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Editorial

Acil tıp, bir çok yönüyle tıbbın diğer alanlarından farklı özellikler gösterir. En zor branşların başında gelmektedir. Değerlendirmelerin bütünsel olması zorunluluğunun yanında, zamanla yarış acil müdahalenin önemli bir niteliğidir. Acil tıp geniş spekturumunda en zor hasta grubu da kritik hasta grubudur. Bir ülkenin sağlık sisteminin en önemli başarı göstergesi kritik hastalarda ulaştığı bakım kalitesidir. Bu zor hasta grubunda başarıya ulaşmak çok önemli bir bilgi birikimini, tüm donanımları içeren üniteleri ve ciddi bir ekip çalışmasını gerektirir. Bu yüzden kritik hasta bakımının gerekli düzeylere ulaşması sağlık sisteminin en önemli hedeflerinden biri olmalıdır.

Üçüncü sayısını çıkardığımız dergimiz bu hedeflere hizmet edecek önemli bir köşe taşıdır ve geleceğe doğru emin adımlarla ilerlemeye devam edecektir. Bu sayımızla ilk yılı geride bırakmış ve uluslar arsı indekslerde yerini almıştır. Dergimizin birinci yılında konuya ilgi gösteren arkadaşlarımıza ,yayın sürecine destek veren tüm ekiplere ve hakemlerimize teşekkür ediyoruz. İkinci yılımızda daha büyük hedeflere doğru hep birlikte yürümeye devam edeceğiz. Gelecek sayılarımızda buluşmak temennisiyle…

In many different aspects of emergency medicine, it shows different features than other fields of medicine. It is one of the most difficult branches. In addition to the necessity of evaluations to be holistic, the race over time is an important feature of emergency response. In the broad spectrum of emergency medicine, the most difficult patient group is the critical patient group. The most important indicator of success of a country's health system is the quality of care in critically ill patients. Achieving success in this difficult group of patients requires a significant knowledge, units with all the equipment and a serious team work. Therefore, achieving the necessary levels of crirtic patient care should be one of the most important goals of the health system.

The third issue of our journal is an important cornerstone to serve these goals and will continue to move forward confidently. This issue left behind the first year and took its place in international indexes. In the first year of our journal, we would like to thank our friends who showed interest in the subject, all the teams and referees who supported the publication process. In our second year, we will continue to walk towards greater goals. We hope to meet you in our next issues…

Tüm editöriyal kurul adına editör;

On behalf of the entire editorial board;

Prof. Dr. Başar Cander

Contents

Review Article
Eurasian Journal of Critical Care

Discussion of Mechanical Chest Compression Device Usage in Cardiac Arrest Cases in Hospitals in Light of Recent Literature

Yahya Kemal GÜNAYDIN, Miraç ALTUN, Dilber ÜÇÖZ KOCAŞABAN
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Abstract

The most important goal of cardio-pulmonary resuscitation is to provide adequate blood flow to brain and heart by return of spontaneous circulation and this can be performed by chest compressions in cardio-pulmonary resuscitation. The most important fact for providing the best circulation is immediate, fast, continuous and high quality chest compression in 2015 American Heart Association (AHA) guideline. Therefore various mechanical chest compression devices are developed recently for more efficient chest compressions and entered the clinical usage. These devices may be considered for use in situations such as prolonged CPR, low number of rescuers, hypothermic cardiac arrest, ambulances in motion, extracorporeal CPR, during angiography which makes it difficult to perform high quality CPR. Apart from these conditions, mechanical chest compression devices should not be preferred to manual chest compression in in-hospital cardiac arrest cases.

Keywords: Cardiac Arrest, CPR, Device

Introduction

Mortality rate of cardiac arrests in hospital is pretty high.[1,2] While survival rates vary between 5% to 50% and varying degrees of brain damage occur in more than half of the patients who can manage to survive.[3-5] The most important goal of cardio-pulmonary resuscitation is to provide adequate blood flow to brain and heart by return of spontaneous circulation and this can be performed by chest compressions in cardio-pulmonary resuscitation. During chest compressions, the intra-thoracic pressure increases and this provides the blood to circulate through the heart. In the decompression phase intra-thoracic pressure decreases and provides return of blood to the heart.[6]

In most of the societies, the progress of the interventions after cardiac arrests cannot provide favorable outcomes or they can only provide restricted recoveries.[7,8] Lack of experience and ability with inadequate chest compressions are the most important reasons of CPR failure in many studies that investigate the reasons. Even well performed chest compressions cannot provide coronary and cerebral perfusion as good as spontaneous circulation.[6] In another study, it was shown that even a 1 minute long CPR on a mannequin causes severe fatigue. Also the rate of accurate and proper CPR decreases by time.[9] The most important fact for providing the best circulation is immediate, fast, continuous and high quality chest compression in 2015 American Heart Association (AHA) guideline.[10] Therefore various mechanical chest compression devices are developed recently for more efficient chest compressions and entered the clinical usage. In this section we aimed to inform about the CPR performed by mechanical chest compression devices in cardiac arrest cases occurred in the hospital with regards to recent knowledge. We aimed to give recent information by considering guidelines that published in last years, randomized controlled clinical and experimental studies.

Mechanical Chest Compression Devices:

Active Compression- Decompression CPR and Impedance Threshold Device

ACD-CPR is performed by manually pump that has penetration feature in mid 1/3 of the sternum. After compression phase, during the decompression phase device move away from the chest and let the blood flow back to the heart. Therefore cardiac output increases.[10]

Impedance threshold device is a valve controlled device that attached to the endotracheal tube or supraglottic airway. The device increases the negative intrathoracic pressure by limiting air flow to the lungs in decompression phase of CPR. Thus venous return to the heart and cardiac output in-

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creases. It does not prevent positive pressure ventilation and exhalation at the same time. When ROSC is achieved the device detached. It can be used individually or together with active compression-decompression CPR device.[10,11]

Coronary and cerebral perfusion increment by elevation of intrathoracic negative pressure in the decompression phase is determined in previous human and animal studies in which the CPR was performed by ACD-CPR and ITD.[12-14] Furthermore, this approach increases the 24 survival rates in clinical trials.[15,16] The study which Wik L et al that performed shows us ACD-CPR increases the short-term and long term survival rates significantly.[17] In another study there was not significant superiority at survival rates and ROSC between traditional CPR and ACD-CPR in cardiac arrest cases occurred in hospital. Only ACD-CPR caused less complications like costa fractures, hemothorax and pneumothorax.[18] But controversial conclusions are determined about ACD-CPR in various studies. Besides depending on rescuer and be obliged to change the rescuer frequently as traditional CPR are the negative sides of ACD-CPR.[19]

American Heart Association (AHA) 2015 guideline stated that there was only one poor quality randomized controlled trial about ACD-CPR and ITD. According the result of this study, routine usage of ACD-CPR and ITD was not recommended. However in the presence of educated crew and appropriate equipment the combined usage of traditional CPR and ACD-CPR should be considered (Class IIb, LOE C-LD). The routine usage of ITD in CPR was not recommended because lack of enough evidence on 2015 AHA guideline (Class III: No Benefit, LOE A).[10]

Mechanical Chest Compression Devices: Piston Device

Chest compressions performed by a piston which places onto the sternum and uses electric or gas. These compressions can be adjusted as fast as required. While some of these devices are designed with vacuum mechanism for active decompression after every compression, some of them not designed as this mechanism. The most frequent used one of these devices is LUCAS (Lund University Cardiac Assist System).[10]

LUCAS provides appropriate amplitude and appropriate rate of compression. The device works automatically and is not depended on rescuer. After compression provided the device gets the initial position which let the heart relaxation. There is no significant difference at short-term, long term survival rates and the neurological outcomes between manual traditional CPR and LUCAS in the randomized controlled studies.[20,21] There are another machines that work with similar mechanism. However there is no significant success on survival rates, discharge from hospital rate and good neurological outcomes in the studies which using these devices.[22,23] Three comprehensive randomized controlled trials show us the usage of LUCAS in cardiac arrests occurred in hospital had no significant effect on ROSC and also the usage of LUCAS may harm the patient. Also by taking the hospital discharge rates into consideration, it is found to be harmful when compared to traditional manual CPR.[24]

In 2015 AHA guidelines, the routine usage of these devices are not suggested as there is insufficient evidence. It is

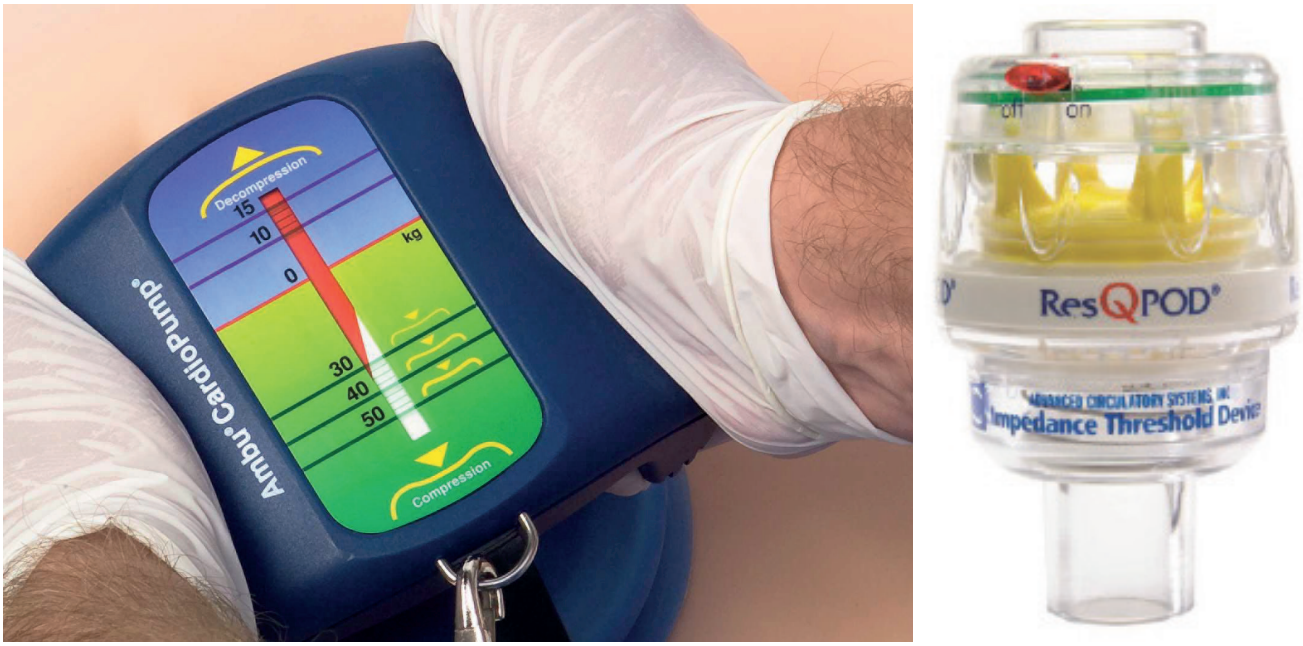

Figure 1. The compression and decompression process made by Cardiopump. (CardioPump ACD-CPR Device and ITD; ADVANCED CIRCULATORY SYSTEMS, INC. ; USA)

only suggested to be used when there is proper educated personnel (Class IIb, LOE B-R). Furthermore, the usage of mechanical chest compression devices with piston mechanism can be considered in situations where good quality CPR is not possible such as lengthened CPR, situations involving few rescuers, hypothermic cardiac arrest, ambulances on the move, during angiography and during preparations for extracorporeal CPR; but the chest compressions should not be interrupted during the placement of these devices. [10]

