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Released granulocytic elastase: An indicator of pathobiochemical alterations in septicemia after abdominal surgery

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To discover the role of lysosomal enzyme release from polymorphonuclear (PMN) leukocytes during septicemia, plasma levels of PMN elastase were measured with a newly developed enzyme-linked immunosorbent assay for detection of the PMN elastase- α_1 -proteinase inhibitor complex (E- α_1 PI). Plasma samples from 41 patients were assayed continuously before and after major abdominal surgery. The patients were divided into a group without infection (group A) and two septicemia groups (survivors in group B and nonsurvivors in group C). The E- α_1 PI levels of the 11 patients in group A without any signs of pre- or postoperative infection were in the normal range (a normal value of 86.5 ± 25.5 ng/ml has been reported in 153 healthy subjects), except for a small increase to 208.8 ± 25.6 ng/ml 12 hours after surgery. When septicemia was confirmed clinically in patients in groups B and C, the E- α_1 PI levels rose on average to six times the norm in group B (649.9 ± 116.3 ng/ml) and to more than 10 times the norm in group C (985.0 ± 154.6 ng/ml). Peak values $>2,200$ ng/ml could be measured in both groups. In patients in group B, the E- α_1 PI levels returned to normal during recovery, while in those in group C they remained significantly elevated (560.5 ± 174.7 ng/ml) until death. Correlations were demonstrated between the amount of elastase released into the circulation and the decrease in the activities of antithrombin III, coagulation factor XIII, and α_2 -macroglobulin, as well as the increased C-reactive protein in plasma. We conclude that release of elastase and other lysosomal factors from PMN cells plays a major role in the pathobiochemical alterations during septicemia. In addition, significantly elevated E- α_1 PI levels in the postoperative course seem to be a suitable indicator for onset and persistence of sepsis as well as of the severity of this disorder in patients after major surgery.

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DESPITE RECENT PROGRESS in prophylaxis and therapy of postoperative sepsis, the high mortality rate is still a major problem (30% to 70% after septicemia, >70% after septic shock).²⁰⁻²² The underlying pathomechanisms of multiple organs failure due to septicemia are not sufficiently understood. Lysosomal proteinases as well as hydrogen peroxide or oxygen radicals released

from stimulated or disintegrating polymorphonuclear (PMN) leukocytes enhance the inflammatory response by destruction of connective tissue structures and cell membrane constituents^{11, 33} as well as plasma proteins either by proteolytic degradation or denaturation by oxidation. There may be a release of toxic peptides.^{2, 6, 7, 17, 29-32}

In this study we used PMN elastase as a marker of such pathologic release reactions. The extracellularly liberated elastase competes with susceptible protein substrates including α_1 -proteinase inhibitor (α_1 PI) and α_2 -macroglobulin (α_2 M), being finally eliminated as inactive enzyme-inhibitor complexes (90% as α_1 PI complex and 10% as α_2 M complex) via the reticuloendothelial system.^{25, 27} Therefore, the PMN elastase-

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Table I. Clinical data of the three patient groups

