© Med Sci Monit, 2012; 18(5): CR308-315
PMID: 22534711

WWW.MEDSCIMONIT.COM
Clinical Research

Received: 2011.06.05
Accepted: 2012.01.05
Published: 2012.05.01

Evaluation of prognostic value of selected biochemical markers in surgically treated patients with acute mediastinitis

Authors' Contribution:
A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
E Manuscript Preparation
F Literature Search
G Funds Collection

Sławomir Jabłoński[1ABDEFG], Marian Brocki[1AF], Kujawski Krzysztof[2BF], Marcin Wawrzycki[1BF], Edyta Santorek-Strumiłło[1BF], Marek Łobos[3BC], Marcin Kozakiewicz[4ACDE]

[1] Department of Thoracic Surgery, General and Oncological Surgery, Medical University of Lodz, Lodz, Poland
[2] Department of Nephrology and Arterial Hypertension, Medical University of Lodz, Lodz, Poland
[3] Department of Laboratory Diagnostics and Clinical Biochemistry Medical University of Lodz, Lodz, Poland
[4] Department of Faciomaxillary Surgery, Medical University of Lodz, Lodz, Poland

Source of support: Departmental sources

Summary

Background: Monitoring of biochemical markers of inflammation in acute mediastinitis (AM) can be useful in the modification of treatment. This study was a retrospective evaluation of selected biochemical parameters with negative impact on the prognosis in surgically treated patients.

Material/Methods: There were 44 consecutive patients treated surgically due to AM of differentiated etiology. Selected biochemical markers (WBC, RBC, HGB, HCT, PLT, CRP, PCT, ionogram, protein and albumins) were assessed before surgery and on the 3rd day after surgery. ANOVA was applied to find factors influencing observations. Numerical data [laboratory parameters] were compared by means of medians.

Results: The overall hospital mortality rate was 31.82%. In the group of dead patients, there were observed statistically significant lower mean preoperative values of RBC [p=0.0090], HGB [p=0.0286], HCT [p=0.0354], protein [p= 0.0037], albumins [p=0.0003] and sodium [p<0.0001] and elevated values of CRP [P=0.0107] and PCT p<0.0001]. High level of inflammatory markers on day 3 after surgery was found to increase the risk of death – for WBC (by 67%), for CRP (by 88%) and for PCT (by 100%).

Conclusions: Poor prognosis was more frequent in patients with preoperative high levels of CRP, PCT, anemia, hypoproteinemia and hyponatremia. The risk of death increases significantly if in the immediate postoperative period no distinct decrease in WBC count and of the CRP and PCT level is observed. In such a situation the patients should be qualified earlier for broadened diagnostic workup and for reoperation.

key words: **acute mediastinitis • sepsis • laboratory risk factors • biochemical markers**

Full-text PDF: http://www.medscimonit.com/fulltxt.php?ICID=882737

Word count: 3577
Tables: 1
Figures: 3
References: 59

Author's address: Sławomir Jabłoński, Departament of Thoracic Surgery, General and Oncological Surgery Medical University of Lodz, Zeromskiego 113 St., 90-547 Lodz, Poland, e-mail: jablonski_s@vp.pl

Background

Acute mediastinitis (AM) is an infectious disease of differentiated etiology, characterized by rapid and severe course. Despite the introduction of modern diagnostics, antimicrobial therapy and early qualification for surgical treatment, the overall mortality remains at 14–47% [1–5]. The invasion of an infection into the sterile mediastinal space leads to rapidly progressing local inflammatory changes, which stimulate systemic inflammatory response syndrome (SIRS). Cellulitis, circumscribed abscesses, phlegmon and necrosis develop locally among loose mediastinal tissues. These changes can be accompanied by uni- or bilateral pyothorax, pericardial exudate, cardiac tamponade, subphrenic abscesses, lung abscesses and other conditions [1,6,7]. Severe sepsis or septic shock develops if it is impossible to surgically control the source of infection and to effectively limit the inflammatory process [8,9]. Unsatisfactory treatment can further lead to multiorgan dysfunction syndrome (MODS) or multiorgan failure (MOF) as well as other complications and death [10].

Over many years researchers have sought to find biochemical markers of inflammation of both diagnostic and prognostic significance, monitoring of which in correlation with the clinical picture and diagnostic imaging can be of great importance to modify therapeutic strategies. The best known widely used markers in everyday practice include the erythrocyte sedimentation rate (ESR), white blood cell count (WBC, leucocyte), white blood cell types (WBC differential), the level of C-reactive protein (CRP) and procalcitonin (PCT) [11]. We think that the information on the behavior of biochemical markers in patients with unfavorable course of AM will enable early recognition of septic complications before their clinical manifestation, as well as in imaging diagnostics. These data can contribute to modification of therapeutic management in the form of earlier qualification for aggressive surgical treatment, application of more effective antibiotic therapy, fluid therapy and advanced life-support care, which in turn can decrease the death rate.

Aim

The aim of this retrospective evaluation of selected biochemical markers determined before and on day 3 after surgery in patients surgically treated due to AM was to search for negative prognostic factors.

Material and Methods

A total of 44 consecutive patients with diagnosed AM were subjected to surgery. The patients fulfilled modified criteria of Esterra et al [2], which in the original version concerned descending necrotizing mediastinitis, including: (1) clinical manifestation of severe infection, (2) demonstration of AM etiological factors, (3) characteristic radiological picture, (4) the pathogen isolation in microbiological cultures from the mediastinal area, and (5) intraoperative or post-mortem documentation of mediastinitis.