Figure 2. The Lund University Cardiac Assist System (LUCAS; Jolife, Lund, Sweden; distributed in the United States by Medtronic, Minneapolis, Minnesota, USA)

Load-Distributing Band Devices (LDB)

It is a mechanical chest compression device that is fixed on the backboard and works by surrounding the patient's chest either electronically or pneumatically. When the device was first entered usage, it was promoted as a very promising method. In a study conducted by Hock Ong et al., chest compressions done by LDB and traditional manual compressions were compared among 1011 in-hospital cardiac arrest cases. As a result they concluded similar results in the return of spontaneous circulation, but, the rates of hospital discharge and good neurological outcome results were found to be better in patients who received chest compressions via LDB.[25] However, in other clinical studies, in regards of 30-day survival rates and good neurological

Figure 3. AutoPulse Resuscitation System. AutoPulse and ZOLL are registered trademarks of ZOLL Medical Corporation in the United States.

outcomes, LDB was found to be inferior when compared to traditional manual CPR.[26-31] In 3 vast randomized controlled studies performed on in-hospital cardiac arrest cases, it is found that the usage of LDB does not provide a significant effect on the return of spontaneous circulation and may even cause harm. Also by taking the hospital discharge rates into consideration, it is found to be harmful when compared to traditional manual CPR.[24]

In 2015 AHA guidelines, the routine usage of these devices are not suggested as there is insufficient evidence. It is only suggested to be used when there is proper educated personnel (Class IIb, LOE B-R). Furthermore, the usage of mechanical chest compression devices with piston mechanism can be considered in situations where good quality CPR is not possible such as lengthened CPR, situations involving few rescuers, hypothermic cardiac arrest, ambulances on the move, during angiography and during preparations for extracorporeal CPR; but the chest compressions should not be interrupted during the placement of these devices.[10]

Conclusion

A recent extensive study on this subject is a meta-analysis published by Brooks SC et al. in 2014. This meta-analysis highlighted that there is not sufficient evidence for the mechanical chest compression devices to replace manual traditional chest compressions in cases of in-hospital cardiac arrest. Moreover, it is not proved to be superior to manual

CPR in aspects of return of spontaneous circulation, hospital discharge rates and good neurological outcomes.[32] Also in 2015 AHA guidelines, the usage of mechanical chest compression devices instead of traditional CPR methods is not suggested.[10] As a result, by considering all the data published in the recent years, in in-hospital cardiac arrest cases the mechanical chest compression devices should not be preferred over manual chest compressions.

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Original Article
Eurasian Journal of Critical Care

Exposure to Infrared Light: Case Series

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Abstract

Introduction : Infrared rays(IR) have been used in many areas in recent years and nowadays, many devices have been developed with the aim of heating in open,closed areas working with infrared technology.IR can cause retinopathy.They have the risk of developing periocular skin burns, corneal epitheliopathy,retinopathy.In this study, we wanted to talk about eight cases who were exposure with infrared light in a chemical manufacturing factory where Infrared light was used for drying and all of 8 patients had burning, stinging, watering,redness in the eyes.

Case: 8 men with ages between 18-33were admitted to the emergency department with burning, stinging, watering, redness in their eyes.It was learnt that it was started after taking a photo in daylight with infrared light that they used to dry the chemical materials at the factory where they were working. In the physical examination, there was watering,hyperemia in both eyes.Eye movements, pupil diameters,light reflexes were natural.Analgesic eye drops and saline irrigations were performed as a first intervention.Afterwards, the cases' eyes were closed with eye closing tapes in order to minimize the photosensitivity,ophthalmology consultation were requested.Analgesic drops were given after ophthalmology consultation and outpatient follow-up was planned,they were discharged.

Conclusion: The use of infrared-ray devices outside of the production purpose can cause eye irritation in the simplest form, as in our study.For this reason, having more warning instructions which are put by the production company in the manual will protect us from the many health problems, users must pay attention to these instructions before they use those device

Introduction

Infrared (IR) has been first discovered by Willian Herschel during 1800s[1]. The well recognized applications of IR are drying of the car paints in automotive industry and paper drying in paper industry. However, it is widely used including a simple light source and medicine[2]. Different from normal electric heaters, the products which operate through infrared system to heat the object by transmitting the heat by light have became popular during recent years[3].

We aimed to discuss eight patients who had watering, redness and sense of stinging and burning on the eyes after exposure to infrared light in a chemical manufacturing factory where infrared light is used for drying.

The Case

Eight male patients with age average between 18 and 33 years referred to emergency department because of burning, stinging, watering and redness on the eyes. Medical history of the patients revealed that the complaints started after taking a photo under day light within the infrared light that they use to dry chemical materials at the factory where they were working (Image 1). Physicial examination of the patients was normal. They had cooperation and orientation; watering and hyperemia were detected on both eyes (Image 2). Eye movements, pupil diameters and light reflexes were natural. Examination of the other systems was normal. Analgesic eye drops and saline irrigations were performed as a first intervention. Afterwards, the eyes of the cases were closed with eye closing tapes in order to minimize the photosensitivity, and ophthalmology consultation was requested. Analgesic eye drops were prescribed following consultation with ophthalmology department; follow-up on outpatient basis was planned and the patients were discharged.

Discussion

IR radiation is an electromagnetic energy and exists between visible light and microwave regions at electromagnetic spectrum[4-5]. IR zone defines the electromagnetic radiation between 1.8 and 3.4 µm wavelengths[6]. The IR lights are divided into 3 classes depending on the wavelength and emission temperature: 1-short wave or close IR zone, 0.72-2µm (3870-1180°C), 2-middle wave or moderate IR zone, 2-4

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Image 1.

Image 2.

μm (1180-450°C), 3-long wave or far IR zone, 4-1000 μm (<450°C)[7]. The most common IR applications include drying of the car paints in automotive industry and paper drying in paper industry. However, it is used within a wide range of processes including a simple light source and medicine[2].

Mori et al. detected in their study that[8] the heat increase on lacrymal glands and eyelids except cornea was significantly higher when compared with other conventional heaters. Takac et al.[9] started that UV lights shorter than 400 nm wavelength and long infrared lights may cause moderate to severe corneal burns. It was reported that chronic exposure to middle length infrared lights (1400-3000 nm) may cause cataract and carcinogenic effects. Use of adequate spectacles was addressed to be protected from such lights. At this point, our cases did not use any spectacles. Unsal et al.[3] Reported a case in form of eyelid burn due to exposure to infrared heater which is used for heating; however, our cases were injured at the workplace.

Conclusion

There is a gradual increase of infrared use at work and during daily life; the increase of incidence for use of these tools would cause an increase in burns involving sensitive organs such as eyes and in referrals to emergency department. Misuse of infrared light devices may simply cause irritation of the eye like our cases. Therefore, providing warning information and signs in the instruction manuals by the manufacturer companies and paying attention to such warnings would protect us from many possible severe health problems.

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Original Article
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The New Biomarkers Used in the Differentiation Between Transudate and Exudate Pleural Effusions

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Abstract

Etiological diagnosis of pleural effusion is sometimes easy and sometimes very difficult challenge. In most of patients with pleural effusion, the symptoms, signs and laboratory data are not pathognomonic for etiologic diagnosis. In a patient with an undiagnosed pleural effusion, the first question to answer is whether the fluid is a transudate or an exudate. This is usually determined by means of Light's criteria. In patients under diuretic treatment, Light's criteria misclassify transudates as exudates, but the pleural fluid NT-pro-BNP levels usually is above 1500 pg/mL in pleural effusions associated with heart failure.

Keywords: pleural effusion, new biomarkers, NT-pro-BNP

INTRODUCTION

Presented by Light et al.[1] to be used in the differentiation of exudates from transudates and also known as the Light's criteria, the criteria are still widely accepted and commonly used today although more than 40 years have passed since its definition. Light's Criteria: Exudative Effusions will have at least one or more of the following: Pleural fluid protein / Serum protein >0.5. Pleural fluid LDH / Serum LDH >0.6. Pleural fluid LDH > 2/3 Serum LDH Upper Limit of Normal. Despite the fact that the Light's criteria have been accepted as the first step towards the diagnosis of pleural effusions (PEs), concerns over the benefit of this approach have always been present since the creation of the criteria[2-5]. A few of the focus points of these concerns can be explained as follows:

1- Light et al.[1] reported the sensitivity of the criteria they defined in their original study as 99% and the specificity as 98% in determining exudates. In later studies, Light's criteria high sensitivity rates were supported but the specificity rates were emphasized to be lower than specified (65%-86%)[2,3]. Despite the superior differentiating power of the Light's criteria, it is known that approximately 20-30% of transudative PEs related to cardiac failure and cirrhosis were wrongly classified as exudates with the Light's criteria[6]. Majority of the cases falsely diagnosed as exudates were demonstrated to be patients receiving diuretic therapy, and diuretic therapy was shown to change the serum and PE biochemical parameters[7]. Is using biochemical parameters to differentiate between transudative and exudative PEs adequate and significant? What should be the primary approach if the clinical prediction contradicts with the biochemical diagnosis methods[8]?
2- Some etiological factors can cause both transudative and exudative PEs. In fact, the same patient sometimes may have two different concurring disorders. It is emphasized that in such cases, primarily clinical decisions should prevail biochemical approaches in differentiating between transudate and exudate[9].
3- Although rarely, transudative PE development or bloody transudative PE formation related to different pathologies is also possible in patients with malignancies. Because of the high LDH levels present in erythrocytes (containing LDH-1 isoenzyme), it is expected to raise the LDH levels in bloody transudative PEs and thus, the liquid can be wrongly classified as exudates (meeting exudate criteria)[10]. However, in a study where 23 patients with bloody PEs having an erythrocyte number of more than 100.000/mm^3 were enrolled, only a slight increase was demonstrated in the PE LDH-1 levels contrary to the expectations[11].
4- A threshold border level to be chosen for a test affects the test's sensitivity and specificity. Knowingly attempting to increase the sensitivity of a test will decrease its specificity and will start to give false positivity in more people. The sensitivity and specificity, and thus the false

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positive and false negative numbers of any test are dependent upon the cutoff value chosen to determine exudative PEs. If the cutoff value is chosen high, all transudates will be determined as true but if it is chosen low, then all exudates will be determined as true. Using this approach, Heffner et al.[12] analysed the data of a total of 1448 patients in eight studies and concluded that the best cutoff values were, respectively, 0.5 for protein rate, 45% of the serum upper limit of normal for PE LDH level and 0.45 for LDH rate. At the same time in this meta-analysis, the authors showed that only two or three combinations of pleural fluid parameters (e.g. combination of LDH and cholesterol or combination of LDH, cholesterol and protein) had diagnosis rates similar to the Light criteria without a need for blood samples and with a lower cost.

5- Tests used in the differentiation or diagnosis of PEs should be cost-effective. For this reason, many studies focused on making the Light criteria more cost-effective without decreasing the accuracy of the diagnosis. In the first studies on the subject, the measurement of PE cholesterol level alone was claimed to be used as an alternative to the Light criteria in the differentiation of transudates and exudates as a cost-effective test[13]. However, these dates could not be verified in the later studies[2,14]. Furthermore, studies on the measurement of PE cholesterol level are still ongoing today but none of the results obtained from the studies show consistency with one another. Hamel et al.[15] reported in their recent study that when the cutoff value for the PE cholesterol level was taken as >45 mg/dL, the sensitivity was 97.7% and the specificity was 100% in the differentiation of transudate-exudate. In a systematic compilation published a very short time ago, the most specific findings for exudate diagnosis were determined to be PE cholesterol level being >55 mg/dL, PE/serum cholesterol rate being >0.3 and PE LDH level being >200 U/L[16].