Patient No.	Age (yr)	Sex	Malignancy	Operation	Source of sepsis	Onset of sepsis (days after surgery)	Outcome*
Group A (without infection)							
1	45	M	+	Rectum resection			15
2	40	M	-	Choledochojejunostomy			10
3	48	M	+	Gastrectomy			22
4	67	F	-	Colon resection			12
5	62	F	-	Colon resection			19
6	29	M	+	Whipple procedure			13
7	51	M	-	Stomach resection			8
8	69	F	+	Colon resection			11
9	63	F	+	Colon resection			10
10	31	F	+	Papillotomy			9
11	24	M	-	Colon resection			16
Group B (sepsis with recovery)							
1	69	F	-	Colon resection	Peritonitis	1	11
2	20	M	-	Portocaval shunt	Pneumonia	2	12
3	50	M	-	Stomach resection	Peritonitis	6	9
4	49	F	+	Gastrectomy	Peritonitis	8	19
5	62	M	-	Liver resection	IA	1	10
6	40	F	-	Papillotomy	IA	1	12
7	70	M	+	Pancreas resection	IA	1	22
8	79	F	-	Papillotomy	Pneumonia	4	8
9	30	M	-	Replantation of thigh	Wound abscess	3	13
10	52	M	-	Pancreas resection	Peritonitis	2	14
11	45	M	-	Jejunum resection	Peritonitis	1	4
12	65	M	+	Jejunum resection	IA	1	14
13	43	M	-	Pancreas resection	IA	2	18
14	77	F	+	Ileum resection	IA	1	6
Group C (sepsis with death)							
1	58	M	+	Pancreas resection	Peritonitis	7	9
2	54	M	-	Stomach resection	Peritonitis	6	81
3	40	F	-	Stomach resection	Peritonitis	22	12
4	67	M	+	Rectum extirpation	Peritonitis	6	57
5	79	M	+	Rectum extirpation	Pneumonia	5	6
6	60	M	+	Pancreas resection	Peritonitis	3	10
7	51	M	-	Portocaval shunt	Pneumonia	7	23
8	50	F	-	Pancreas resection	Peritonitis	41	8
9	68	M	+	Colon resection	Wound abscess	27	23
10	46	M	-	Stomach resection	Peritonitis	11	30
11	69	F	+	Ileum resection	Peritonitis	1	6
12	68	M	-	Pancreas resection	Peritonitis	1	32
13	60	M	+	Gastrectomy	Peritonitis	5	12
14	63	M	-	Jejunum resection	Peritonitis	1	11
15	73	F	-	Stomach resection	Peritonitis	1	9
16	78	F	-	Colon resection	Peritonitis	1	1

Legend: M = male; F = female; IA = intra-abdominal abscess.

*Group A, discharge (days after surgery); group B, recovery (days after onset); group C, death (days after onset).

α_1 PI (E- α_1 PI) complex should be a suitable indicator for lysosomal enzyme release. The aims of our study were threefold: (1) to show the influence of major surgery on PMN elastase release; (2) to verify the

effect of postoperative infections on E- α_1 PI levels; and (3) to investigate a possible correlation between E- α_1 PI levels, patient outcome, and the consumption of important blood proteins.

MATERIAL AND METHODS

Patients. From March 1980 to June 1981, 41 patients were investigated according to the study protocol before major abdominal surgery and during the postoperative course. Patients were attributed to one of three clearly defined groups. In group A, the 11 patients showed no signs of infection pre- and postoperatively. In group B, the 14 patients had septicemia in the postoperative course but survived. In group C, the 16 patients died due to septicemia after major abdominal surgery. Clinical data of the three patient groups are listed in Table I. The diagnosis of septicemia was established by: (1) clearly defined source(s) of sepsis with positive culture of the infectious organisms; (2) leukocyte counts $>15,000/\mu\text{l}$ or $<5000/\mu\text{l}$; (3) platelet counts $<100,000/\mu\text{l}$ or a platelet drop $>30\%$ within 24 hours. Positive blood culture was not presupposed but was registered in 30% of patients in group B and in 50% of patients in group C. Septic shock (defined by cardiac index >6 L/min/m² and systemic vascular resistance <600 dynes \cdot sec/cm⁵) could be demonstrated in nine patients in group C but in no patient in group B.

Sampling procedure. Blood sampling and registration of all clinical parameters were performed 12 hours before surgery, 12 hours after surgery, and every 12 hours thereafter until discharge (group A), recovery from any sign of infection (group B), or death due to septicemia (group C).

Apart from all laboratory data normally monitored for seriously ill patients, plasma samples for detection of specific parameters such as E- α_1 PI, antithrombin III (AT III), coagulation factor XIII (F XIII), α_2 M, and C-reactive protein (CRP) were obtained by drawing 4.5 ml of venous blood into plastic syringes containing 0.5 ml of sodium citrate (2.2 gm per 100 ml distilled water). Plasma was separated from blood cells within 30 minutes after sample collection to prevent leakage of leukocyte constituents. Plasma samples were stored at -70°C until assayed. Plasma levels of patients receiving banked blood with high levels of complexed elastase^{12,14} were corrected for the transfused amount, taking into account the elimination rate of the complex in vivo (half-life equals 1 hour).