Exponents of sepsis including fever, tachycardia, hyperventilation and leucocytosis were observed in all the patients. The age of the patients was from 19 to 83 years, mean age 52.5 years (median 54.5). There were 31 men, mean age 50.9 years (median 55) and 13 women, mean age 56.4 years (median 58). The time of hospitalization was on the average about 3 weeks (23.84±11.96 days, median 21.5); 14 patients died. The total death rate was 31.82% (38.7% in males and 15.4% in females).

The etiology of AM was extremely differentiated (Table 1). Iatrogenic complications were the most frequent cause of mediastinal infection. They were found in 19 patients (43.2%) and were associated with esophageal and tracheal surgeries or with injuries to these organs during endoscopy or intubation. Non-iatrogenic esophageal and tracheal injuries were the cause of AM in 11 patients (25%). This group also included perforations caused by a foreign body. Descending AM was detected in 9 patients (20.4%). In 5 patients (11.4%) AM resulted from a spontaneous perforation of advanced esophageal cancer or lung cancer with infiltration to the esophagus (neoplastic etiology).

All patients underwent surgery. The time from the establishment of the diagnosis of AM to the introduction of surgical treatment ranged from 2 h to 11 days, with a mean of 1.62 days (±1.86) and median of 1.0 days. Surgical strategy was determined individually, dependent on the etiology, delay from the diagnosis establishment, local conditions and the patient's general state. The surgery, first of all, aimed at controlling the infection source and at limiting local inflammation by means of a wide cervical and/or mediastinal drainage through various surgical approaches. A detailed list of the performed surgical procedures and mortality rate of AM patients is presented in Table 1. The following methods were used in the treatment of 11 patients with iatrogenic complications following esophageal surgeries: primary repair (7), esophagectomy (2), and esophageal exclusion (2). Five patients died in this group. In the other 5 patients with iatrogenic etiology, a wide cervical and mediastinal drainage was the basic method of treatment. The next 2 patients required surgical repair of tracheal lesions. The last patient suffered from post-sternotomy mediastinitis. Debridement, revirage and mediastinal drainage were performed, but the patient died. Esophageal primary repair (7) and then tracheal repair (2) were the most frequently used methods of treatment of traumatic mediastinitis (11); all patients survived. One patient with esophageal perforation caused by a chemical burn required esophagectomy. The patient died. In the last patient from this group, a metal foreign body stabbed into the mediastinum was removed after a motorcycle accident. In 9 patients with mediastinitis of descending etiology, the basic method of treatment was to eradicate the primary focus of infection on the face or neck and wide cervical and mediastinal drainage (3 deaths). By far the worst results of treatment were noted in 5 patients with neoplastic etiology of the disease. All patients died despite esophageal exclusion (3), T-tube drainage (1) and primary repair (1).

Laboratory investigations

White blood cell count (WBC), red blood cell count (RBC), hemoglobin (HGB), hematocrit (HCT), platelet count, C-creative protein (CRP), procalcitonin (PCT), total protein, albumins, and electrolytes (sodium and potassium ions) were determined preoperatively. On day 3 after surgery the level of 3 markers of inflammation (WBC, CRP and PCT) were estimated.

Table 1. Aetiology and surgical procedures in patients with acute mediastinitis.

Aetiology	Number	Procedure	Death
Iatrogenic (19)			
Oesophageal endoscopy	4	PR-3 ESPH-1	1
Oesophagotomy	4	EX-2 PR-2	2
Nissen operation	2	ESPH-1 PR-1	1
Post-intubation tracheal rupture	2	PR-2	0
Complications of thyroid surgery	2	CRD+MDV-1 CRD+MDT-1	0
Oesophageal rupture during intubation	1	PR-1	0
Sternotomy complications in cardiac surgery	1	RVM-1	1
Colonic perforation to retroperitoneal space	1	MDV-1	0
Complications of neurosurgical procedures	1	CRD+MDV-1	0
Complications of cobbler's chest surgery	1	RVM-1	0
Traumatic (11)			
Oesophageal perforation	3	PR-3	0
Tracheal rupture	2	PR-2	0
Oesophageal rupture (foreign body)	3	PR-3	0
Boerhaave syndrome	1	PR-1	1
Burn of oesophagus with perforation	1	ESPH-1	0
Foreign body in mediastinum (traffic accident)	1	MDS-1	0
Descending (9)			
Dental abscess	4	DCUM- 2 CRD+MDT-2	1
Retropharyngeal abscess	3	DCUM- 2 CRD+MDV- 1	1
Peritonsillar abscess	2	CRD+MDT- 1 DCUM- 1	1
Neoplastic (4)			
Spontaneous perforation of prosthesis/stent	2	EX+PN-1 EX-1	2
Spontaneous oesophageal perforation in advanced cancer	3	PR-1 EX-1 TD-1	3

EX – exclusion; PR – primary repair; ESPH – oesophagectomy; MDT – mediastinal drainage by thoracotomy; MDV – mediastinal drainage by videothoracoscopy; MDS – mediastinal drainage by sternotomy; TD – T-tube drainage; CRD – cervical drainage; PN – pneumonectomy; DCUM – drainage of cervix and upper mediastinum; RVM – revirage and mediastinal drainage.