6- There is a need for new biomarkers that could be used in the differentiating diagnosis (e.g. malignant, tuberculosis, parapneumonic pleurisy) within the exudative PEs after the transudate-exudate differentiation; that could lead the way for diagnostic (e.g. pleura biopsy, thoracoscopy) or therapeutic (e.g. recurring thoracentesis, chest tube insertion) procedures; that could reveal the etiological cause in malignant PEs (malignant mesothelioma, lung cancer metastasis or metastasis from extrapulmonary malignancies); that could demonstrate early whether the non-purulent parapneumonic PEs are complicated or not.

In light of these controversial subjects, we will talk about new biomarkers proposed for the diagnosis of exudative PEs and presented as a contribution or an alternative to the Light criteria in the following parts of the article. Although there are dozens of new biomarkers used in studies related to PEs, unfortunately very few of them conform to the criteria of ideal biomarkers that could be used clinically, and primarily these will be discussed in this paper. An ideal biomarker is one that can easily be measured, that has a reasonable price that helps in decisions, that is repeatable and that gives the same results each time it is repeated[17].

New Biomarkers

While the Light criteria are widely used in clinical practice, it can particularly wrongly classify more than 25% of transudative bloody PEs, developing in patients receiving diuretic therapy due to cardiac failure[18]. After albumin and protein gradient calculations were started to use, the wrong classification of transudative PEs in patients receiving diuretic therapy was decreased. If albumin gradient (serum-PE albumin difference) is >1.2 g/dL or protein gradient (serum-PE protein difference) is >3.1 g/dL, PE is classified as transude. However, these gradient calculations should not be used as a starting parameter due to their low sensitivity. In PEs classified wrongly as exudates with the Light criteria despite the clinical situation supporting transude, calculation of albumin or protein gradients in PEs is recommended[2,19,20]. Bielsa et al.[6] reported that albumin gradients classified correctly more PEs compared to protein gradient (83% vs. 55% of wrong classifications). Nevertheless, in clinical practice it is recommended that first protein gradient is calculated and that if no result is obtained, albumin gradient is calculated or N-terminal pro-B-type natriuretic peptide (NT-pro-BNP) level is measured because protein level is measured at baseline due to the purposes of the Light criteria[10].

Natriuretic Peptides

Natriuretic peptides (ANP, proANP, BNP, NT-pro-BNP) are neurohormones used to help the diagnosis of cardiac failure and excreted by myocardium myocytes depending on the increased pressure or volume burden[21]. In clinical practice, while serum BNP level being lower than 100 pg/mL or NT-pro-BNP level being lower than 300 pg/mL excludes the diagnosis of cardiac failure, BNP level being higher than 500 pg/mL or NT-pro-BNP level being higher than 450-1800 pg/mL (depending on conditions such as threshold value age, gender, renal failure and increasing with age) supports cardiac failure diagnosis[22].

Increased ***NT-pro-BNP*** levels in PEs due to cardiac failure were first demonstrated in 2004 by Porcel et al.[23]. Many later studies also support the use of PE NT-pro-BNP level in determining PEs developing due to cardiac failure[24-26]. In three different studies, Porcel et al.[21] measured NT-pro-BNP levels in 150 PEs developing due to cardiac failure and in 158 PEs related to factors other than cardiac failure (58 ma-

lignant, 31 parapneumonic, 28 tuberculosis, 18 hepatic, 13 pulmonary embolism, 5 transudates with other reasons and 5 exudates with other causes). They reported that median NT-pro-BNP levels were significantly higher in PEs related to cardiac failure (6203 pg/mL) than PEs related to other causes (342 pg/mL). The best cutoff value for the diagnosis of PEs related to cardiac failure was determined to be 1300 pg/mL with ROC analysis and for this cutoff value, sensitivity was calculated as 93.3%, specificity as 89.9%, and the area under ROC curve as 0.96. If the cutoff value is taken as 1500 pg/mL, the sensitivity (91%) and the specificity (93%) of the test becomes more diagnostic for cardiac failure. Liao et al.[27] compared the NT-pro-BNP levels in ten patients, each with PEs related to cardiac failure, pulmonary thromboembolism, coronary artery bypass surgery and malignancy, and they reported that PE NT-pro-BNP levels were above 1500 pg/mL in all PEs related to cardiac failure and that they were below this level in all PEs related to other causes. In the meta-analysis of data from a total of 1120 PEs (429 developing secondary to cardiac failure and 691 developing due to causes other than cardiac failure) obtained from 10 studies, including the studies by Seyhan et al.[28] and Bayram et al.[29] from Turkey, Janda and Swiston[30] calculated the sensitivity of NT-proBNP as 94% (95% CI: 90-97), the specificity as 94% (95% CI: 89-97) and area under ROC curve as 0.98 (95% CI: 0.96-0.99). The authors stated in the conclusion of this meta-analysis that the best diagnosis threshold value for PE NT-pro-BNP was ≥1500 pg/mL. This threshold is widely accepted and commonly used today.

In a study by Cincin et al.[26], 8 of 21 PEs related to cardiac failure (38.1%) was wrongly classified as exudate. 5 of those were patients receiving diuretic therapy prior to thoracentesis. It was reported that PE NT-pro-BNP levels were significantly much higher in the ones wrongly classified as exudates (2024 pg/mL) than actual exudates (367 pg/mL). Porcel et al.[31] reported that 31 of 129 PEs related to cardiac failure (24%) were wrongly classified as exudates with the Light criteria, that NT-pro-BNP levels provided diagnosis accuracy in 27 of these 31 PEs (87%), that the diagnosis accuracy of protein gradient was 53% and of albumin gradient was 79%. That NT-pro-BNP levels were measured to be significantly much lower (551 pg/mL) in other conditions (6931 pg/mL) such as cirrhosis causing transudative PEs in another study by the same researchers brings forward NT-pro-BNP as a biomarker specific to PEs related to cardiac failure[23]. That there is a strong correlation between serum and PE NT-pro-BNP levels reiterates NT-pro-BNP being a good biomarker for cardiac PEs more. Bayram et al.[29] measured NT-pro-BNP levels in 133 patients and calculated the correlation covariance between serum and PE as 0.91. Similarly, four other studies on the subject support the strong correlation between serum and PE for NT-pro-BNP test, and the correlation covariance values in these studies vary between 0.90 and 0.95[26,31-33].

Another reason making NT-pro-BNP a more ideal biomarker for cardiac PEs is their superiority to BNP. Several studies investigated the diagnostical value of PE BNP in the differential diagnosis of PEs and compared it to NT-pro-BNP head-to-head. In the first study on the subject, BNP and NT-pro-BNP levels of 90 PEs related to cardiac failure and 91 PEs related to other causes were measured. When the cutoff value for PE ***BNP*** level in determining PEs related to cardiac failure was taken as >115 pg/mL, the sensitivity was calculated to be 74% and the specificity to be 92%. These values were lower values compared to PE NT-pro-BNP. Furthermore, area under ROC curve was found to be lower in BNP (AUC: 0.90) than in NT-pro-BNP (AUC: 0.96), and the correlation between BNP and NT-pro-BNP was shown to be weak (r=0.78)[34]. In another study conducted later, the facts that BNP (AUC=0.70) was a weaker test than NT-pro-BNP (AUC=0.84) in determining PEs developing due to cardiac failure and that there was a weaker positive correlation (r=0.57) between these two tests were supported[35]. In a recent study, Marinho et al.[36] investigated 34 PEs related to cardiac failure and 43 PEs related to other causes and reported that BNP levels were significantly much more higher in PEs developing due to cardiac failure (386 pg/mL) than PEs related to other causes (43 pg/mL). In this study, when the cutoff value for PE BNP level in determining PEs related to cardiac failure was taken as >127 pg/mL, sensitivity was calculated to be 97%, specificity to be 88% and AUC to be 0.98. Another superior aspect of NT-pro-BNP to BNP is the in-vitro stabilization process. NT-pro-BNP can remain stable in in-vitro environments after serum or PE sample is taken (1-2 hours) compared to BNP (20 minutes), which provides NT-pro-BNP with a measurement advantage and superiority[34].

The diagnostical value of the other two members of natriuretic peptide family, ***midregional proatrial natriuretic peptide (MR-proANP)*** and midregional ***proadrenomedullin (MR-proADM)*** in PEs developing due to cardiac failure was recently investigated by Porcel et al.[37]. The researchers measured the levels of MR-proANP, MR-proADM and NT-pro-BNP in a total of 185 PEs, 95 of which were related to acute decompensated cardiac failure and reported that the diagnostical value of MR-proANP was closer to NT-pro-BNP but the diagnostical value of MR-proADM was very low. In the diagnosis of PEs related to cardiac failure, when the best cutoff value for MR-proADM was taken as >2.5 nmol/L, sensitivity was 60%, specificity was 56% and AUC was =0.620; when the best cutoff value for MR-proANP was taken as >260 pmol/L, sensitivity was 84%, specificity was 83% and AUC was =0.918; and when the best cutoff value for NT-pro-BNP was taken as >1700 pg/mL, sensitivity was 92%, specificity was 82% and AUC was =0.935.

In summary, NT-pro-BNP, a member of natriuretic peptide family, is an ideal biomarker that could be used in determining whether the PEs that are wrongly classified with the Light criteria but clinically considered to be related to

cardiac failure are related to cardiac causes or not because of the following reasons: that it can differentiate cardiac-related PEs from exudates and other transudate causes, that it is superior to protein gradient and albumin gradient in differentiating PEs wrongly classified as exudates, that there is a strong positive correlation between the serum and PE levels of the test, and that it has a measurement advantage because it can stay for a longer time in an in-vitro environment compared to BNP. The best diagnosis threshold value that is widely accepted and commonly used for PE NT-pro-BNP today is ≥1500 pg/mL. BNP and MR-proADM, the other members of the family, have no diagnostical superiority to NT-pro-BNP. That MR-proANP has a close diagnostical value to NT-pro-BNP brings to mind the hypothesis that this test could be superior to NT-pro-BNP in distinguishing cardiac-related liquids from liquids of other nature in the future, and there is a need for new studies that are well-planned and that can verify or exclude this possibility.

Ischemia Modified Albumin

Ischemia modified albumin (IMA) is a new biomarker that is shown to be better than classical markers such as troponin and creatinine kinase MB in revealing ischemia and that is studied to be used in the early diagnosis of ischemic heart diseases. There are only 2 studies on the measurement of IMA concentration in PEs, both of which were conducted in Turkey and published recently. Both of the studies reported that there was an increased IMA concentration in transudative PEs and that IMA was a good biomarker to be used in the differentiation of transudates and exudates. The first study was conducted by Ozsu et al.[38], and this study investigated the IMA levels in PEs, 10 of which were related to cardiac failure and 30 of which were related to causes other than cardiac failure (10 pulmonary thromboembolism, 10 parapneumonic, 10 malignant). The researchers reported that IMA concentration was significantly higher in PEs related to cardiac failure than PEs related to other causes and that the sensitivity of IMA was 90%, specificity was 80% and area under ROC curve was 0.927. This study reported that there was not a strong correlation between serum and PE IMA levels (r=0.540) but measurement of IMA levels could help in differentiating cardiac-related liquids. In the second study by Dikensoy et al.[39], more PE cases were included (total 160 PE; 50 transudate and 66 exudate) and it was reported that IMA concentration was significantly higher in transudates (7986 ng/mL) than exudates (3376 ng/mL) and that when the cutoff value was taken as >4711 ng/mL, the sensitivity was 82%, specificity was 78% and area under ROC curve was 0.837 in differentiating between transudates and exudates. The study found no difference between the IMA levels of transudates related to cardiac failure and transudates related to other causes. There was no significant correlation detected between serum and PE IMA levels. While the results of both studies had no superiority to the Light criteria, the results indicate that IMA can be a candidate as a good biomarker.