Bacteriologic data. Microbiologic examinations were performed in wound secretions, abdominal drainages, tracheobronchial secretions, closed drainage systems of indwelling urethral catheters, and blood. Specimens were collected every 2 days. Additional indications for performing blood cultures were a sudden increase in the patient's temperature ($>38.5^\circ\text{C}$), a change in sensorium, and the onset of chills. In these

cases, samples were taken at least three times within a 24-hour period. Other secretions were collected by established techniques. Results of microbiologic cultures and antimicrobial susceptibility tests were tabulated by 2 days after collection.

Hematologic data. Leukocytes were counted by an electronic counter (Coulter Counter, model B, Coulter Electronics Inc., Hialeah, Fla.) and thrombocyte counts were performed with the Neubauer counting chamber (Assistant, Sontheim, West Germany).

Plasma factors

Enzyme-linked immunosorbent assay for E- α_1 PI. With the technique of Neumann et al.,^{23,24} E- α_1 PI was determined by a two-site sandwich enzyme-linked immunosorbent assay including antisera against both elastase and α_1 PI (E. Merck, Darmstadt, West Germany). Concentrations (in nanograms per milliliter) are given for the amount of complexed elastase only.

AT III, F XIII, α_2 M, and CRP. The inhibitory activity of AT III against thrombin was determined with use of the chromogenic peptide substrate S-2238 (Deutsche Kabi, Munich, West Germany). The biologic activity of the fibrin-stabilizing factor F XIII was measured by a commercial test system (Factor XIII-Schnelltest; Behring Werke AG, Marburg, West Germany). Both assays were performed as previously described.¹⁶ Plasma levels of F XIII subunits A and S were detected according to Laurell¹⁹ with the use of monospecific antisera from Behring Werke AG (Clotimmun-Faktor XIII-A, Clotimmun-Faktor XIII-S). The inhibitory activity of α_2 M was determined with a commercial test system (α_2 -Macroglobulin Test Combination; Boehringer, Mannheim, West Germany) according to Ganroth.⁹ Plasma concentrations of CRP and α_2 M were evaluated by a radial immunodiffusion technique with standardized immunodiffusion plates (LC Partigen CRP, M-Partigen α_2 -Makroglobulin; Behring Werke AG).

Classification of data. To allow comparison between measured data, mean values are presented for identical clinical periods: period 1, 12 hours before operation; period 2, 12 hours after operation; period 3, postoperative course without infection (group A) or before onset of sepsis (groups B and C); period 4, time of onset of sepsis (groups B and C); period 5, course after onset of sepsis (groups B and C); and period 6, day of discharge (group A), day of recovery from infection (group B), or day of death due to septicemia (group C).

Statistics. Unless otherwise stated, results are given as the percentage of the value obtained in a standard

Table II. Mean values (\pm SEM) E- α_1 PI (ng/ml)

Group	Period 1 (12 hr before operation)	Period 2 (12 hr after operation)	Period 3 (postoperative course)	Period 4 (onset of sepsis)	Period 5 (course of sepsis)	Period 6 (discharge, recovery, or death)
A	90.9 \pm 7.6	208.8 \pm 25.6	143.8 \pm 10.8			96.0 \pm 5.6
B	99.2 \pm 18.6	258.9 \pm 25.9	316.4 \pm 25.0	646.9 \pm 116.3	266.1 \pm 35.4	93.2 \pm 7.6
C	353.4 \pm 71.5	347.3 \pm 46.6	229.1 \pm 15.9	985.0 \pm 154.6	517.6 \pm 41.6	560.5 \pm 174.7
Student <i>t</i> test						
A:B	NS	NS	$p < 0.0005$			NS
A:C	$p < 0.025$	$p < 0.01$	$p < 0.0005$			$p < 0.0125$
B:C	$p < 0.025$	NS	$p < 0.0125$	NS	$p < 0.0005$	$p < 0.0125$

Table III. Mean values (\pm SEM) of AT III (% of normal activity), F XIII (% of normal activity), and α_2 M (% of preoperative inhibitory activity) in patients in groups A, B, and C