The determination of peripheral blood cell count parameters (RBC, HGB, HCT, PLT and WBC) was performed with a Pentra 120 DX hematology analyzer. The determination of the level of total protein, albumins, sodium and potassium ions as well as ultrasensitive CRP by immunoturbidimetric method with tris buffer and anti-CRP antibodies was performed with an Olympus AU 640 analyzer. The PCT level in human serum was measured with the use of electrochemiluminescence method (ECLIA) with a Cobas E411 analyzer.

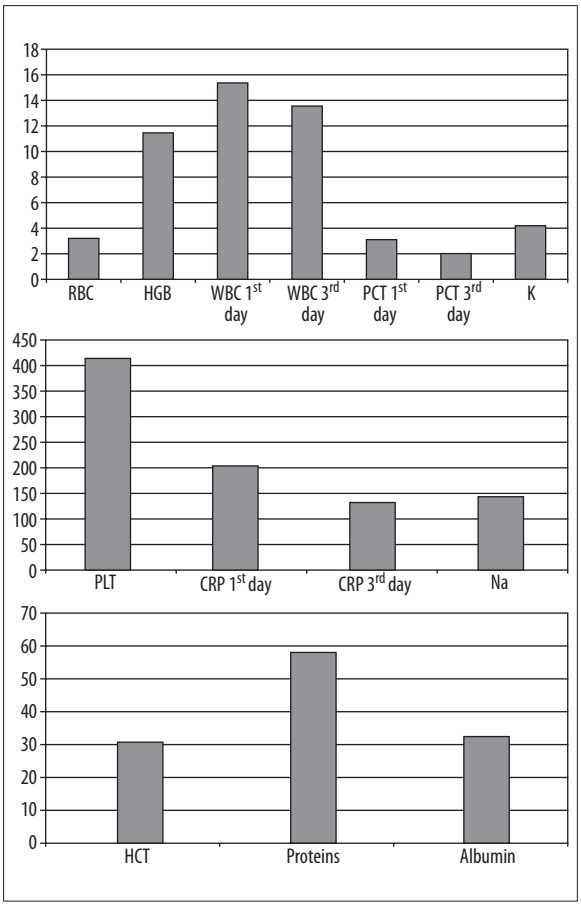

Figure 1. Average values of investigated biochemical parameters. The parameters were combined together in one figure concerning similar values of means: RBC [10^6/ml], HGB [mg/dL], WBC [10^3/dL], PCT [µg/dL], K [mEq/dL], PLT [10^5/dl], CRP [µg/dL], and Na [mEq/dL], HTC [%], Proteins [mg/dL], and Albumin [mg/dL].

Due to the retrospective nature of the study, no data were available that would allow us to assess the behavior of the analyzed markers in all patients in other definite time intervals after the surgery.

Statistical methods

ANOVA was applied to check the selected factors of the investigated parameters. As the variance differed significantly in the subgroups [Levene test], the Kruskal-Wallis test was used. Means of the parameters [numerical] were compared using the T-test for normal distribution or by medians and the W-test for non-normal distribution.

Results

Analysis of peripheral blood morphotic elements

Mean values of biochemical parameters analyzed in the study are presented in Figure 1.

The mean value of RBC was 3.41×10^6/µl, (median: 3.47), below the normal range. Moreover, insignificantly decreased levels of HGB with mean of 11.57 g/dl (median: 11.15) and HCT with mean of 30.94% (median: 31.5%) were observed. The mean level of PLT (412.92×10^3/µl) was elevated (median: 436.5).

The mean WBC count exceeded the upper limit of the normal range in the preoperative period (15.24×10^3/ml; median: 14.6), but 3 days after surgery its mean value decreased (13.39×10^3/ml; median 12.35). A statistically significant difference was confirmed between the value of medians for WBC pre- and postoperatively at the level of significance 95% ($p<0.01$).

In another analysis, selected laboratory markers were estimated in 2 groups of patients: "O" – recovery and "1" – death (Figure 2). Then, the mean value of selected laboratory parameters was evaluated for each group.

In the group of non-survivors, statistically significant lower values of RBC (p=0.0090), HGB (p=0.0286) and HCT (=0.0354) were observed. No similar dependence was found for PLT (p=0.6201) and preoperative WBC count (p=0.2960) (no statistical significance). However, WBC count on day 3 after surgery was significantly higher in group "1" (mean, 17.04×10^3/ml) in relation to group "O" (mean, 11.70×10^3/ml) (Figure 3). The result was of statistical significance (p=0.0002). It was calculated that the risk of death, when WBC count after surgery did not decrease, reached 67%.

Analysis of CRP level

The preoperative values of CRP were significantly elevated in relation to the whole material (Figure 1), with a mean of 202.89±50.12 mg/l (median, 191.8). Mean CRP level evaluated on day 3 postoperatively decreased but it did not exceed the upper normal range, with a mean of 132.25±78.19 mg/l, (median: 96.25) (Figure 2). There was a statistically significant difference between the medians of CRP pre- versus postsurgical treatment at the 95.0% confidence level ($p<0.00001$).

The CRP level was analyzed in the group of non-survivors and survivors (Figure 2). In the group of patients with fatal outcome, the initial CRP level was higher (mean, 230.54 mg/l) in relation to the group of survivors (mean, 189.99 mg/l). This correlation was statistically confirmed (p=0.0107). Three days after surgery the difference between mean CRP values was even more distinct (Figure 3). These values were significantly higher in group "1" (mean, 215.88 mg/l) compared to group "O" (mean, 93.22 mg/l). The result was of statistical significance ($p<0.0001$). If the CRP values did not decrease immediately after surgery, the death risk was estimated to be 88%.