Soluble Urokinase Plasminogen Activator Receptor

Another biomarker that was investigated to be used in differentiating cardiac-related PEs from PEs related to other causes is soluble urokinase plasminogen activator receptor (suPAR). suPAR is actually a newly discovered inflammatory biomarker, and the only study published on its diagnostical value in cardiac PEs was conducted by Ozsu et al[40]. In the study that included 18 PEs developing due to cardiac failure and 56 PEs developing due to other causes, it was detected that suPAR was significantly lower in cardiac-related PEs (11.8 [5.4-28.9] ng/mL) than PEs of other causes (26.7 [8.2-102.8] ng/mL) and that when the cutoff value for suPAR level was taken as ≥17.6 ng/mL to exclude the causes other than cardiac failure, sensitivity was 88%, specificity was 83% and AUC was 0.878.

Others

Apart from these biomarkers, the following have been studied in the differentiation of transudates and exudates so far: alkaline phosphatase, bilirubin, creatinine kinase, uric acid, PE protein electrophoresis, acute phase proteins, pseudocholinesterase, PE/serum cholinesterase rate, cholinesterase, cytokines, HDL/LDL rate, triglyceride, cholesterol, glycosaminoglycan, copeptin, YKL-40 and ceruloplasmin. However, these markers were not shown to be superior to the Light criteria or when repeated, similar results were not obtained, or the diagnostical values could not be verified in later studies.

CONCLUSION

It is sometimes hard to reveal the cause of PE using routine methods. Despite the fact that the Light criteria have been accepted as the first step towards the diagnosis of pleural effusions (PEs), there are concerns over the benefit of this approach. There are new biomarkers proposed for the diagnosis of exudative PEs and presented as a contribution or an alternative to the Light criteria. An ideal biomarker is one that can easily be measured, that has a reasonable price that helps in decisions, that is repeatable and that gives the same results each time it is repeated[17]. Although there are dozens of new biomarkers used in studies related to PEs, only NT-pro-BNP, a natriuretic peptide conform to the criteria of ideal biomarkers that could be used clinically. NT-pro-BNP, a member of natriuretic pep-

tide family, level of pleural effusion being above 1500 pg/mL could be used in determining whether the PEs that are wrongly classified with the Light's criteria but clinically considered to be related to cardiac failure.

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Original Article
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Evaluation of Infants Undergoing Cranial Computed Tomography in the Emergency Department Due to Head Trauma

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Abstract

Pediatric head trauma is one of the most important public health issues worldwide and is one of the most common causes of morbidity and mortality in this age group. In infants, trauma of the same severity with adults bring different outcomes as the body structure is different compared to adults. Imaging methods are frequently used in infants presenting with trauma, since children in the 0-2 age group cannot express themselves, their neurological examination is limited and families of infants cannot provide clear information about trauma. The best imaging method for trauma is cranial computed tomography (CCT). In this study, patients aged 0-2 years who applied to an emergency department of a training and research hospital in Istanbul between January 1, 2018 and January 31, 2018 were examined retrospectively. The aim of this study was to determine a clinical pathway by evaluating cranial computed tomography imagings of 0-2 year-old pediatric patients brought to the emergency department for head trauma. In our study, 523 patients aged 0-2 years were admitted for trauma between the specified time period and 166 of these patients underwent CCT. Of the patients who underwent CCT, 9% (n=15) were found to have pathology due to trauma. Ten of these patients underwent follow-up CCT. One of 15 patients was found to have bone fracture. The other 14 patients were suspected of contusion. Of the patients who underwent CCT, 3.1% (n=5) were admitted to the ward for follow-up. When the patients were evaluated in terms of clinical course, hospitalization or discharge in our study, they were found to correlate with pathologies detected on CCT. Due to the intensity of the patients in the emergency department, sufficient time cannot be allocated for the examination and informing of patients. It was observed that Cranial CT request frequency increased especially between 16: 00-00: 00 hours when emergency department intensity increased. In infant trauma patients, imaging studies should be determined considering the severity and mechanism of trauma.

Keywords: head trauma, infant, computed tomography, emergency department

Introduction

Head trauma is a trauma cause with high mortality and morbidity in developed and developing countries.Pediatric head trauma is one of the most important public health issues worldwide and is one of the most common causes of morbidity and mortality in this age group[1,2]. Head trauma clinics exhibit mild to severe outcomes. Since the head region is like a closed box, diagnostic examinations are required in addition to neurological evaluations. In infants, trauma of the same severity with adults bring different outcomes as the body structure is different compared to adults. Although the incidence of intracranial pathology in children with head trauma varies between 3-5%, the incidence is slightly higher in infants[3,4]. Imaging methods are frequently used in infants presenting with trauma, since children in the 0-2 age group cannot express themselves, their neurological examination is limited and families of infants cannot provide clear information about trauma. The best imaging method for trauma is cranial computed tomography (CCT)[5]. The aim of this study was to determine a clinical pathway by evaluating CCT imagings of 0-2 year-old pediatric patients brought to the emergency department for head trauma.

Materials and Methods

This study retrospectively analyzed patients aged 0-2 years who were admitted to the emergency department of Istanbul Health Sciences University, Kanuni Sultan Suleyman Training and Research Hospital for head trauma between January 1, 2018 and January 31, 2018 and underwent CCT. In this study, patients who were above 2 years of age and who did not have isolated head trauma and whose data were incomplete were excluded from the study as the exclusion criteria. As statistical analyses, compliance with the parametric test criteria was evaluated by performing normality test for all variables. Demographic analysis of the patients was determined by basic descriptive statistics. For correlations between the data, the Spearman's rank-order correlation was

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used in non-parametric data and the Pearson correlation in parametric data.

Results

In our study, 523 patients aged 0-2 years were admitted for trauma between the specified time period and 166 of these patients underwent CT. Of these patients, 89 (54%) were male and 77 (46%) were female. When the hours of admission to the emergency department were evaluated, of the patients, 32.5% (n=54) were admitted between 08:00-16:00 hours, 59% (n=98) between 16:01-00:00 hours, 8.4% (n=14) were admitted between 00:01-07:59 hours (Figure 1). When the admission types were evaluated, of the patients, 92.8% (n=154) were admitted as outpatient and 7.2% (n=12) were brought to the emergency department by ambulance. When the admission days of were evaluated, it was found that of the patients, 13.2% (n=22) were admitted on Monday, 19.2% (n=32) on Tuesday, 10.2% (n=17) on Wednesday, 11.4% (n=19) on Thursday, 13.8% (n=23) on Friday, 14.4% (n=24) on Saturday and 11.4% (n=19) were admitted on Sunday, respectively. Of the patients, 81.9% (n=136) were readmitted to the emergency department. Of the patients who underwent CCT, 9% (n=15) were found to have pathology due to trauma (Figure 2). Ten of these patients underwent follow-up CCT. One of 15 patients was found to have bone fracture. The other 14 patients were suspected of contusion. Of the patients who underwent CCT, 12.7% (n=21) were consulted with neurosurgery with clinical findings and CCT results. Of the patients who underwent

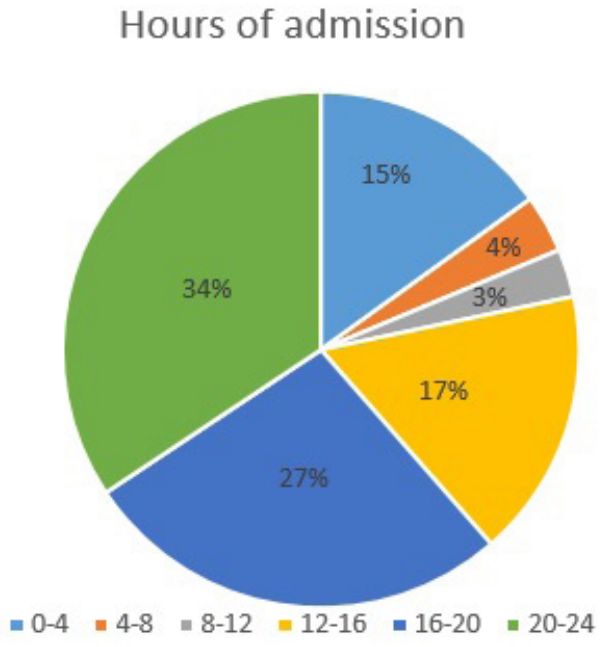

Figure 1.

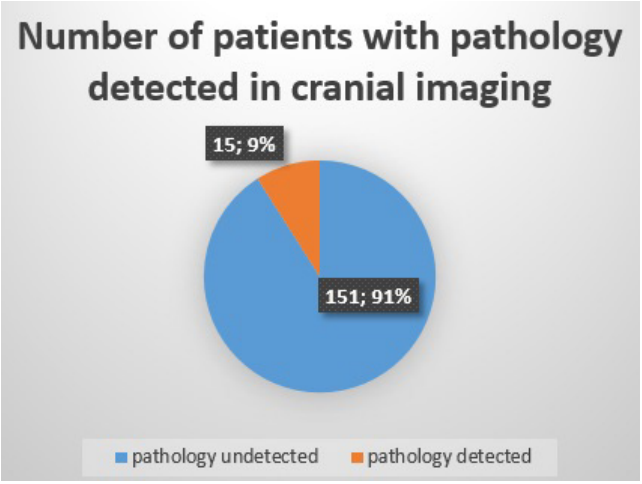

Figure 2.

CCT, 3.1% (n=5) were admitted to the ward for follow-up and 96.9% (n=161) were discharged after examination and follow-up. Ten of the 15 patients who were found to have pathology were discharged after emergency department follow-up on consulting physician's recommendation. A total of 17 patients underwent follow-up CCT; although 7 of these had no pathology on their first CCT, follow-up CCT was considered necessary due to clinical suspicion during the emergency department follow-up. No pathology was visualized on follow-up CCT of these patients and they were discharged. It was found that mortality did not develop in any of the patients included in the study within the following 1 year. In our study, a moderate positive correlation was found in terms of the follow-up CCTs of the patients and the clinical course (r: 0.405, p=0.000). In our study, a moderate positive correlation was found in terms of performing consultation and the clinical course (r: 0.462, p=0.000).

Discussion

In the United States (USA), more than 500,000 children are admitted to emergency departments for head trauma each year[1,6]. Head traumas account for the vast majority of childhood injuries[7,8]. In a study conducted on adults, it was reported that young males in our country carry a high risk for head trauma[9,10,11]. Since our study involves the 0-2 age range, the difference between genders is not seen in our study. In our study, the admission times and days of the infant patients presented with head trauma were analyzed and an intensity was observed between 16:00-00:00 hours. Patients presenting with trauma do not show difference between days of the week.

Computed tomography is preferred as the diagnostic examination for trauma cases to evaluate inside the head. Although the presence of subcutaneous hematoma in the head region under the age of 1 increases the likelihood of bone fracture, it is not clinically sufficient and radiological

imaging is recommended in these patients[12,13].In some studies, the rate of ordering cranial CT as a diagnostic examination in children with head trauma varies between 5-50%[9]. In our study, the rate of cranial CT order was 31.7%, which is similar to previous studies. Especially in infants, CT-induced radiation exposure increases the risk of malignancies and associated mortality[14,15].In our study, the percentage of the patients presented with head trauma and found to have pathology was approximately 1%. The hospitalization percentage of the patients for follow-up is similar to previous studies[16]. In a study on head traumas, no surgical intervention was required for patients similarly to our study[17]. In the study by Da Dalt et al. on infant head trauma, there were patients with fatal outcome. This differs from our study. When the patients were evaluated in terms of clinical course, hospitalization or discharge in our study, they were found to correlate with pathologies detected on CCT.