	Period 1 (12 hr before operation)	Period 2 (12 hr after operation)	Period 3 (postoperative course)	Period 4 (onset of sepsis)	Period 5 (course of sepsis)	Period 6 (discharge, recovery, or death)
Group A						
AT III	97.1 \pm 7.6	83.8 \pm 5.4	83.8 \pm 1.9			103.8 \pm 6.7
F XIII	71.9 \pm 3.1	43.7 \pm 6.2	44.4 \pm 1.2			65.6 \pm 10.7
α_2 M	100	87.5 \pm 3.6	87.7 \pm 1.8			97.3 \pm 4.2
Group B						
AT III	84.3 \pm 10.2	64.9 \pm 9.2	53.6 \pm 9.4	50.4 \pm 4.4	62.4 \pm 2.1	93.0 \pm 8.3
F XIII	68.0 \pm 6.5	53.2 \pm 5.9	45.0 \pm 3.8	37.5 \pm 8.8	40.6 \pm 3.1	88.6 \pm 8.1
α_2 M	100	63.5 \pm 6.2	61.2 \pm 6.6	50.0 \pm 4.9	57.8 \pm 1.5	75.4 \pm 5.5
Group C						
AT III	81.1 \pm 14.3	77.6 \pm 7.6	56.7 \pm 3.8	50.4 \pm 5.1	52.1 \pm 2.5	45.8 \pm 6.2
F XIII	70.0 \pm 13.3	62.5 \pm 7.2	49.7 \pm 2.0	46.8 \pm 5.9	29.9 \pm 4.3	28.1 \pm 10.6
α_2 M	100	77.3 \pm 2.6	72.6 \pm 1.3	62.2 \pm 4.5	69.8 \pm 1.5	72.2 \pm 6.2

plasma pool (mean \pm SEM). Statistical evaluation was performed by the Student *t* test; p values ≤ 0.05 were considered significant.

RESULTS

Septic parameters

Primary source of septicemia and bacteriologic results. Clinical data are summarized in Table I. Peritonitis was the primary source of septicemia in patients in groups B and C, followed by pneumonia and wound abscess. Bacteriologic data from a septical focus showed mainly mixed cultures. In patients in group C, gram-negative bacteria and fungal infections predominated (15 patients in group C and eight in group B). More than 10^4 colony forming units of *Candida albicans* were found in three patients in group B and in 12 in group C. Gram-positive cocci were equally distributed (10 and 11 patients in groups B and C, respectively). Anaerobic bacteria were detected in five patients in group B and only one patient in group C. Blood cultures were found to be positive during

onset of sepsis in patients 1, 5, and 8 in group B and in patients 1, 3, 5, 6, 10, 11, 15, and 16 in group C.

Temperature, leukocyte count, and thrombocyte count. Patients in group A showed a slight rise in temperature ($<38^\circ$ C) after surgery but no significant changes in leukocyte or thrombocyte counts compared with normal values. During infection in patients in groups B and C we recorded temperatures $>38^\circ$ C, leukocyte counts $>12,000/\mu\text{l}$ or $<5000/\mu\text{l}$, and platelet counts $<125,000/\mu\text{l}$. Four patients in group B (Nos. 1, 3, 9, and 12) and four in group C (nos. 1, 8, 13, and 15) showed leukopenia <5000 cells/ μl during the onset of sepsis. In 10 of 14 patients in group B and in 11 of 16 patients in group C an absolute level $<100,000$ thrombocytes/ μl could be measured during onset of sepsis. In the other patients in both groups the thrombocyte count was diminished $>30\%$ compared with the level 24 hours earlier.

Plasma factors

E- α_1 PI. The mean (\pm SEM) values in each group for the different periods are listed in Table II. In

