Chronic Prostatitis

Clinical, Microbiological, Cytological and Immunological Aspects of Inflammation

Edited by

H. Brunner, Wuppertal
W. Krause, Marburg
C. F. Rothauge, Gießen
W. Weidner, Gießen

With 52 Figures and 59 Tables
CIP-Kurztitelaufnahme der Deutschen Bibliothek

Deutsche Bibliothek Cataloguing-in-Publication Data


ISBN 3-7945-0978-1

NE: Brunner, Helmut [Hrsg.]

The reproduction of general descriptive names, trade names, trade marks etc. in this publication, even when there is no special identification mark, is not to be taken as a sign that such names, as understood by the Trade Marks and Merchandise Marks Law, may accordingly be freely used by anyone.

All rights reserved, no part of this book may be translated or reproduced in any form without written permission from Schattauer Verlag.

© 1985 by F. K. Schattauer Verlag GmbH, Stuttgart, Germany

Printed in Germany

Composing, printing and binding: Allgäuer Zeitungsverlag GmbH, Kempten

ISBN 3-7945-0978-1
Contents

E. M. Meares Jr.
Chronic Bacterial Prostatitis ........................................... 1

N. J. Blacklock
Surgical Concepts in the Treatment of Chronic Bacterial Prostatitis .. 13

L. Baert, J. Mattelaer, P. de Nollin
Treatment of Chronic Bacterial Prostatitis by Local Injection of Antibiotics into Prostate ........................................... 29

M. C. Shepard
Role of Ureaplasma urealyticum in Male Lower Genital Tract Infections ................................................................. 39

D. Taylor-Robinson, P. E. Munday, N. F. Hanna, B. J. Thomas, P. H. Furr
Microbiological Aspects of Non-Gonococcal Urethro-Prostatitis and its Probable Consequences ........................................... 47

M. Peeters, A. Polak-Vogelzang, F. Debruyne, J. van der Veen
Abacterial Prostatitis: Microbiological Data ................................ 55

H. Brunner, W. Weidner, H. G. Schiefer
Studies on the Role of Ureaplasma urealyticum and Mycoplasma hominis in Prostatitis ...................................................... 63

Prostatitis as Sequela of Non-Gonococcal Urethritis — A Prospective Study ................................................................. 75

Significance of Chlamydia trachomatis in “Abacterial” Prostatitis .... 85

G. M. Colpi, A. Zanollo, M. L. Roveda, A. Tommasini-Degna, G. Beretta
Bacterial Flora in Expressed Prostatic and Vesicular Secretions of Infertile Subjects .......................................................... 93

Th. Mertens, A. Lanvers, H. J. Eggers
Can Herpesvirus hominis be Isolated from the Genitourinary Tract of Men Having no Manifest Symptoms of Herpesvirus Infection? .... 101
L. M. D. Shortliffe, T. A. Stamey
The Immunologic Characterization of Bacterial Prostatitis Caused by Enterobacteriaceae .................................................. 107

R. U. Anderson, S. H. Ma
Immunological Studies in Abacterial Prostatitis .......................... 113

R. U. Anderson
Leukocyte Studies in Abacterial Prostatitis ............................... 121

E. Johanniesson, P. Graber
Cellular Changes in the Prostatic Massage Fluid and Prostatitis ..... 125

W. Ludvik
The Diagnostic Significance of Leukocyte Counts in Prostatic Fluid ... 133

W. Weidner, H. Ebner
Cytological Analysis of Urine After Prostatic Massage (VB3) — A New Technique for a Discriminating Diagnosis of Prostatitis ............. 141

H. Hofmann, H. Brunner, G. Hammer, O. Kurz
Clinical, Spermatological, and Microbiological Findings in Patients with Fertility Disorders and Chronic Prostatitis ..................... 153

H. -H. Riedel
Leucocytospermia — A Sign for Chronic Infections of the Male Genital Tract? ................................................................. 157

W. Weidner, W. Krause, H. Brunner, H. G. Schiefer
Semen Quality in Men with Urogenital Infections by Ureaplasma urealyticum ................................................................. 165

M. Balerina, G. M. Colpi, A. Campana, L. Roveda, A. Tommasini- Degna, A. Zanollo
Electrophoretical Analyses of Human Expressed Prostatic Secretion (EPS) and the Diagnosis of Prostatitis .............................. 173

H. W. Bauer, W. Sturm, J. Schüller, E. Schmiedt
Biochemical Analysis of Prostatic Fluid in Chronic Inflammation: pH, Immunoglobulins and Proteins ........................................ 181