Conclusion

The limitation of neurological examination in infant and pediatric traumas and inability to express themselves lead us to diagnostic imaging examinations in emergency departments. Due to the intensity of the patients in the emergency department, sufficient time cannot be allocated for the examination and informing of patients. However, the anxious attitude of infant families especially causes medico-legal concerns in physicians and forces the physician to work examination-weightedly. These concerns become more pronounced, especially during the hours when the intensity of emergency department increases. In our study, it is seen that CCT was ordered especially between 16:00-00:00 hours. In terms of ordering a diagnostic examination, a more selective attitude should be adopted for diseases that may further develop and it is thought that imaging examinations should be ordered considering the severity and mechanism of occurrence in infant traumas.

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Original Article
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İnvestigation of the Efficacy of Aminoguanidine in an Experimental Rat Model with Isolated Bilateral Pulmonary Contusion Due To Blunt Thoracic Trauma

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Abstract

İntroduction: In severe thoracic trauma pulmonary contusions are almost inevitable are associated with high morbidity and mortality. In this study we aimed to evaluate the antioxidant activity of aminoguanidine in pulmonary contusion.

Method: Sixty-three Sprague-male rats were used. Sham and aminoguanidine groups were exposed to isolated blunt thoracic trauma with a force of 1,512 joules. Aminoguanidine was administered intraperitoneally at a dose of 100 mg/kg 3 hours before the trauma and on the 1. and 2. day after the trauma. The contusion group was exposed to blunt thoracic trauma only. In all groups, arterial blood gas analysis and catalase and NO levels were done on the 0th, 1st, 2nd and 3rd days.

Results: PO2 levels were higher in the sham group compared to the contusion group, without statistical significance. On the third day, SaO2 levels were higher in the AG group compared to the contusion group. SaO2 levels were comparable in the AG and sham groups on days 1, 2 and 3. There was no difference between the PaO2 levels of the contusion and sham groups on the 2nd and 3rd days. There was no difference between the PaO2 levels of the AG and sham groups on the 1st, 2nd and 3rd days. We found no difference between the PaCO2 levels of the contusion and sham groups on the 0-3 days. There was no difference between the PaCO2 levels of the AG and sham groups on the 1st, 2nd and 3rd days. No difference was observed between the PaCO2 levels of the AG and contusion groups on the 1st, 2nd and 3rd days. No significant difference was found between the NO levels of the sham and the contusion groups on day 0. There was a significant difference between the sham and contusion groups on the 1st, 2nd and 3rd days. There was no statistically significant difference between the catalase enzyme activities of the sham and AG groups.

Conclusion: In our study, we showed that the use of aminoguanidine did not significantly reduce the severity of pulmonary contusion and the inflammatory reaction induced by thoracic trauma in rats.

Keywords: Blunt Thoracic Trauma,Aminoguanidine,Catalase activity

Introduction

Among all trauma cases, chest trauma is the third most common after head and neck and extremity traumas, respectively. It is the most common cause of death, especially in the first 4 decades of life. It occurs due to traffic accidents, occupational accidents, falls and assaults, and constitutes 25% of trauma-related deaths[1]. The mortality rate of isolated chest trauma is 5,5%, but if an additional organ system is injured, this rate rises to 12-15%, and if there is multiple organ injury, it increases up to 30-35%[2]. Pulmonary contusion is defined as trauma-induced alveolocapillary damage associated with overstretching or even rupture of alveoli, separation of alveoli from bronchioles, intraalveolar bleeding, and interstitial edema. Pulmonary contusion occurring in 30-75% of major thoracic trauma cases is a serious injury with high mortality and morbidity. It may be associated with various serious conditions ranging from simple dyspnea to respiratory failure requiring mechanical ventilation. Acute respiratory distress syndrome and multiple organ dysfunction may develop depending on the extent of the contusion[3]. Because the mortality rate of 11% in an isolated severe contusion increases to 22% in the presence of additional injuries. The incidence of ARDS is 17% in isolated contusions, but up to 78% with additional injuries. Pulmonary embolism and pneumonia are important factors that increase the mortality caused by pulmonary contusion. Pulmonary contusion involves an inflammatory process that occurs due to a mechanical damage, with a mechanism that is not fully known. Inflammatory response, and therefore, neutrophils, which are the major factors in endothelial / epithelial damage, are the most important factors determining prognosis[4]. Therefore, it is an issue open to the development of new treatment methods through further researches. In trauma-induced lung injury, gas exchange in bronchioles and alveoli is impaired, resulting in hypoxemia and hypercarbia. Leukocyte infiltration, production of inflammatory mediators, and free oxygen radi-

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cals have a significant role in the pathogenesis of this chain of events. Free oxygen radicals cause oxidative damage through lipid peroxidation, thereby disrupting the integrity of the cell membrane and increasing the permeability of the cells. The resulting hypoxia and hypovolemia may cause ischemia at the cellular level, resulting in necrosis. However, the formation of free oxygen radicals during reperfusion usually aggravates the tissue damage. Therefore, it is necessary to reduce the harmful effects of free radicals in patients with pulmonary contusion. In a healthy organism, the balance between the formation of free radicals and their elimination by antioxidants is called the oxidative balance. In animal models, antioxidant therapy has been shown to be beneficial against lung injury. Nitric oxide, which is effective in the inflammatory process, is synthesized from the amino acid L-arginine by the effect of nitric oxide synthetase. Nitric oxide (NO) synthesized from endothelium leads to pulmonary vasodilatation, reducing shunting in well ventilated lung regions, increasing oxygenation and decreasing pulmonary edema[5]. Expression of NOS in acute lung injury and inflammatory lesions of the colon has been shown to lead to overproduction of NO, resulting in the production of superoxide and peroxynitrite. The aminoguanidine, a nucleophilic hydralazine derivative from the biguanide group, inhibits nitric oxide synthase via its hydrazine group. It acts as an antioxidant to prevent the formation of reactive oxygen compounds and lipid peroxidation in cells and tissues, thus performing as a kind of free radical scavenger[6-7]. Catalase is an antioxidant enzyme found in peroxisomes within the cell. It catalyzes the reaction that converts hydrogen peroxide to water and oxygen. It oxidizes organic compounds such as phenol, formaldehyde and alcohol using H2O2, which is formed by the oxidation of glucose molecules during metabolism, especially in liver and kidney cells. Thus, toxic substances from the bloodstream are detoxified. Catalase exhibits its reducing activity on small molecules such as H2O2, methyl and ethyl hydroperoxides. It has no effect on lipid hydroperoxides which have large molecular structure[8]. One way of reducing tissue damage associated with NO and peroxynitrite is to inhibit the overproduction of NO by a specific inhibitor of iNOS, such as AG. The involvement of oxidative stress and inflammatory process in tissue damage induced us to investigate the effects of aminoguanidine on pulmonary contusion in an experimental rat model with isolated bilateral lung contusion due to blunt thoracic trauma. In the light of this information, our study aimed to reduce the endothelial / epithelial damage in lung contusion by suppressing the inflammatory mechanism and to investigate the effects of this reduction on mortality and morbidity.

Methods

The study was approved by the Animal Experiments Ethics Committee (14.05.2011) of the Hospital (2010/19) and used a total of 63 Sprague Dawley rats from Experimental Animal Production Laboratory. Before the experiment, the rats were kept in wire cages for 12 hours at night and 12 hours at daytime, in circadian rhythm at an ambient temperature of 20 to 26°C for 10 days. Twelve hours before the experiment, feeding was stopped, except for water. All rats were cared for in accordance with the principles of Care of Experimental Animals formed by the National Society for Medical Research (NSMR) and with the Guide for the Care and Use of Laboratory Animals prepared by the Institute of Laboratory Animal Resources and published by the National Institute of Health. The groups were formed by randomly assigning 7 rats to each group (Table 1). Group 1 (Sham) (n = 7), subjected to blunt thoracic trauma and not given aminoguanidine. Group 2 (Control) (n = 7), not subjected to blunt thoracic trauma but given aminoguanidine. Group 3 (Contusion) (n = 28), subjected to pulmonary contusion by blunt thoracic trauma and given saline via intraperitoneal route. A total of four subgroups were created for the 0th, 1st, 2nd and 3rd days. Group 4 (Aminoguanidine) (n = 21),

Table 1. SatO2, PaO2 (Blood gas) values between groups

Day	Sham	Contusion	Aminoguanidine	Aminoguanidine + control
SatO2				
0	94.85 (91.0-96.6)	65.03 (31-90)	-	90.51(84-96.6)
1	94.85 (91.0-96.6)	65 (32-89.1)	87.3 (78.1-95)	90.51(84-96.6)
2	94.85 (91.0-96.6)	77.27 (64.3-86.1)	81.48 (75-86.4)	90.51(84-96.6)
3	94.85 (91.0-96.6)	80.7 (71.8-83)	89.78 (84.5-96.8)	90.51(84-96.6)
PaO2				
0	81.98 (64.7-94.1)	47.03 (27.3-70.2)	-	75.17(53.7-85.7)
1	81.98 (64.7-94.1)	48.03 (27.3-71.2)	79.3 (57-104)	75.17(53.7-85.7)
2	81.98 (64.7-94.1)	67.17 (57.3-78.7)	61.94(55.2-77.4)	75.17(53.7-85.7)
3	81.98 (64.7-94.1)	65.25 (52-87)	72.88 (58-92.4)	75.17(53.7-85.7)

subjected to pulmonary contusion by blunt thoracic trauma and given aminoguanidine. A total of three subgroups were created for the 1st, 2nd and 3rd days. In the groups given aminoguanidine, the administration was performed by intraperitoneal route at a dose of 100 mg / kg 3 hours before the trauma. In the control and aminoguanidine groups, 100 mg / kg dose of aminoguanidine was continued on the 2nd and 3rd days. Rats which were fasted 12 hours prior to the procedure were anesthetized with 100 mg / kg xylazine and 10 mg / kg ketamine. The lexon platform was used to create pulmonary contusion through the effect transmitted to the thoracic wall by saving the sternum and heart. The rats were exposed to blunt thoracic trauma with a force of 1,512 joules, except for sham and control groups. After the trauma, 2 cc blood samples were taken from the abdominal aorta of 7 rats in contusion and sham groups under anesthesia. In these blood samples, it was aimed to determine the levels of PaO2, SaO2 and PCO2, and of catalase and malondialdehyde among oxidative stress parameters. On day 1, the sham group, 0th and 1st day subgroups of the contusion group and 1st day subgroup of the AG group were sacrificed. There are a total of 4 rats died on the 1st, 2nd and 3rd days. On day 2, administration of the drug continued in the remaining rats (not sacrificed). On the 2nd day, the 2nd day sub-groups of the contusion group and AG groups were subjected to the procedures performed in the sham group and the 1st day subgroup of the AG group. On the 3rd day, the 3rd day subgroups of the contusion group and AG groups were subjected to the procedures performed in the sham group and the 1st day subgroup of the AG group. The exploration revealed that the causes of death in the rats were pneumothorax, hemothorax, and cardiac tamponade (Figure 1, 2).

Blood samples were taken into EDTA-K3 anticoagulant tubes and centrifuged at 3000 rpm for 10 minutes. After centrifugation, the supernatant plasma was transferred to Eppendorf tubes and stored at -80 ° C until analysis. Plasma levels of NO and catalase were studied (Figure 3).

Statistical Analysis

Data were recorded in pre-prepared forms. The recorded data was numbered and transferred to the computer. Statistical analysis were performed using SPSS for Windows version 15.0. Median, minimum and maximum values of the groups were calculated. Intergroup comparison was performed by Kruskal-Wallis variance analysis. Chi-Square Test was used for intergroup comparison of categorical data. A p value of <0.05 was considered significant.