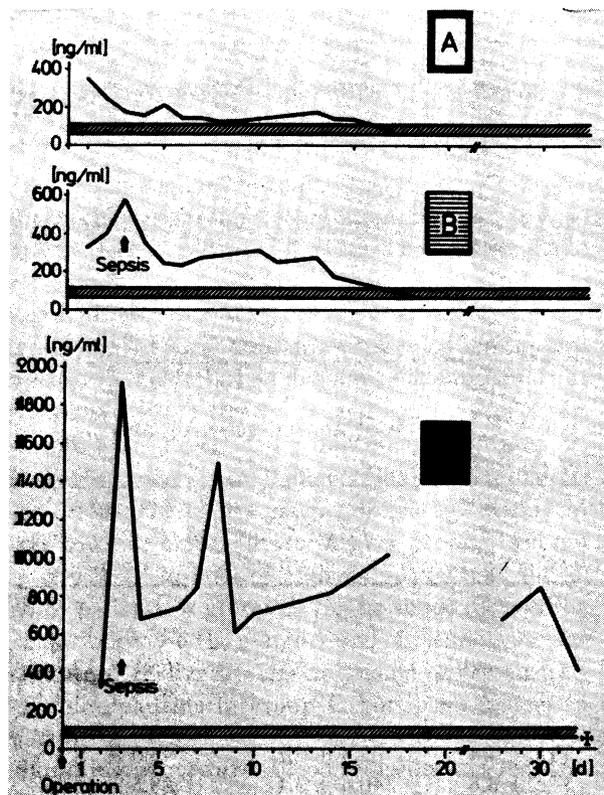


Fig. 1. Typical individual graphs of E- α_1 PI during the postoperative course of patients in groups A (without infection; patient no. 3), B (sepsis with recovery; patient no. 7), and C (sepsis with death; patient no. 12).

patients without any sign of preoperative infection (groups A and B), the operative trauma was followed by an increase of E- α_1 PI levels up to threefold the norm (86.5 ± 25.5 ng/ml). Group C showed significantly elevated E- α_1 PI levels before operation compared with the norm; a slight but not significant decrease could be observed 12 hours after operation. This is because six of 16 patients of these group were operated on because of peritonitis. They had higher leukocyte counts ($11,600 \pm 2600$ cells/ μ l, i.e., significantly more than in groups A and B) but showed no other sign of septicemia.

In the later postoperative phase there was clear normalization in group A, whereas the E- α_1 PI levels in groups B and C showed a moderate but not significant elevation compared with the levels 12 hours after operation. At the onset of septicemia, however, a highly significant increase in the E- α_1 PI levels could be detected, up to sixfold in group B and up to tenfold in group C. Peak levels were >2200 ng/ml in both groups. The E- α_1 PI levels of patients with sepsis who

recovered showed a clear tendency toward normalization. When septicemia persisted, however, levels of E- α_1 PI were high until death.

Fig. 1 demonstrates typical individual graphs of the E- α_1 PI concentration for one patient in each group. While group A showed a moderate elevation of E- α_1 PI levels in the early phase after surgery only, in group B higher peak levels as well as a prolonged period of elevated E- α_1 PI levels was observed. Group C showed very high levels several times and no tendency towards normalization. At the time of death the levels were still high.

AT III, F XIII, and α_2 M. Concomitant with the increase in E- α_1 PI there was a significant decrease of AT III, F XIII, and α_2 M (Table III). These proteins or inhibitors are known to be easily susceptible to proteolytic degradation or cleavage (α_2 M) by elastase or other lysosomal proteinases. The diminished activities at onset of septicemia were normalized in all patients overcoming the infection, whereas a further significant decrease was found in group C (lethal outcome).

Values for α_2 M are given as the percentage of preoperative levels, because they were already low before surgery. Thereby, remarkable differences were found between the patients with and without sepsis. While the highest loss of inhibitory activity was only 13% in group A, it reached 50% in group B and 40% in group C. At the time of the last measurement, group A showed normal values, whereas those in groups B and C were still significantly decreased. Remarkably, both the inhibitory activity of α_2 M and the immunologically measured concentration were likewise diminished. Forty-three pairs of values of both measurements were selected for linear regression analysis. The correlation coefficient ($r = 0.9155$) confirmed that there was no difference between concentration and inhibitory activity of α_2 M within the three groups after major surgery.

CRP. Preoperative values were found to be close to normal (0.05 to 1.0 mg/100 ml¹⁸) in groups A and B but were elevated in group C (group A, 1.8 ± 0.9 mg/100 ml; group B, 2.8 ± 1.7 mg/100 ml; group C, 4.5 ± 2.1 mg/100 ml). During the postoperative course, CRP plasma levels could be differentiated between patients without infection (group A, 6.1 ± 0.7 mg/100 ml) and those showing signs of beginning septicemia (group B, 14.9 ± 1.6 mg/100 ml; group C, 15.1 ± 1.5 mg/100 ml). During sepsis onset, both groups reached equally high CRP levels (group B, 15.1 ± 1.4 mg/100 ml; group C, 15.1 ± 2.6 mg/100 ml). During sepsis there was a decrease in CRP in both

groups, but this did not reflect patient outcome (group B, 9.7 ± 0.7 mg/100 ml; group C, 9.9 ± 0.7 mg/100 ml). Only at the time of the last measurement were values in groups A and B again normal, but those of group C were further elevated (group A, 2.4 ± 0.6 mg/100 ml; group B, 2.5 ± 0.6 mg/100 ml; group C, 8.8 ± 1.4 mg/100 ml).

