FIBRINOGEN DEGRADATION PRODUCTS (F.D.P.) IN ASCITIC FLUID OF PATIENTS AFFECTED BY OVARIAN CANCER

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SUMMARY

Ascitic fluid samples from 10 patients with ovarian carcinoma and 10 with cirrhosis of the liver were examined for their content of components of the fibrinolytic system. Large amounts of fibrin/fibrinogen degradation products (F.D.P.) were found in the ascitic fluid in all patients with malignant tumors, but not in the other group.

Determination of F.D.P. may therefore make it possible to differentiate between malignant and non-malignant ascitic fluid. Benz (¹) studied coagulation factors in ascitic fluid and showed that fibrinogen levels and concentrations of the IInd, VIIth and Xth factors are higher in neoplastic ascitis than in cirrhotic ones.

Astedt (²) too noted a significant increase in plasminogen values in ovarian neoplastic ascitis compared to those caused by cirrhosis or benignant tumours.

He also stressed that the "malignant" ascitic fluid shows fibrinolytic activity when it is studied on unwarmed fibrin plates, but does not on warmed fibrin plates.

This proves that fibrinolytic enzymes activate plasminogen $(^{2, 3})$.

The activator of plasminogen released from ovarian carcinoma cells in culture has proved immunologically identical to urine urokinase (³).

Trypsin inhibitors and F.D.P. levels too proved significantly higher in ascitic fluid associated with ovarian carcinoma than in those associated with a benignant ovarian tumor or cirrhosis of the liver (²).

This confirms the fibrinolytic properties of carcinoma cells.

On the assumption that fibrin acts as a substratum for vascular proliferation and that the fibrinolytic activity of the tumour removes the remaining fibrin, cases of ovarian carcinoma treated with fibrinolysis inhibitors, such as transenamic acid and heparin, have been reported in literature $\binom{5, 6}{2}$.

The results support the idea of an antifibrinolytic therapy direct blocking effect on tumour growth.

The aim of this study is finding out whether the ascitic fluid of patients affected by ovarian neoplasia performs fibrinolitic activity through fibrinogen degradation products (F.D.P.) dosage and what clinical implications this determination may have.

MATERIAL AND METHODS

10 patients affected with ovarian carcinoma at an advanced stage (IIIrd, IVth F.I.G.O.) were examined.

The examination concerned the dosage of F.D.P. determined by Thrombo-Wellco-test in ascitic fluid obtained through paracentesis or during surgical intervention.

Dosage was also performed on the ascitic fluid of a control group made up of ten patients suffering from cirrhosis of the liver.

The results were statistically compared.

RESULTS AND CONCLUSIONS

The results of the determinations are showed in tab. 1.

Table 1. — F.D.P. in ascitic fluid (ng/mg).

	No. cases	- X	S.D.	Ρ.
Ovarian carcinomas Cirrhosis	10	1355.77	27.3	< 0.001
of the liver	10	8	1.7	

There is a very significant difference (p < 0.001) between the average F.D.P. values in ovarian carcinoma patients' ascitic fluid and those contained in cirrhopatients' ascitic fluid (calculated tic through Student's "t"), which confirms the thesis of a remarkable local fibrinolysis activation due to neoplasia.

This study adds to the wide debate that has been under way in the literature on the subject of malignant neoplasias growth and spreading in loco and through metastasis, and the intervention of fibrinolytic and coagulative mechanisms.

There are two main opinion trends: according to the first (6, 7, 8, 9) fibrin intravascular deposit is essential to solid tu-

mours growth and spreading, in that it provides the matrix for the formation of new capillaries. This matrix is supported by the considerable tromboplastinic activity of the neoplastic, particularly ovarian, tissue $(^{10})$.

Hence the logical use of anticoagulants as a supplementary therapy in the treatment of ovarian carcinoma.

According to the second opinion trend, fibrin is rather an obstacle to the growth and spreading of malignant neoplasias, as it encapsulates the neoplasia (5, 11).

Hence the potential use of fibrinolysisinhibitors in the treatment of advanced ovarian neoplasias.

Regardless of the trend supported by this study, it is clear that an increased fibrinolytic activity associated with ovarian carcinoma is a useful marker of neoplasias as it can also distinguish neoplastic ascitis from cirrhotic ones.

BIBLIOGRAPHY

- 1) Benz J. J.: Thromb. Diath. Haemorrh., 20, 226, 1968.
- 2) Svamberg L., Astedt B.: Cancer, 35, 1382, 1975.
- 3) Astedt B., Holmberg L.: Nature, 261, 595, 1976.
- 4) Astedt B., Glifberg I., Mattsson W., Tropè C.: J.A.M.A., 238, 154, 1977.
 Soma H., Sashida T., Yoshida M., Miyashita
- H., Nakamura A.: Acta Obst. Gyn. Scand., 55, 285, 1980.
- 6) O'Meara R. A. Q .: Irish J. Med. Sci., 394, 474, 1958.
- 7) O'Meara R. A. Q., Thornes R.: Irish J. Med. *Sci.*, 423, 106, 1961. 8) Laki K.: *J. Med.*, *5*, 32, 1974. 9) Clifton E., Grossi C.: *J. Med.*, *5*, 107, 1974.

- 10) Svamberg L.: Thromb. Research., 6, 307, 1975.
- 11) Peterson H., Kjartansson I., Korsan-Songsten K., Rudenstam C. M., Zettergren L.: Acta Chir. Scand., 139, 219, 1973.