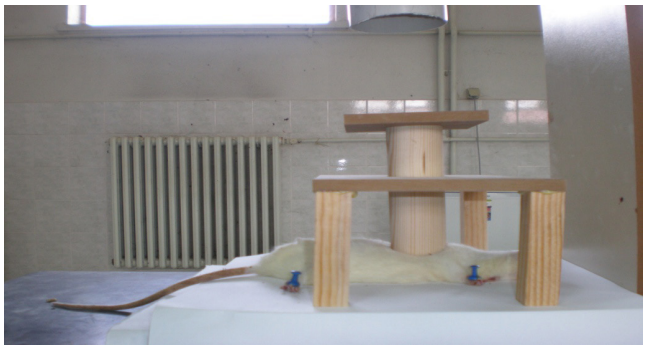

Figure 1. Mechanism used for blunt thoracic trauma

Figure 2. System for collection of blood samples for serial blood gas and other studies in rats which were under anesthesia and underwent trauma.

Figure 3. Lung taken from the sham group after sacrification

Results

Evaluation of the SaO2 and PaO2 levels showed no difference between the Sham and AG groups on the 1st, 2nd and 3rd days, whereas a significant difference between sham and contusion groups on the 2nd and 3rd days ($p < 0.05$). There was no significant difference in the SaO2 levels of AG and contusion groups on the 1st and 2nd days ($p > 0.05$). The SaO2 levels of the AG group on day 3 were significantly different compared to the contusion group ($p < 0.05$). There was no statistically significant difference between paO2 levels of AG and contusion groups. No significant difference was found between the groups Sham and AG in terms of PaCO2 levels on the 1st, 2nd and 3rd days ($p > 0.05$). No significant difference was found between the sham and contusion groups in terms of PaCO2 levels on 0th and 1st days. Significant differences were found in paCO2 levels between the sham and contusion groups on the 2nd and 3rd days ($p < 0.05$) (Table 1). No significant difference was found between the AG and the contusion groups in terms of paCO2 levels ($p=0.05$) (Table 2). No significant difference was found between the NO levels of the sham and the contusion groups on day 0. There was a significant difference between the sham and contusion groups on the 1st, 2nd and 3rd days ($p < 0.05$) (Table 2). We found a significant difference between the groups Sham and AG on the 1st and 2nd days, with no significant difference on the 3rd day ($p > 0.05$). No significant difference was found between the Sham and AG control groups on the 1st, 2nd and 3rd days ($p > 0.05$). Comparison of AG and contusion groups showed a decrease in NO levels in AG group, with no statistically significant difference ($p > 0.05$). No significant difference was found between the AG and AG control groups in terms of NO levels ($p > 0.05$) (Table 3). No significant difference was found between the catalase enzyme activities of sham and contusion groups on all days ($p > 0.05$) (Table 3). There was no statistically significant difference between Sham and AG groups ($p > 0.05$). No significant difference was found between Sham and AG control groups ($p > 0.05$). The comparison of AG and contusion groups showed a significant increase in catalase enzyme activity on days 1 and 2 in AG group ($p < 0.05$), without significant difference on day 3 ($p > 0.05$). No significant difference was found between the AG and AG control groups in terms of catalase enzyme activity ($p > 0.05$) (Figure 4).

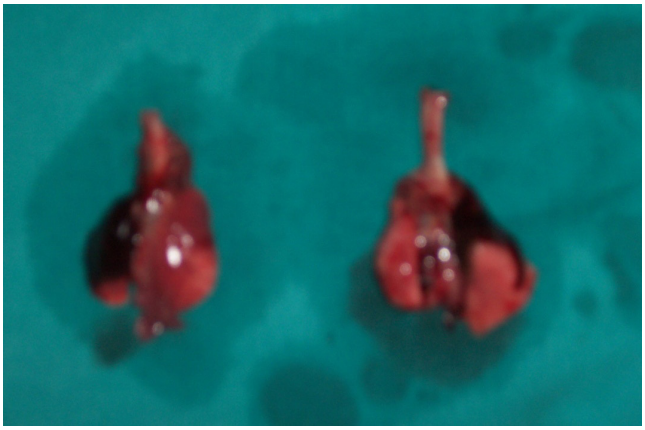

Figure 4. Hemothorax in rats died after contusion

Table 2. Nitric oxide values between groups

Day	Sham	Contusion	Aminoguanidine + contusion	Aminoguanidine + control
0.	3.7(1.68-6.76)	8.58 (1.72-20.05)	-	3.06(0.35-4.6)
1.	3.7(1.68-6.76)	10.3(4.64-16,6)	5.25(1,6-10.68)	3.06(0.35-4.6)
2.	3.7(1.68-6.76)	9.77(2.6-11.6)	7.26(4.38-9.54)	3.06(0.35-4.6)
3.	3.7(1.68-6.76)	9.78(2.6-13.5)	4.32 (3.37-5.5)	3.06(0.35-4.6)

Table 3. Nitric oxide values between groups

Day	Sham	Contusion	Aminoguanidine + Contusion	Aminoguanidine + control
0.	12.51(7.5-18.9)	12.16 (7.7-14.3)	-	11.4(6.8-15)
1.	12.51(7.5-18.9)	9.8 (7.42-10.8)	12.7 (9.5-14.7)	11.4(6.8-15)
2.	12.51(7.5-18.9)	9.28 (6.1-11.7)	11.6 (9.7-13.2)	11.4(6.8-15)
3.	12.51(7.5-18.9)	8.76 (5.6-11.6)	10.8 (9.6-13)	11.4(6.8-15)

Discussion

Trauma is one of the most common causes of death in the young adult population. Chest traumas, which usually occur as a result of traffic accidents, occupational accidents, assaults and falls, are the third most common after head and limb traumas.[9] Pulmonary contusion can be seen in approximately 50% of patients with chest trauma, which is one of the most important factors that increase the mortality by adversely affecting the clinical course. It is important to investigate the preventable causes of death to reduce mortality from chest trauma seen predominantly in young adults. Secondary damage following a trauma causing pulmonary contusion involves oxidative stress, lipid peroxidation, and inflammatory response.[10, 11] Oxidative stress is the deterioration of the physiological balance between the formation of free radicals and their elimination by antioxidants. Lipid peroxidation is the process of degradation of polyunsaturated fatty acids by oxidation. After this process, the resulting products can adversely affect the surrounding tissues. The effects of lipid peroxidation on the cell membrane are irreversible.[12] The presence of superoxide radicals (SORs) in the environment leads to the synthesis of NO and peroxynitrite and triggers lipid peroxidation. These released SORs can cause oxidative damage through lipid peroxidation.[13, 14] Aminoguanidine is a compound that inhibits selectively and competitively inducible nitric oxide synthase and thereby causes reduced nitric oxide formation. This is an effective antioxidant and free radical scavenger. It prevents the formation of lipid peroxidation in cells and tissues.[15] There are studies which have proven that aminoguanidine reduces both the inflammatory response and the degree of pulmonary injury.[16] In a study, O. Soy et al. showed that initiation of aminoguanidine treatment (100mg / kg) immediately after the trauma prevented both nitric oxide production and lipid peroxidation and improved the functional status of animals.[17] The administration of systemic antioxidant agents in intensive care patients with mechanical ventilator support reduced the plasma levels of lipid peroxidation products and the amount of mucus in the respiratory tract, resulting in better clinical results. In the present study, NO levels in the contusion group were significantly higher than the sham group. In addition, NO levels were lower in the aminoguanidine group compared to the contusion group. These elevated levels were attributed to trauma-induced alveolar capillary damage and subsequent ventilation/perfusion mismatch. No statistically significant difference was found between NO levels of aminoguanidine and contusion groups. In addition, there was no significant difference between NO levels of the sham and aminoguanidine control groups. In our study, it was found that CAT enzyme levels increased significantly in the contusion group compared to the Sham group. No significant difference was found between CAT enzyme activities of the contusion and AG groups ($p > 0.05$). In addition, there was no significant difference between CAT enzyme activities of the sham and AG control groups. The data obtained in our study were consistent with the literature and it was shown that aminoguanidine at a dose of 100 mg / kg might have protective effects against pulmonary damage mechanisms. We believe that it may be useful to carry out further studies with different doses to reveal the maximum efficacy of AG.[18]

In our study, we observed that the use of aminoguanidine in rats exposed to isolated pulmonary contusion by blunt trauma reduces the severity of pulmonary contusion and minimizes the inflammatory reaction.

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Conflict of interest: None declared.

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Case Report
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A Rare Trauma Complication: Pneumopericardium

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Abstract

Pneumopericardium is a rare condition that is defined as the presence of fre eair in the pericardial space. Although it is secondary to the most frequent traumatic or iatrogenic causes, it can rarely be seen spontaneously. A 32-year-old female patient was brought to our emergency service by 112 due to a car accident. Radiological imaging of the patient revealed spinous process fracture in thoracic vertebra (ninth thotacic vertebra), sacroiliac joint dissociation, iliac wing, pubis, ischium and femur diaphysis fracture. There was minimal free fluid under the spleen and the pelvis. Thorax tomography revealed multiple rib fractures and fractures in the left clavicle. In addition, wide spread contusion and hemopneumothorax were observed in the left lung. In addition, air density was determined in the pericardial space and mediastinum. Consultations were requested for the patient and the patient was admitted to the intensive care unit for further examination and treatment. Pneumopericardium can be defined as the presence of air in the pericardial space. In case of clinical suspicion for diagnosis of pneumopericardium, further investigation with thorax tomography and echocardiography is required. In the emergency services, which are the first referral places of multiple traumas, they should keep in mind the diagnosis of pneumopericardium in these patients, especially when chest trauma is accompanied by these patients.

Keywords: Trauma, emergency medicine, pneumopericardium

Introduction

Pneumopericardium is a rare condition that is defined as the presence of fre eair in the pericardial space[1]. Although it is secondary to the most frequent traumatic or iatrogenic causes, it can rarely be seen spontaneously[2]. It is usually associated with pneumothorax and pneumomediastinum after blunt chest trauma[3]. It often limits itself. However, it is a clinical diagnosis that should be considered in patients with blunt chest trauma because of the high mortality rate in case of accompanying tamponade findings[4].

Case

A 32-year-old female patient was brought to our emergency service by 112 due to a car accident. There were no features in the patient's medical history. The patient had head, chest and pelvic trauma. The glaskow coma score was 13. In the vital parameters of the patient, blood pressure was 110/60 mmHg, pulse was 117 beats / min, respiratory rate was 22 / minute and fever was 36 °C. Her electrocardiography was consistent with sinüs tachycardia. Cardiovascular system examination showed heart sounds deep, no additional sound or frotman was detected. There were no pathological findings except tachycardia. Respiratory examination showed a decrease in respiratory sounds in the left lung. His abdominal examination wascomfortable, there were no defenses or rebounds. There was tenderness in the pelvis and left femoral head. Hemoglobin level was 11.7 g / dL and hematocrit was 36%. Biochemical parameters were alanine aminotransferase 68 U/L and aspartate aminotransferase 148 U/L. After stabilizing the patient, radiological examinations were performed. Radiological imaging of the patient revealed spinous process fracture in thoracic vertebra (ninth thotacic vertebra), sacroiliac joint dissociation, iliac wing, pubis, ischium and femur diaphysis fracture. There was minimal free fluid under the spleen and the pelvis. Thorax tomography revealed multiple rib fractures and fractures in the left clavicle. In addition, wide spread contusion and hemopneumothorax were observed in the left lung. In addition, air density was determined in the pericardial space and mediastinum (Figure 1). Pneumomediastinum and pneumopericardium were considered. An emergency bedside echocardiography was performed. Although air density could not be selected clearly in echocardiography, there was no myocardial pathologic finding. Consultations were requested for the patient and the patient was admitted to the intensive care unit for further examination and treatment. In the intensive care follow-ups,

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lung expansion was achieved with tube thoracostomy and no thoracotomy was performed, and tube thoracostomy was terminated. After stabilizing the general condition, the patient was operated for orthopedic fractures. Echocardiography did not revealany pathology. The patient was discharged from the intensive care unit after being stabilized.