DISCUSSION

Detailed analyses of various components of the clotting, fibrinolysis, complement, and kallikrein-kinin systems performed in clinical as well as experimental studies indicate continuous activation of these cascade systems during septicemia, septic shock, or endotoxemia.^{1-5, 8, 15, 16} Leukocytes, especially the PMN cells, are supposed to play a central role in such pathobiochemical events.^{6, 17, 32} Recently, Aasen et al.² observed in an experimental study (lethal endotoxin shock in dogs) a relationship between the initial drop in leukocyte levels (probably combined with degranulation of these cells) and the appearance of E- α_1 PI in plasma. They attributed the disturbances or activation of the cascade systems mainly to the action of liberated leukocytic proteinases.

So far, only Egbring et al.⁵ have tried to measure E- α_1 PI levels in patients with sepsis by rocket immunoelectrophoresis.¹⁹ However, levels of complexed elastase as low as 0.1 μ g/ml plasma cannot be quantitated by this method. Moreover, from this study no statement is available concerning the course of E- α_1 PI levels during septicemia. With the newly developed enzyme-linked immunosorbent assay, the detection limit of E- α_1 PI is 0.2 ng per assay, i.e., 20 ng per milliliter of plasma. Therefore, E- α_1 PI levels in plasma samples of healthy individuals (mean value 86.5 ± 25.5 ng/ml) could be accurately measured.²³ The presence of the complex in normal plasma confirms the assumption of a continuous release of low doses of lysosomal PMN proteases into the circulation.

Our data are comparable with those of Ohlsson and Olsson,²⁶ who found 135 ng of granulocytic elastase per milliliter of freshly drawn normal human serum applying a specific radioimmunoassay. With a radioimmunoassay developed by Plow and Plescia,²⁸ elastase-related antigen was assessed to be 24 ng/ml in five normal plasma samples. The corresponding serum specimens showed a mean value of 326 ng/ml. Because we observed similar irregular rises in the E- α_1 PI level during serum formation, plasma samples should be used exclusively in clinical studies.

To our knowledge, our study represents the first and most extensive documentation of granulocytic elastase

release during septicemia after major surgery. In patients without preoperative infections, surgical trauma was followed by a moderate liberation of elastase, most likely as a result of enhanced phagocytotic activity of leukocytes. The a priori elevated E- α_1 PI level in patients who already have preoperative infections showed no further increases after surgery. Thus the release of elastase induced by surgical trauma might have been compensated for by a reduced leukocyte response due to elimination of the infectious focus.

In the postoperative phase, the mean E- α_1 PI concentrations correlated well with the severity of infection. Highly elevated E- α_1 PI levels at sepsis onset were normalized in those patients recovering from septicemia, whereas in the fatal cases a continuous increase of the complex levels was measured. Possibly, the high endotoxin levels (determined by a modified Limulus test)* in the plasma specimens of some of the latter patients were responsible for this enhanced elastase liberation.

Parallel to the increase in E- α_1 PI levels there was a decrease in levels of F XIII subunit S (carrier protein) in groups B and C, although the absolute degree of carrier protein consumption did not correspond in each case to the amount of E- α_1 PI. On the other hand, patients without infection (group A) and normal or only slightly elevated E- α_1 PI levels had F XIII subunit S concentrations in the normal range but often clearly reduced fibrin-stabilizing activity (due to F XIII subunit A). As demonstrated by Egbring et al.⁵ and Ikematsu et al.,¹⁰ reduction of both subunits of F XIII cannot be due to activation of the clotting cascade alone. During clotting, i.e., by the activation of thrombin, subunit A is consumed simultaneous with the F XIII activity, but subunit S is not. These data and our results suggest that in patients suffering from septicemia, unspecific proteolytic degradation by granulocytic elastase or other lysosomal proteinases is involved to a significant degree in the depletion of F XIII.