Analysis of PCT values

The mean preoperative PCT value was 2.93 ng/ml (median, 1.35) and it was distinctly elevated (Figure 1). Three days after surgery it decreased to 2.0 ng/ml (median, 0.7); however, it still exceeded the normal range. There was a statistically significant difference between the medians of PCR pre- versus postoperatively at the 95% confidence level ($p<0.0001$) (Figure 2).

The preoperative mean PCT level was found to be much higher in the group of non-survivors (mean, 6.39 ng/ml)

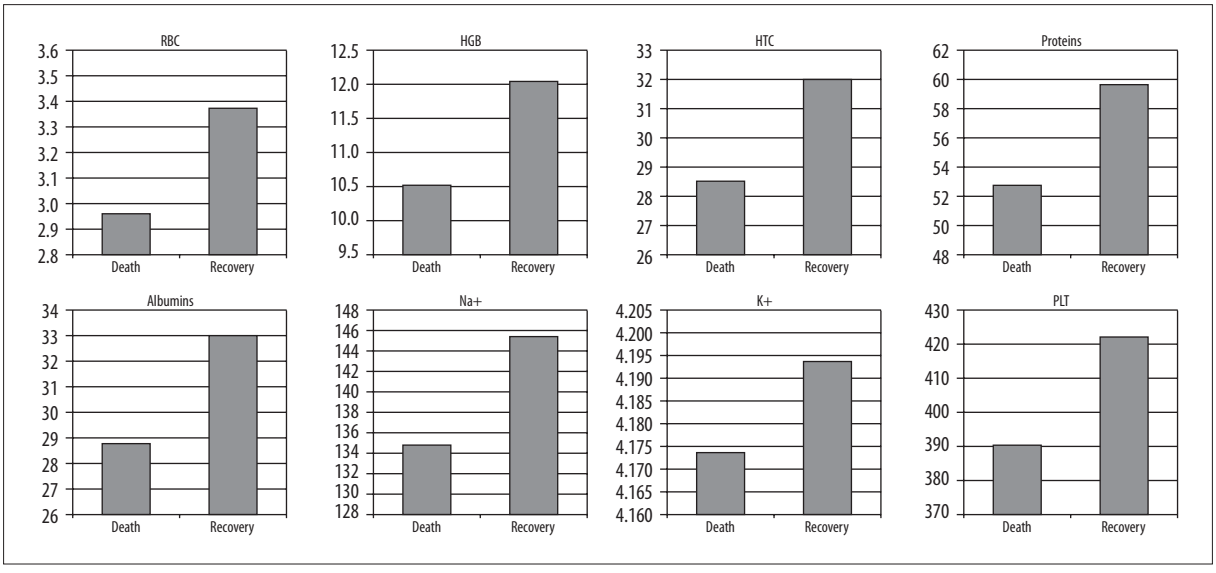

Figure. 2. Selected biochemical parameters in subgroups of patients [death] and [recovery].

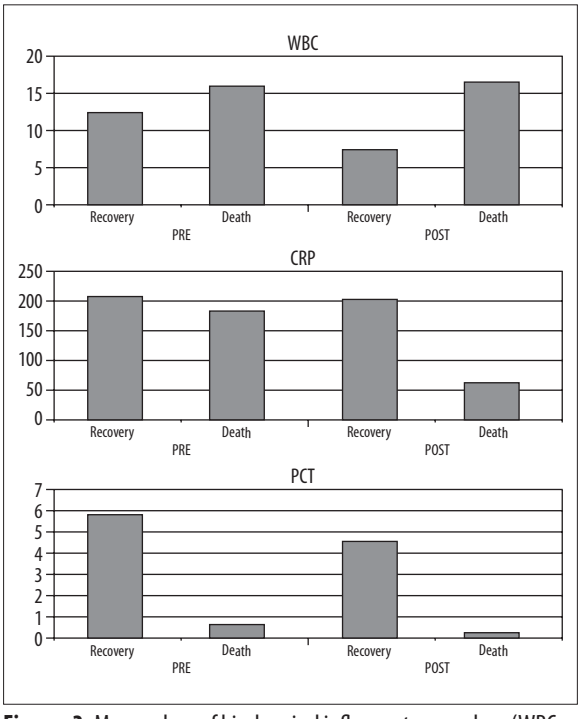

Figure. 3. Mean values of biochemical inflammatory markers (WBC, CRP and PCT) in the group of non-survivors and survivors in pre- and postoperative period.

than in the group of survivors (mean, 1.33 ng/ml). The mean preoperative value of this marker appeared to be higher in the dead patients than the value of the mean in all patients at a high level of statistical significance ($p<0.0001$) The PCT level estimated on day 3 after surgery was similar (Figure 3). It still remained high in the group of non-survivors (mean, 4.97 ng/ml), whereas in the group of survivors it was close to the normal range (mean, 0.61 ng/ml). This correlation was confirmed statistically ($p<0.0001$). Lack of PCT value decrease in response to the surgery was found to be associated with a nearly 100% death risk.