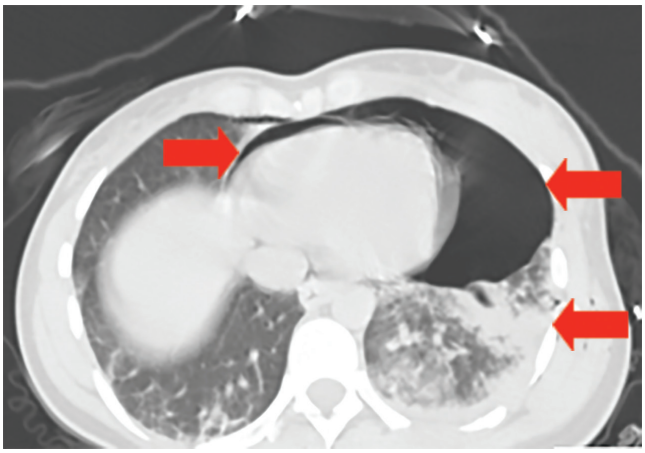

Figure 1. Pneumopericardium together with pneumohemothorax and contusion in the left lung

Discussion

Pneumopericardium can be defined as the presence of air in the pericardial space. It is usually caused by blunt and penetrating trauma or iatrogenic causes. It may also ocur spontaneously[2]. The air can pass through the perivascular sheath through a direct connection to the pericardial space, or through the congenital plevropericardial connection. Although it is usually a self-limiting condition, it may rarely be presented with tension pneumopericardium or tamponade[5]. Simple pneumopericardium may be introduced to the tension pneumopericardium by mask valve ventilation or positive pressure ventilation by mechanical ventilator[6]. Therefore, in patients diagnosed with simple pneumopericardium, follow-up is necessary for tamponade and tension pneumopericardium. The diagnosis of pneumopericardium is difficult. The use of plain radiography in the diagnosis of pneumopericardium is limited, only useful in demonstrating additional pathologies such as pneumothorax and hemothorax[7]. In case of clinical suspicion for diagnosis of pneumopericardium, further investigation with thorax tomography and echocardiography is required. Tomography is highly sensitive to both the diagnosis of pneumopericardium and to show additional pathologies[3]. Simple pneumopericardium often limits itself and does not require treatment. Hypotension, tachycardia, deep heart sounds, electrocardiography findings in the presence of low voltage in the precordial leads should be thought of cardiac tamponade and if the diagnosis is made, emergency pericardiocentesis should be performed. Pericardial decompression and pericardial window drainage should be evaluated as definitive treatment[3].

Conclusion

In the emergency services, which are the first referral places of multiple traumas, they should keep in mind the diagnosis of pneumopericardium in these patients, especially when chest trauma is accompanied by these patients. It should be noted that in cases such as hypotension and deep heart sounds, tamponade may be developed. Due to the risk of progression of simple pneumopericardium to the tamponade and tension pneumopericardium, these patients should be under cardiac follow-up in intensive care.

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Case Report
Eurasian Journal of Critical Care

Does A Potato Threat Your Life? A Case Study

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Abstract

The mistakes on consuming the foods that are stored in improper conditions or that are kept longer than what it is supposed to be can cause some of the clinical symptoms from basic to life threatening ones. We present a case study of a 28 years old patient that came to our clinic with hemodynamic disturbance after eating green potatoes where he/she kept them in a storage to consume them in the winter season. Our discussion based on the fact that the formation of the Solanine toxin which is likely seen on the foods whose natural form changed can cause poison-ing.z

Keywords: Potatoes, Solanine, Poisoning

Introduction

Consuming the green surface of the potatoes that is formed by the germination is so harmful for the human health because of the toxic substance that is s called Solanine. The normal tuber contains only small amounts of solanine in the peel and none in the flesh. Solanine is initially de-veloped in the sprout of the potatoes, but eventually it can be detected in the flesh of the tuber so that concentration-gradient between the peel and the flesh is lost. Solanine is an inhibitor of ace-tylcholinesterase that blocks the breakdown of acetylcholine. Solanine poisoning displayed by gastrointestinal and neurological disorders. Symptoms include nausea, diarrhea, vomiting, stom-ach cramps, burning of the throat, headaches, and dizziness. Solanine is strongly offensive and corrosive to gastrointestinal mucosa, hence we should also consider the possibility of observing severe haemorrhagic ulceration of gastrointestinal system. Hallucinations, loss of sensation, and paralysis, fever, jaundice, mydriasis have been reported in more severe cases. We want to dis-cuss the case of the patient that came to our emergency room with the symptoms of acute hy-potension and high fever as a result of food poisoning.

Case

A 28 year of male patient were transferred to our clinic with food poisoning. He was poisoned in one to two hours after eating potatoes that he brought from his hometown for his dinner. He de-scribed the nausea, vomiting, and diarrhea. He was conscious and cooperative. His vital signs were: the blood pressure 100/70 mmHg, pulse 98 ppm, respiration rate 20 ppm, SpO2 96%, and his temperature was 37.1°C.

According to the patients physical examination the patient had a sensation on the epigastric re-gion, and he had a hyperactive intestinal peristaltic sound. The patient was hemodynamically stable. He was transferred to observation room, and then we established a vascular access for the fluid treatment, but in the next two hours the patients general situation was getting worse, it was observed the disorientation in his conscious and excessive sweating. To know the patient's vital signs he was monitored, and we observed that the patient's blood pressure 70/40 mmH, pulse 124 ppm, respiration rate 22 ppm, SpO2 97%, and his temperature was 39.8°C. The fluid treatment for hypotension was accelerated and 1gr paracetamol IV accompanied by periph-eral cooling were applied to drop his fever. Following this we called the national poisoning consul-tation center to enrolled the patients and to hear their suggestions. We were told that the patient likely was poisoned by solanine after eating green potatoes and they advised to monitor the patient in the next 24 hours. In the next four hours we observed that the patient hemodynamically stable:

His blood pressure 110/65 mmH, pulse 98 ppm, respiration rate 18 ppm, and his temperature was 37.4°C. The patient's blood test came normal, and following the 24 hours observation the patient's disorders was disappeared, therefore the patient was discharged from the hospital with a return condition after 24 hours.

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Discussion

The most common cause of the food poisoning is the toxic substances produced by bacteria. Some of the bacteria are reproduce in millions when they find the necessary tem-perature, mois-ture, nutriment, and time. Potatoes that are stocked for winter-use are germinated when the tem-pera-ture of the weather raise and when they found enough light and moisture. In our cases, the potatoes that are stocked for winter are germinated when they found necessary tem-perature, light, and moisture (figure 1). Therefore, the foods which are not in the natural form should not be consumed. Solanine is a toxic found in many food plants. It is formed in many food plants such as potatoes, tomatoes, eggplants, and grapes. It is rare to see its poisoning in literature, but the high doze of consumption might be very serious. When solanine consumed at 0.4-0.6 mg/kg, the symptoms of nausea and emesis may occur, and the lethal dose is 3-6 mg/kg. Even the Sola-nine poisoning cases have seen in history, it is very rare in nowadays. The reason of discussing this case is that we like to stress out the importance to know how to store the foods properly and not to consume the food plants that are not in their natural form as explained in our example.

Figure 1. Germinating potatoes

The case we encounter seemed at first a simple food poi-soning, but on the advancing hours the patient's situation got worse that led us to examine the case in detail and later we knew that this was a serious food poisoning case. Therefore, food poisoning case should be investigated holis-tically in detailed by questioning the patient's stories, his vital signs, physical examination and so on.

Result

As emergency service physicians we should always consid-er the food poisoning cases serious-ly, even the first phase of the food poisoning related to green potatoes is monitored as a simple food poisoning later on the situation might turn out serious, hence it is advised to observe the patient 24 hours before discharge him/her from the hospital. It is also advised not to underesti-mate ant type of food poisonings, the food caused the poisoning should be investigated in terms of its characteristics.

To eliminate the food poisoning we should develop a social consciousness through educating the society about how and under which circumstances the toxic substances are formed on foods.

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Case Report
Eurasian Journal of Critical Care

Periorbital Air Following Sneeze

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Keywords: Emphysema, Periorbital Air, Snreze

Introduction

Presence of air in the orbit and periorbital tissues is called orbital emphysema[1]. Frontalprocess of themaxillary bone, lacrymal bone andlateralwall of theethmoid bone (laminapaprycea) formsthemedialwall of theorbit. Maxillary sinus and frontal sinus are located adjacent to orbit base and orbit floor, respectively. Any possible defect on these bones causes orbital emphysema[2]. Orbital emphysema usually appears within first 24 hours in fractures of paranasal sinus involving the orbital walls[3]. Spontaneous absorption of orbital emphysema which is a transient phenomenon occurs within two weeks[4]. Orbital emphysema resolves without sequel by spontaneous resolution; however, it may cause severe visual loss. Treatment approach includes observation, systemic cortisones, nasal decongestants and surgery when required[5,6].

The aim of this report was to present a case who developed periorbital emphysema following sneezing after an impact onto the nose.

Case 1

A 46-year old female patient referred Emergency Room (ER) around 3:00 p.m. due to swelling on the left eye. Medical history of the patient revealed that she was hit onto the nose by her child accidentally around 10 a.m. on the same day. The patient then squeezed her nose and closed her nostrils during sneezing around 1 p.m. She felt a blood clot on her hand; her left eye was swollen and she had temporary vision loss. Physical examination revealed that vital signs were stable, her left eye was swollen and closed due to edema, crepitation is detected on the left upper eyelid; her vision was normal and light reflex was positive when the eyelid is opened. According to the patient's maxillofacial computer tomography, free air densities on the left periorbital area and edematous appearance were detected; left nasolacrimal channel was prominent due to air (Figure 1). Consultation with otorhinolaryngology and ophthalmology was performed for the patient. Otorhinolaryngologistperformed an endoscopic examination and identified hemorrhagic spotting on the left meatus of the orbit; however, no bleeding was detected and monitoring on outpatient basis was recommended. Treatment of the patient was planned in consultation with otorhinolaryngology and ophthalmology clinics; she was advised not to blow her nose fast and to sneeze with open mouth. The patient was discharged at a stable state.

Case 2

A 45-year old male patient referred Emergency Room (ER) around 9:00 p.m. due to swelling on the left eye. It was learned from medical history of the patient that he hit his nose on the wall in the same day; he closed his nostrils during sneezing and his eyes swell. In the physical examination, vital signs were stable, left eye was swollen and closed due to edema; crepitation was detected on the left lower eyelid; the patient had normal vision when his eyelid was opened with a positive light reflex (Figure 2). Maxiofacialcomputertomographyscan was performed; free air densities were detected in the left periorbital area and left maxillary sinus roof was detected broken (Figure 3). A consultation was performed with otorhinolaryngology and ophthalmology clinics. Both consultants recommended medical treatment and outpatient follow-up.