Moreover, the very low AT III activity in patients with permanently elevated E- α_1 PI levels may also be due, at least in part, to degradation by elastase. This conclusion is based on recent in vitro studies showing that purified AT III is rapidly inactivated by isolated granulocytic elastase.¹³

α_2 M, probably the most important proteinase inhibitor of the organism, is responsible for the inhibition and elimination of nearly all types of neutral and acid proteinases liberated from various body cells under physiologic and pathologic conditions. The half-life of

*Stemberger A: Personal communication.

α_2 M-proteinase complexes in the circulation is believed to be 10 to 12 minutes.²⁵ In our study no difference between activity and concentration of α_2 M could be observed. From this fact we conclude that the clearance function of the reticuloendothelial system was not impaired in our patients, although high amounts of lysosomal proteinases were liberated.

In contrast to the consumption of diverse plasma proteins, a marked rise of the acute-phase reactants occurs during infection. The acute-phase reaction, in which proteins of different function and origin participate, is thought to represent a systemic host response to injury.³⁴ Of all known factors, CRP reacts most rapidly and significantly. Because of its various biologic functions (e.g., recognition of microorganisms; activation of the classic complement pathway; reaction with lymphoid cells, phagocytic cells, and platelets¹⁸, CRP seems to play a central role in unspecific host defense. Therefore, comparison of the acute-phase response of CRP with the degree of liberated granulocytic elastase was of special interest to us. Preoperative and postoperative values of CRP and E- α_1 PI in the control and septicemia groups designed both factors as similarly sensible to the inflammatory stimulus. However, in contrast to the E- α_1 PI complex, a clear discrimination of the severity of septicemia was not indicated by the CRP level in both infected groups, either at onset of sepsis or during septicemia. Only the last determination was in agreement with clinical conditions. Obviously, the E- α_1 PI level does reflect more specifically severe infections such as septicemia or septic shock.

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Elastasa granulocítica liberada después de cirugía abdominal

Con el objeto de determinar el papel de la liberación de enzimas de lisosoma liberadas por los leucocitos polimorfonucleares (PMN) durante la septicemia, se midieron los niveles plasmáticos de PMN elastasa utilizando un método introducido recientemente de ensayo inmunosorbente con unión enzimática para la detección del complejo inhibitor de la PMN elastasa- α_1 -proteinasa ($E-\alpha_1PI$). En forma continua se examinaron muestras del plasma de 41 pacientes tomadas antes y después de cirugía abdominal mayor. Los pacientes fueron divididos en tres grupos: un grupo A no infectado, y dos grupos con septicemia: el grupo B de sobrevivientes y el grupo C de no-sobrevivientes. Los niveles de $E-\alpha_1PI$ de los 11 pacientes sin datos de infección se encontraron dentro de límites normales (valores normales: 86.5 ± 25.5 ng/ml determinados en 153 voluntarios sanos) con excepción de un pequeño aumento a 208.8 ± 25.6 ng/ml a las doce horas del postoperatorio. Cuando se confirmó la presencia de septicemia en los pacientes de los grupos B y C, los niveles de $E-\alpha_1PI$ se elevaron un promedio de 6 veces sobre los valores normales en los pacientes B (649.9 ± 116.3 ng/ml) y 10 veces en los pacientes del grupo C (985.1 ± 154.6 ng/ml). En ambos grupos se llegaron a medir valores máximos de 2200 ng/ml. En los pacientes del grupo B los valores de $E-\alpha_1PI$ regresaron a lo normal durante del periodo de recuperación, en tanto que en los pacientes del grupo C, permanecieron elevados hasta el momento de la muerte (560.5 ± 174.7 ng/ml). Se demostró correlación entre la cantidad de elastasa liberada en la circulación y la disminución de la actividad de antitrombina III (AT III), factor de coagulación XIII (F XIII), y macroglobulina- α_2 (α_2M), así como con el aumento de proteína C reactiva del plasma (CRP). Concluimos de este estudio que la liberación de elastasa y otros factores del lisosoma de las células PMN, juegan un papel muy importante en las alteraciones pato-bioquímicas que ocurren durante la septicemia. Además, consideramos que la presencia de niveles elevados de $E-\alpha_1PI$ en el postoperatorio parece ser un indicador de la aparición y persistencia de infección, así como de la severidad del problema.

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