The level of total protein and albumin

Hypoproteinemia with hypoalbuminemia were observed in AM patients (Figure 1). The mean level of total protein was 57.39 g/l, (median, 59.15) and of albumin 31.68 g/l (median, 32.6). We investigated whether there were differences in the level of total protein and albumins between non-survivors and survivors (Figure 2). The obtained mean for the level of total protein was lower in the group of non-survivors (52.82 g/l) than in that of survivors (59.51 g/l) (Figure 3). The difference in the value of total protein was statistically significant ($p=0.0037$). Similar correlations were observed for the level of albumins (Figure 3). The mean level of albumins was lower in the group of non-survivors (28.73 g/l) vs. survivors (33.05 g/l) (Figure 5). The result was of statistical significance ($p=0.0003$).

Ionogram

The mean values of ionogram did not differ significantly from the normal range (Figure 1). The following values were obtained – for sodium ions, mean 141.92 mmol/l (median, 141.75); for potassium, mean 4.19 mmol/l (median, 4.1).

The distribution of ionogram values was investigated in the group of non-survivors and survivors (Figure 2). In the group of non-survivors the mean value for sodium ions was decreased to the level of 134.46 mmol/l (mild hyponatremia). In the group of survivors the mean value for sodium ions was within the normal range – 145.41 mmol/l. The result of statistical analysis ($p<0.0001$) confirmed the correlation between hyponatremia and the higher death risk. However, no significant deviations of the mean value for potassium ions were observed in both groups – non-survivors (4.17 mmol/l) and survivors (4.20 mmol/l).

Discussion

Anatomical conditions in the mediastinum, in which organs essential for life are surrounded by relatively loose connective tissue, contribute to rapid spread of the infection. In

response to the local inflammatory process and tissue damage there is a systemic response to infection in the form of acute-phase reaction, which can then turn into sepsis, septic shock and other complications leading to death.

It is thought that acute-phase response is induced by bacterial endotoxins and it takes place with pro-inflammatory cytokines (TNF-α, IL-6, IL-8), leading to the increase of body temperature and synthesis of acute-phase proteins such as C-reactive protein, haptoglobin, α-1-antitripsin, fibrinogen and others [12,13]. Considering the range of clinical symptoms, there was distinguished a systemic inflammatory response syndrome (SIRS), which is most frequently associated with sepsis. The criteria for SIRS and sepsis diagnosis have been defined by the American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference definitions of sepsis [10]. According to these guidelines, SIRS is diagnosed on the basis of the following clinical symptoms: fever, leucocytosis with the increase of polymorphic cells (or leucopenia), tachycardia and tachypnea. Sepsis is a heterogenic clinical entity which was defined on the basis of the occurrence of at least 2 or more physiological changes characteristic for SIRS in the course of documented infection or its suspicion [14].

In the course of AM, complications such as SIRS and sepsis, when treated ineffectively, can lead to the development of septic shock, multiorgan failure and death. Monitoring of the disease course is supported by widely understood laboratory diagnostics in correlation with the clinical picture and the results of diagnostic imaging. Deviations in the values of biochemical markers often signal the intensification of septic state long before the appearance of the first visible clinical symptoms. We made an attempt to identify negative biochemical prognostic factors in patients with AM, which could contribute to the improvement of the results of the treatment by expanding indications for diagnostics and by more aggressive qualification for surgery.

Determination of WBC when infection is suspected is the simplest and cheapest diagnostic investigation; however, it has low sensitivity and specificity [15].

In our patients, high leucocytosis was observed in the preoperative period, proving an active severe infection, and its decrease on day 3 after effective surgery. However, the mean preoperative WBC count appeared to be higher in patients whose treatment was unsuccessful. The high level of WBC on day 3 after surgery without distinct tendency to decrease, contrary to the patients who responded positively to surgery, was an important observation. This correlation was statistically confirmed (p=0.0002); thus, it should be regarded as a sign of poor prognosis. The increase of WBC count can also be induced by non-infectious factors such as myocardial infarction, catecholamines, corticosteroids and acute bleeding [16,17]. Moreover, the course of some infectious diseases can lack characteristic elevation of leucocytosis [16,18–20]. Thus, the value of leucocytosis itself in the diagnostics and monitoring of sepsis in the course of AM without combination with other data should be considered as limited.

Anemia in combination with other coexisting diseases is a known negative prognostic factor. Garson et al proved that anemia and substantial operative blood loss increase the death risk in patients with cardiovascular disease [21]. Welch et al demonstrated that mortality increases with the decrease of HGB concentration, and even mild forms of anemia increase the death risk [22]. According to Carson [23], the risk of death is low for patients with decreased HGB level of 7.1 to 8.0 g/dl. However, if HGB level drops in the postoperative period below 5 to 6 g/dl, the risk for morbidity and mortality rises significantly. In our patients, mean values of blood morphotic elements, except PLT, were decreased. The lower mean level of RBC, HGB and HCT found in patients whose treatment was unsuccessful is noteworthy. The performed statistical analyses of the level of RBC, HGB and HCT confirmed that anemia in patients with AM can be a negative prognostic factor but the number of blood platelets does not affect the final prognosis. Other authors have emphasized that thrombocytopenia can be an adverse prognostic factor [24–26].

