patients/visitors into the hospital. As a multi-hospital healthcare system, we had a unique opportunity to evaluate the effects of screening in different ED settings.