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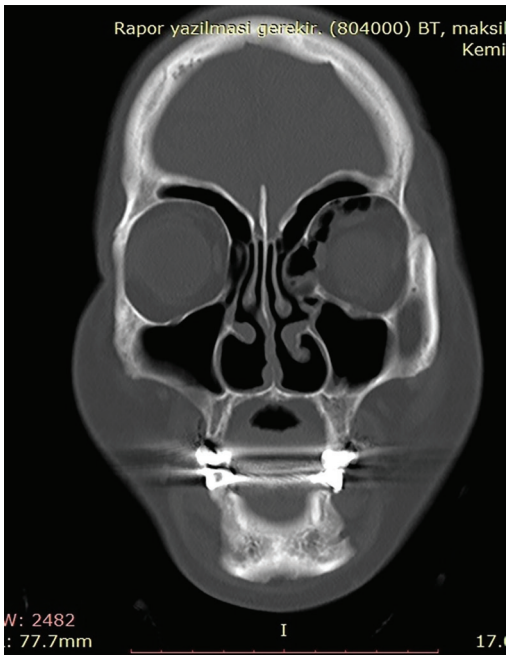

Figure 1. CT of Case 1

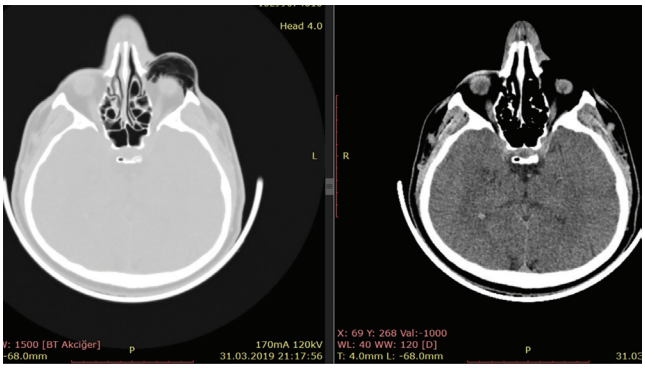

Figure 2. CT of Case 2

Figure 3. EYE of Case 2

Discussion

The studies showed that intranasal pressure increases when mouth and nose are closed during sneezing. High pressure facilitates appearance of fractures of the bone which is defective as a result of chronic sinusitis[7]. Furthermore, fracture due to trauma on the bone line leads to air leak into the orbit; however, theairdoes not leavetheorbita[8]. Our cases developed the emphysema following sneezing after trauma to the nose. Jonathan et al.[9] and Oba et al.[10] presented traumatic periorbital cases similar to our cases. Levent Sahin[1] alsopresented a case with periorbital emphysema after nose blowing and the emphysema was drained in such case. Ophthalmologyclinicdid not recommenddrainageandsuggestedantibiotherapyandpolyclinicontrol. Oba et al.[10] also did not performdrainageandarrangedthetreatmentbyantibiotherapy.

Conclusion

The eye has connections with many bones on the face including the connection with nose, nasal and lachrymal canals. Air may leak around the eye due to trauma after sneezing while the mouth is closed following any trauma onto the nose and other face regions. These patients may refer to the emergency room. Consequently, the emergency department physicians should be aware that such air has a benign progress and may be treated by antibiotics; they also should arrange consultations and follow-ups with ophthalmology clinics.

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Case Report
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Bilateral Facial Paralysis Following Tooth Extraction

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Keywords: Bell Palsy, Facial Paralysis, Tooth Extraction

Introduction

Anatomy and function of the facial nerve (FN) was first identified by Sir Charles Bell in 1800s[1]. The facial nerve is the seventh of 12 cranial nerves innervating the muscles which control facial expressions and transmits the taste sense from oral cavity and two thirds of the posterior side of the tongue[2]. Facial nerve palsy (FNP) may appear due to many reasons. Although possible reasons include genetic factors, vascular ischemia and inflammation due to a viral infection, autoimmune diseases, temporal bone fractures, head-neck tumors, central nervous system lesions, and etiology is still contradictory. Despite all reasons mentioned above, two third of FNPs has no reason and is called "idiopathic"[3, 4].

Motor nuclei of the facial nerve on the pons are innervated by the motor cortex bilaterally; upper 1/3 of the face receives fibers from double innervated part of the nucleus on the pıns whereas lower 2/3 of the face receives fibers from single innervated part. In consideration of this trace, there are two types of facial paralysis; facial and central facial paralysis[5, 6, 7, 8, 9].

We aimed to present a rare case with bilateral facial paralysis after tooth removal.

The Case

A 44-year old male patient referred emergency clinic because of numbness on both halves of the face and difficulty to close the eyelids. Medical history investigation revealed that the patient had removal of one of the teeth on the right mandible and he had pain and numbness on the chin 4 days before the referral. Past medical history of the patient was nonspecific.

The following findings were obtained during physical examination at referral to the emergency clinic; overall state was well, arterial blood pressure was 130/80 mmHg, pulse was 95/min, body temperature was 36.5°C. Neurological examination of the patient revealed that the patient was conscious; he has cooperation and orientation; pupils were isochoric; light reflex was positive bilaterally; conjugated eye movements were liberated at four directions; eye squeezing was weak bilaterally; eyebrow elevation loss bilaterally; nasolabial sulcus was indistinct bilaterally; no neck stiffness was detected. Muscle strengths are complete, cerebellar tests are normal and no ataxia was detected. There was not any pathological finding in examination of the respiratory system, cardiovascular system and abdomen.

Blood tests were as follows; WBC: 12.12 10^3/uL, Hb:15.8 g/dl, Plt:434 10^3/uL, Urea:34 mg/dl, creatinine: 0.86 mg/dL, Na:137 mmol/L, K:3.9 mmol/L, AST:39 U/L, ALT: 16 U/L. Normal sinus rhythm existed in the ECG of the patient. Computed tomography of the brain was normal and there was not any pathology in diffusion magnetic resonance (MR) imaging. Neurology department was consulted for the patient. Contrast cranial MRI was taken by suggestion of the neurology department and there was not any pathology detected. Ear-nose-throat department was consulted for the patient. Ear-nose-throat department recommended 1 mg/day methylprednisolone with gradually decreasing doses. Ophthalmology department was consulted. Artificial tear was recommended. Neurology clinic was consulted again and follow-up in an advanced medical center and the patient was referred to a tertiary medical center for further treatment.

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Discussion

Facial palsy is a clinical presentation of a paralysis which develops in two forms including peripheral and central due to dysfunction of seventh cranial nerve along the facial nerve; and treatment planning includes acceleration of the healing by cortisone, preventing corneal complications and other possible sequels and inhibition of viral replication, if any[2]:

The causes for peripheral facial palsy may be viral (HSV, VZV, CMV, EBV)[5,6,8,9,10,11,12,13,14,15,16,17,18]. However, trauma such as dental treatment may cause the condition[5, 6, 8, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18]. Facial palsy usually appears unilaterally; bilateral form is rare. Sowmya et al.[19] and Owsley et al.[20] reported unilateral facial palsy after tooth removal; however, such cases developed unilateral paralysis. The case presented here developed bilateral facial paralysis after tooth removal.

Kutluhan et al.[21] and Akdag et al.[22] reported cases with facial palsy due to bilateral otitis media and Melkersson-Rosenthal Syndrome, respectively. As is seen, bilateral facial palsy is rarely detected due to different causes. The cause presented in this case report was tooth removal.

Conclusion

Emergency medicine physicians should be aware that bilateral facial palsy may exist due to different underlying causes; and they should investigate the etiology during medical history taking.

It should be noted that traumatic procedures such as tooth removal may cause paralysis which is rarely bilateral; neurology and ear-nose-throat clinics should be consulted for investigation of the etiology and the patient should be referred to an advanced medical center for further research.

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Case Report
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Post Ercp Perforation

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Introduction

Endoscopic retrograde cholangiopancreatography (ERCP) is a commonly used method in the diagnosis and treatment of biliary and pancreatic duct diseases. Depending on the contrast agent and interventional procedures performed, serious complications such as pancreatitis, bleeding, cholangitis, perforation and sepsis can occur as much as 3-10%[1]. Perforation is one of the most feared complications of endoscopic retrograde cholangiopancreatography. Depending on the degree of perforation, medical treatment may be sufficient or surgical interventions may be required[2].

Case

A 90-year-old male patient was admitted to the emergency department with dyspnea. According to the anamnesis obtained from the patient, the patient's shortness of breath was long-lasting, but he had complaints of new onset abdominal pain. He had chronic obstructive pulmonary disease, coronary artery disease, and chronic renal failure. When the patient's anamnesis was deepened, it was learned that he underwent ERCP for choledocholithiasis 10 days ago. The vital parameters of the patient were 36.2°C, pulse 75 / min, TA: 113/6 mmHg, respiratory rate 20 / min, sPO: 99%. In the physical examination, the patient had severe pain in the right upper quadrant of the abdomen. Other system examination findings were normal. In the patient's hemogram, WBC: 20,7 * 10 ^ 9 / L and creatinine were 2.35 mg / dL, but other biochemical parameters were normal. The CRP of the patient was15.8 mg / dL (normal range0.35). Abdominal ultrasonography was requested in accordance with physical examination and laboratory values. The patient's abdominal ultrasonography revealed that the gall bladder was of normal size, wall thickness and echo were normal, and a large number of Stone echoes and common bile duct dilated (7 mm). Then the patient with CRF was asked for non-contrast abdominal CT. Non-contrast abdominal CT revealed suspicious free air densities in the paraduodenal area and was first evaluated in favor of intra-retroperitoneal abscess secondary to duodenum perforation (Figure-1). The patient was referred to the general surgery intensive care unit.

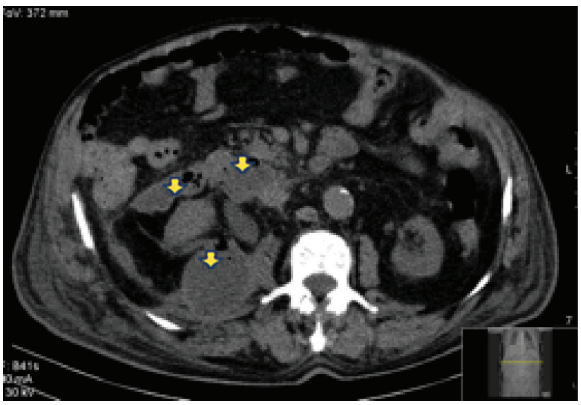

Figure 1. Intra-retro peritoneal abscess secondary to duodenum perforation

Discussion

The diagnosis of duodenal perforation after ERCP is generally based on physical examination findings, fluroscopic imaging and in some cases by computed tomography imaging[2]. Treatment of these perforations is still controversial[3]. The general principles of treatment include discontinuation of the patient's oral intake, administration of nasogastric cath-

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eter and initiation of iv antibiotherapy. Stapfer classification is currently used in the selection of patients to undergo surgery[4]. According to this classification;

Type I: Free bowel wall perforation
Type II: Retroperitoneal duodenal perforation secondary to periampullary injury
Type III: Perforation of the pancreas or bile duct
Type IV: Retroperitoneal air only

Among these, the most common type is II. Among these groups, the most common type 1 and type 2 injuries are surgically treated and conservative treatment methods are preferred in most patients4-8. However, it requires careful observation and early surgical consultation, as the result may be poor in patients who are unable to receive fast and appropriate treatment. In addition to the type of injury, the age, concomitant diseases, previous surgery and medical history of the patient are also must considered. The prognosis of patients with perforation depends on the rate of recognition, clinical structure and patient comorbidities9-11. Our case was also evaluated as type 2 injury. When the patient's current medical condition, delay in diagnosis and type of injury were taken into consideration, medical treatment decision was made and the patient was taken to intensive care follow-up.

Conclusion

Perforation should be kept in mind in patients with abdominal pain starting with endoscopy and ERCP. A careful history and physical examination in emergency departments can be diagnosed by direct radiography and computed tomography. Most of the cases diagnosed early can be followed by conservative treatment. Delayed diagnosis and treatment may have adverse consequences such as sepsis and death, so early surgical consultation should be sought.

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