Hypoproteinemia and hypoalbuminemia have a negative impact on the prognosis of critically ill patients [27–30]. This particularly concerns the patients after neoplastic [31], cardiosurgical [32] procedures and after severe injuries [33]. It is thought to be a consequence of malnutrition, liver dysfunction, disturbed protein and albumin synthesis by inflammatory mediators that can directly inhibit transcription of genes responsible for albumin synthesis [34,35]. Trauma and sepsis initiate a cascade of events that lead to accelerated protein degradation, decreased rate of synthesis of selected proteins, and increased amino acid catabolism and nitrogen loss [36]. The results of our studies are similar to the observations of other authors. We have confirmed that hypoproteinemia and hypoalbuminemia in patients with AM can be a factor associated with poor prognosis. The obtained values were of high statistical significance confirming this correlation – p=0.0037 for protein and p=0.0003 for albumins.

An altered ionogram is often observed in critical infectious diseases, which undoubtedly include AM. These alterations can result from the disease itself, cessation of food intake, past surgeries, accompanying fever, developing renal failure, and other factors. The decrease of the level of potassium ions can be an important parameter for the final prognosis. Schoenfeld and Barak demonstrated that hypophosphatemia is an interesting phenomenon observed in the early phase of sepsis, which correlates well with elevated level of pro-inflammatory cytokines [37–39]. The drop of potassium value is particularly suggestive in the case of gram-negative bacteria [40]. Hyponatremia is also a form of clinical manifestation of critical diseases, many of which have a high mortality rate [41,42]. Hyponatremia has a negative impact on the prognosis in oncological patients [43,44], cardiological patients [42,45], and in the elderly [46]. In our patients we did not observe marked alterations in ionogram values, except for decreased mean of the value of sodium ions in the group of non-survivors. Thus, hyponatremia in patients with AM can be regarded as a factor determining poor prognosis (p<0.0001).

In our patients no statistical correlation was found between the prognosis and serum level of potassium ions; however, many of them suffered earlier from chronic inflammatory diseases, neoplasms, underwent surgery, or had intravenous therapy which could have difficult-to-foresee impacts on ionogram alterations.

Acute phase proteins and C-reactive protein play a significant role in the assessment of the general condition and monitoring of septic patients [47–50].

Increased CRP levels can also be associated with poor prognosis in some chronic diseases such as heart failure [51,52].

CRP is a plasma gammaglobulin synthesized in the liver. Its level increases markedly after trauma, infection, inflammation and other factors stimulating tissue damage [53]. It is a good and sensitive marker of bacterial infection, the serum level of which increases significantly 6-10 h after activation of the stimulus inducing hepatocytes in liver, and drops rapidly when the stimulating signal is removed [54]. In most cases elevated CRP values are associated with unsuccessful treatment; however, there are reports that confirm this correlation [55]. In our patients the preoperative CRP values were high, proving severe infection. The level of this marker in non-survivors was observed to exceed mean values calculated for the whole study group, both in pre- and postoperative periods. Both these dependences found confirmation in statistics ($p=0.0107$ and $p=0.0001$). Thus, the initial high CRP level and lack of its decrease after surgical treatment should be associated with poor prognosis.

Procalcitonin (PCT) is an important biochemical marker useful in diagnosing and differentiation of severe infections including sepsis [56,57]. The bacterial toxin-induced increase of PCT concentration (with preserved insensitivity to other pro-inflammatory stimuli) and close correlation between PCT concentration and severity of infection are the most important properties of PCT. Thus, PCT is a laboratory marker of high specificity for sepsis. It has been suggested that the increase of PCT level >1.1 ng/ml in combination with SIRS within 24 h postoperatively allows prediction of postoperative complications, with a positive predictive value of 81% [58,59]. In our patients, elevated preoperative PCT values were observed from 0.5 to 18.3 ng/ml (mean: 2.93). Three days after surgery, the mean PCT level decreased gradually, coming close to normal values. It was found out that in the group of non-survivors, the mean PCT level was higher than in the whole group, both before the surgery and on day 3 after the procedure. Similar PCT values in response to the therapy should be considered as an important negative prognostic factor. High risk of death for patients with elevated PCT values after surgical treatment were statistically confirmed ($p<0.0001$).

Conclusions

We observed that a series of patients with AM died despite proper surgical treatment. We think that more attention should be paid to preoperative biochemical investigations as well as to monitoring of patients in the postoperative period, as their altered results provided useful information about septic complications ahead of the clinical picture and diagnostic imaging. High levels of inflammatory markers – CRP and PCT, anemia, hypoproteinemia with hypoalbuminemia as well as electrolyte alterations (hyponatremia) – appeared to be the most important biochemical factors associated with poor prognosis in the preoperative period. The death risk increases significantly if no distinct drop in WBC count and CRP and PCT level is observed immediately after surgery. In such a situation the patients should be qualified earlier for extended diagnostics and for reoperation.

Acknowledgement

The authors wish to thank professor Jacek Rysz, MD, PhD for making the hospitalized patients' data available, for his professional advice in preparing this article and for providing necessary support.