H. Blenk, A. Hofstetter
The Behaviour of Complement C3 and other Serum Proteins in the Ejaculate in Chronic Prostatitis and their Diagnostic Importance .... 189

A. Sziégoeit, W. Krause, H.-C. Becker, W. Weidner
Presence of Secretory- and Serum-IgA in Seminal Plasma ............. 199
Rheumatoid Factor-negative Arthritis and Urogenital Infections in
Men ................................................................. 203

M. Jochum, W.-B. Schill, E. Fink, A. Friesen, A. Hofstetter,
B. Schiessler
Proteinases and Proteinase Inhibitors in Ejaculates of Men with Adnex
Affections ............................................................. 209
Proteinases and Proteinase Inhibitors in Ejaculates of Men with Adnex Affections

M. Jochum, W.-B. Schill, E. Fink, A. Friesen, A. Hofstetter, H. Schiessler

Introduction

Proteinases of the male genital tract are important primarily for the fertilization process. They are taking part also in coagulation and liquefaction of ejaculate as well as in stimulation of sperm migration within the female genital secretions. Moreover, proteinases are involved as mediators of the inflammatory response in the male genital tract aggravating the clinical symptoms (Schill, 1975; Hafez, 1976; Havemann and Janoff, 1978).

These proteinases are either sperm specific enzymes (acrosin), secretory products of the accessory sexual glands (seminin, BAEE-splitting enzyme, urokinase, tissue kallikrein) or they are liberated in the adnexal region during degranulation of leukocytes (elastase).

In the male genital tract proteolytic activity of the proteinases are kept under control by potent antagonists, the high molecular weight plasma proteinase inhibitors α₁-proteinase inhibitor (formally α₁-antitrypsin) and α₁-antichymotrypsin as well as the low molecular weight, acid-stable secretory products leukostatin (HUSI-I) and acrostatin (HUSI-II) (Schiessler et al., 1976; Schill, 1976; Schiessler and Schill, 1977; Schill and Schiessler, 1977).

We were interested, therefore, to see whether in chronic adnex affections alterations in the proteinase/proteinase inhibitor system of the ejaculate are measurable, probably qualified as diagnostic criteria.
Material and methods

Ejaculates were withdrawn from patients undergoing standard diagnostics such as 3-tube test and analysis of the urethral smear, prostatic fluid and urine. Diagnosis of chronic adnexitis was confirmed by the following criteria: significantly high numbers of gram-negative bacteria, enterococci, mycoplasmas, Ureaplasma urealyticum and Chlamydia trachomatis, leukocytes more than 20 per visual field; evidence of complement factor 3c and coeuruloplasmin in seminal plasma. Ejaculates from 40 patients with adnex affections were examined; 14 patients showed vegetative urogenital syndrome (VUG) and anogenital symptomcomplex (AGS), 24 patients suffered from chronic and 2 from acute adnexitis. Within the group of chronic adnexitis patients were further differentiated according to the concentration of the complement factor 3c as follows: patients with C3c values lower (n = 14) or higher (n = 10) than 1.2 mg%, respectively. Activities of seminin, urokinase, and the BAEE-splitting enzyme in seminal plasma were determined using known methods (FRITZ, 1972; SCHILL, 1973). Tissue kallikrein was quantified by a specific radioimmunoassay (FINK and GÜTL, 1978). The immunological concentrations of α1-proteinase inhibitor, α1-antichymotrypsin, acrostatin and leukostatin were measured by radial immunodiffusion ("Mancini-technique"). The concentration of liberated granulocytic elastase bound to α1-proteinase inhibitor (E-α1PI) was estimated by a newly developed enzyme-linked immunoassay (NEUMANN et al., 1983). Statistical evaluation was performed by the independent Student-t-test.

Results and discussion

Significance and physiological function of the BAEE-splitting enzyme from prostatic secretions is rather unknown. So far, only during viscosity disturbances of seminal plasma, a clearly lowered enzyme activity could be proven (SCHILL, personal communication). Seminin, originating also from prostate, is involved in clotting and liquefaction of sperm. Moreover, it may facilitate penetration of spermatozoa into cervix mucus. In contrast to normal ejaculates, both enzymes showed a significant decrease during chronic adnexitis as well in VUG as in AGS. This might be caused probably by massage of prostatic before ejaculation. With acute adnexitis, a clear increase in enzyme-concentrations was measurable in accordance with a commonly observed enhancement of organ function and synthesis during acute inflammatory reactions (Fig. 1).
Proteinases and Proteinase Inhibitors in Ejaculates of Men with Adnex Affections

Urokinase and tissue kallikrein were also demonstrable in prostatic secretion; their physiological relevance, however, is still unknown. Probably, tissue kallikrein takes part in stimulation and maintenance of spermatozoon motility via liberation of pharmacologically highly active kinins from sperm plasma kininogen. In comparison to healthy persons, both enzymes showed a slight, though statistically not significant reduction in patients with chronic adnexitis and VUG; again, in acute adnexitis an expected increase of the enzymes' concentrations was measurable (Fig. 2).