References:

1. Freeman RK, Vallières E, Verrier ED, Karmy-Jones R, Wood DE: Descendin necroziting mediastinitis: an analysis of the effects of serial surgical debridement on patient mortality. J Thorac Cardiovasc Surg, 2000; 119: 260–26
2. Estrera AS, Lanay MJ, Grisham JM et al: Descending necrotizing mediastinitis. Surg Gynecol Obstet, 1983; 157: 545–52
3. MartyAne CH, Alric P, Alauzen M: Descending necrotizing mediastinitis. Advantage of mediastinal drainage with thoracotomy. J Thoracic Cardiovas Surg, 1994; 107: 55–56
4. Brinster CJ, Sunil S, Lee L et al: Evolving options in the management of esophageal perforation. Ann Thorac Surg, 2004; 77: 1475–83
5. El Oakley RM, Wright JE: Postoperative Mediastinitis: Classification and Management. Ann Thorac Surg, 1996; 61: 1030–36
6. Carrol CL, Jeffrey RB, Federle MP et al: CT evaluation of mediastinal infections. J Comput Assist Tomogr, 1987; 11: 445–54
7. Akman C, Kantarci F, Cetinkaya S: Imaging in mediastinitis: a systematic review based on aetiology. Clinical Radiology, 2004; 59: 573–85
8. Singhal P, Kejriwal N, Lin Z et al: Optimal Surgical Management of Descending Necrotising Mediastinitis: Our Experience and Review of Literature. Heart, Lung and Circulation, 2008; 17: 124–28
9. Altorjay A, Kiss J, Vörös A, Szirányi E: The Role of Esophagectomy in the Management of Esophageal Perforations. Ann Thorac Surg, 1998; 65: 1433–36
10. American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference: definitions for sepsis and organ failure and guidelines on the use of innovative therapies in sepsis-Members of the American College of Chest Physicians/Society of Critical Care Medicine Consensus Conference Committee. Crit Care Med, 1992; 20: 864–74
11. Chan Y-L, Liao H-C, Tsay P-K et al: C-Reactive Protein as an Indicator of Bacterial Infection of Adult Patients in the Emergency Department. Chang Gung Med J, 2002; 25: 437–45
12. Zimmerman JJ: Sepsis/septic shock in Pediatric Critical Care Fuhrman BP, Zimmerman JJ (red) Mosby St. Louis, 1998; 1088–100
13. Marik PE, Varon J: Sepsis: State of the Art. Dis Mon, 2001; 47(10): 461–532
14. Bone RC, Balk RA, Cerra FB et al: Definitions for sepsis and organ failure and guidelines for the use of innovative therapies in sepsis. The ACCP/SCCM Consensus Conference Committee. American College of Chest Physicians/Society of Critical Care Medicine. Chest, 1992; 101: 1644–55
15. Weitzman M: Diagnostic utility of white blood cell and differential cell counts. Am J Dis Child, 1975; 129: 1183–89
16. Povoa P: C reactive protein: a valuable marker of sepsis.Intensive Care Med, 2002; 28: 235–43
17. Dale DC, Fauci AS, Guerry DI, Wolff SM: Comparison of agents producing a neutrophilic leukocytosis in man. Hydrocortisone, prednisone, endotoxin and etiocholanolone. J Clin Invest, 1975; 56: 808–13
18. Ugarte H, Silva E, Mercan D et al: Procalcitonin used as a marker of infection in the intensive care unit. Crit Care Med, 1999; 27: 498–504
19. Cunha J, Glória C, Vilela H, Lopes V: C-reactive protein: a good parameter for sepsis diagnosis (abstract). Intensive Care Med, 1997; 23: p61
20. Adnet F, Borron SW, Vicaut E et al: Value of C-reactive protein in the detection of bacterial contamination at the time of presentation in drug-induced aspiration pneumonia. Chest, 1997; 112: 466–71
21. Garson JL, Duff A, Poses RM et al: Effect of anaemia and cardiovascular disease on surgical mortality and morbidity. Lancet, 1996; 348: 1055–60
22. Welch HG, Mehan KR, Goodnough LT: Prudent strategies for elective red blood cell transfusion. Ann Intern Med, 1992; 116: 393–402
23. Carson JL, Noveck H, Berlin JA, Gould SA: Mortality and morbidity in patients with very low postoperative Hb levels who decline blood transfusion. Transfusion, 2002. 42(7): 812–18
24. Balk RA: Severe sepsis and septic shock: definitions, epidemiology, and clinical manifestations. Crit Care Clin, 2000; 16: 179–92

25. Strauss R, Wehler M, Mehler K et al: Thrombocytopenia in patients in the medical intensive care unit: bleeding prevalence, transfusion requirements, and outcome. Crit Care Med, 2002; 30: 1765–71
26. Raghavan M, Marik PE: Management of sepsis during the early,,Golden Hours". J Emerg Med, 2006; 31(2): 185–99
27. Goldwasser P, Feldman J: Association of serum albumin and mortality risk. J Clin Epidemiol, 1997; 50: 693–703
28. Herrmann FR, Safran C, Levkoff SE, Minaker KL: Serum albumin level on admission as a predictor of death, length of stay, and readmission. Arch Intern Med, 1992; 152: 125–30
29. McCluskey A, Thomas AN, Bowles BJ, Kishen R: The prognostic value of serial measurements of serum albumin concentration in patients admitted to an intensive care unit. Anaesthesia, 1996; 51: 724–27
30. Finfer S, Bellomo R, McEvoy M et al: Effect of baseline serum albumin concentration on outcome of resuscitation with albumin or saline in patients in intensive care units: analysis of data from the Saline versus Albumin Fluid Evaluation (SAFE) Study. BMJ (Clinical Research Ed.), 2006; 333: 1044
31. Ryan AM, Hearty A, Prichard RS et al: Association of hypoalbuminemia on the first postoperative day and complications following esophagectomy. J Gastrointest, Surg, 2007; 11: 1355–60
32. Fritz HG, Brandes H, Bredle DL et al: Post-operative hypoalbuminaemia and procalcitonin elevation for prediction of outcome in cardiopulmonary bypass surgery. Acta Anaesthesiol Scand, 2003; 47: 1276–83
33. Schirmer-Mikalsen K, Vik A, Gisvold SE et al: Severe head injury: control of physiological variables, organ failure and complications in the intensive care unit. Acta Anaesthesiol Scand, 2007; 51: 1194–201
34. Moshage HJ, Janssen JA, Franssen JH et al: Study of the molecular mechanism of decreased liver synthesis of albumin in inflammation. J Clin Invest, 1987; 79: 1635–41
35. Brenner DA, Buck M, Feitelberg SP, Chojkier M: Tumor necrosis factor-alpha inhibits albumin gene expression in a murine model of cachexia. J Clin Invest, 1990; 85: 248–55
36. Biolo G, Toigo G, Ciocchi B et al: Metabolic Response to Injury and Sepsis: Changes in Protein Metabolism. Nutrition, 1997; 13(9 Suppl.): 52–57
37. Shoenfeld Y, Hager S, Berliner S et al: Hypophosphatemia as a diagnostic aid in sepsis. NY State J Med, 1982; 82: 163–65
38. Shoenfeld Y, Khoury M, Ben-Yehuda O et al: Hypophosphatemia in sepsis. Eur J Intern Med, 1991; 2: 67–71
39. Barak V, Schwartz A, Kalickman I et al: Prevalence of Hypophosphatemia in Sepsis and Infection: The Role of Cytokines. AmJ Med, 1998; 104: 40–47
40. Riedler CF, Scheitlin WA: Hypophosphatemia in septicemia: higher incidence in gram-negative than in gram-positive infections. BMJ, 1969; 1: 753–56
41. Lee C-T, Guo H-R, Chen J-B: Hyponatremia in the emergency department. Am J Emerg Med, 2000; 18: 264–68
42. Kłopotowski M, Kruk M, Przyluski J et al: Sodium level on admission and in-hospital outcomes of STEMI patients treated with primary angioplasty: The ANIN Myocardial Infarction Registry. Med Sci Monit, 2009; 15(9): CR477–83
43. Langfeldt LA, Cooley ME: Syndrome of inappropriate antidiuretic hormone secretion in malignancy: review and implications for nursing management. Clin J Oncol Nurs, 2003; 7: 425–43
44. Poe CM, Taylor LM: Syndrome of inappropriate antidiuretic hormone: assessment and nursing implications. Oncol Nurs Forum 16: 373–381 Silverman P, Distelhorst CW (1989) Metabolic emergencies In clinical oncology. Semin Oncol, 1989; 16: 504–15
45. Brophy JM, Deslauriers G, Rouleau JL: Long-term prognosis of patients presenting to the emergency room with decompensated congestive heart failure. Can J Cardiol, 1994; 10(5): 543–47
46. Terzian C, Frye EB, Piotrowski ZH: Admission hyponatremia in the elderly: Factors influencing prognosis. J Gen Intern Med, 1994; 9: 89–91
47. Blackburn WD Jr: Validity of acute phase proteins as markers of disease activity. J Rheumatol Suppl, 1994; 42: 9–13
48. Castelli GP, Pognani C, Cita M et al: Procalcitonin, C-reactive protein, white blood cells and SOFA score in ICU: diagnosis and monitoring of sepsis. Minerva Anestesiol, 2006; 72: 69–80
49. Chung JL, Kong MS, Lin SL et al: Diagnostic value of C-reactive protein in children with perforated appendicitis.Eur J Pediatr, 1996; 155: 529–31
50. Holm A, Pedersen SS, Nexoe J et al: Procalcitonin versus C-reactive protein for predicting pneumonia in adults with lower respiratory tract infection in primary care. Br J Gen Pract, 2007; 57: 555–60
51. Ridker PM: Inflammatory biomarkers and risks of myocardial infarction, stroke, diabetes, and total mortality: implications for longevity. Nutr Rev, 2007; 65(Suppl.): S253–59
52. Kozdağ G, Ertaş G, Kılıç T et al: Elevated level of high-sensitivity C-reactive protein is important in determining prognosis in chronic heart failure. Med Sci Monit, 2010; 16(3): CR156–61
53. Pepys MB, Baltz ML: Acute phase proteins with special reference to C-reactive protein and related proteins (pentraxins) and serum amyloid A protein. Adv Immunology, 1983; 34: 141–212
54. Hind CRK, Pepys MB: Acute phase proteins. In: LessofMH, Lee TH, Kemeny DM (eds.), Allergy: an international textbook. New York: John Wiley & Sons, 1987: 237–53
55. Matson A, Soni N, Sheldon J: C-reactive protein as a diagnostic test of sepsis in the critically ill. Anaesth Intensive Care, 1991; 19: 182–86
56. Dalton H: Procalticonin: a predictor of lung injury attributable to sepsis. Crit Care Med, 1999; 25: 2304–8
57. Whang KT, Vath SD, Becker KL et al: Procalticonin and proinflamatory cytokine interactions in sepsis. Shock, 2000; 4: 73–78
58. Clec'h C, Fosse JP, Karoubi P et al: Differential diagnostic value of procalcitonin in surgical and medical patients with septic shock. Crit Care Med, 2006; 34(1): 102–7
59. Monkhouse D: Postoperative skepsis. Current Anaesthesia & Critical Care, 2006; 17: 65–70