The high molecular weight plasma proteinase inhibitors α₁-proteinase inhibitor and α₁-antichymotrypsin, transudated from serum into seminal plasma, showed no significant alterations during VUG, AGS or chronic adnexitis, respectively. However, throughout acute adnexitis the well-known dramatic increase of the inhibitors' concentrations was demonstrable in seminal plasma. This enhancement may be due not only to a considerably increased synthesis of the inhibitors as acute phase proteins during the acute inflammatory response but also to a lowered blood-seminal plasma-barrier (Fig. 3).

The acid-stable, low molecular weight proteinase inhibitor leukostatin synthesized in the vesicular glands is supposed to be the natural antagonist of leukocyte proteinases. It did not exhibit significant alterations. The same holds true for acrostatin (Fig. 4), the highly potent inhibitor of the penetration enzyme acrosin which is localized in the acrosome of the spermatozoon. The low
Fig. 2. Activity of urokinase and concentration of tissue kallikrein in ejaculates of men with adnex affections.

Fig. 3. Concentrations of α₁-proteinase inhibitor and α₁-antichymotrypsin in ejaculates of men with adnex affections.
molecular weight, acid-stable acrostatin — synthesized in vesicular glands and epididymis — showed a clear reduction of the inhibitor concentration only in man with occlusive azoospermia (SCHILL and SCHIESSLER, 1977).

Summarizing the given data, the proteinases and proteinase inhibitors described above did not turn out to be significant diagnostic parameters in chronic adnexitis, VUG and AGS, respectively. This is, however, different with leukocytic proteinases during these inflammatory processes.

The biological function of leukocytic proteinases is the intracellular protein catabolism of wasted endogenous substances and the degradation of phagocytized invasive organisms. If released extracellularly due to degranulation or disintegration of leukocytes, these proteinases may enhance tissue damage and activation of the inflammatory response. Of the leukocytic proteinases known so far, the neutral proteinase elastase from polymorphonuclear granulocytes deserves special interest because of its high amount within the granules as well as its nearly unlimited cleavage specificity. However, due to a relatively rapid reaction with α₁-proteinase inhibitor, the most important inhibitor of this enzyme, granulocytic elastase liberated extracellularly is found nearly exclusively in an already inactivated form.

With a highly sensitive enzyme-linked immunoassay about 180 ng complexed elastase (E-α₁-PI) per ml were found in ejaculates of 10 healthy persons. Patients with VUG and AGS showed a highly significant elevation up to x =
1100 ng/ml. In chronic adnexitis a similar increase (x = 1200 ng/ml) could be shown in seminal plasma containing C3c concentrations below 1.2 mg%, whereas C3 amounts above 1.2 mg% were combined with a further dramatic increase of complexed elastase up to 4500 ng/ml. Moreover, in acute adnexitis elastase levels far more than 10,000 ng/ml were measured (Fig. 5).

From these preliminary results of 40 ejaculates, which of course will be confirmed with a more extended patient collective, we draw the following conclusions:

1. The levels of complexed granulocytic elastase proved to be a highly sensitive and qualified parameter of inflammatory processes in adnex affections.

2. Most of the biochemical parameters determined so far in ejaculates of patients with adnex affections are only an indirect criterion of an impaired blood-seminal plasma-barrier during the inflammatory process. Levels of complexed granulocytic elastase, however, represent a direct quantification of an inflammatory mediator in the adnexial region.

3. Quantification of complexed elastase might become a reliable biochemical parameter additional to diagnostic criteria used now in adnex affections. This new parameter seems to reflect activity and severity of an inflammatory process more specifically and offers, therefore, new aspects for controlling the course of the disease as well as the therapy.

![Graph](image-url)  
Fig. 5. Concentrations of complement factor 3c and granulocytic elastase in complex with α1-proteinase inhibitor (E-α1PI) in ejaculates of men with adnex affections.
References


(3) HAFez, E. S. E. (ed.): Human Semen and Fertility Regulation in Men. Mosby, St. Louis 1976.


