OVARIAN TERATOMA AND DIFFUSE PERITONEAL GLIOMATOSIS

(Analysis of a case)

G. M. ARU (*), M. R. PELIZZO (*),
G. E. GERUNDA (*), S. VALENTE (**),
M. GIORDANO (***), F. MEDURI (*)

(*) Dept. of Surgical Path., University of Padua
(**) Dept. of Gyn. Obst., University of Padua
(***) Dept. of Pathology, University of Padua

SUMMARY

The Authors report a case of an ovarian teratoma with diffuse peritoneal gliomatosis and describe their opinion on the pathogenesis and biological characteristics of this rare association.

The association between ovarian teratomas and diffuse peritoneal gliomatosis has been rarely described in literature. It is all the more unusual to discover such a peritoneal dissemination after only a laparoscopic exam, as instead occurred in our experience.

Although many Authors retain the teratoma as the source of scattered glia, the development of this dissemination, its prognostic significance and its therapeutic implication, are still not clear.

With this report the Authors wish to contribute their experience on ovarian teratoma and diffuse peritoneal gliomatosis.

CLINICAL CASE

The patient is a young student of 16 years of age. There are few significant elements from her anamnesis. Her first menstruation occurred at the age of 15. After that she complains of secondary amenhorrea and progressive abdominal distension and stipsys. On admission a physical examination of the patient reveals an abdominal mass of the size of a watermelon, that parts from the pelvis, is connected to the right adnex and which posteriorly dislocates the normal sized uterus. All blood and urine examinations are normal. Negative the presence of α-1-seric feto protein. A series of abdominal x-rays shows irregular calcification within the mass, several of which had a tooth-like morphology. X-rays of the upper G.I. tract and an I.V.P. show the small bowel dislocated toward the spleen and a compressed right ureter with normal passage of the contrast medium. Echographic examination reveals the presence of a partially solid and a partially liquid mass. Laparoscopic investigation shows an abdominal cavity occupied by a mass prevalently cystic. The peritoneum, entirely hyperemic, presents scattered small whitish and bright nodules with not clear defined borders. We perform a biopsy of such nodules. In the abdominal cavity there is also a small amount of yellowish fluid which is positive to the Rivalta's reaction. On the basis of hystological results we make a preoperative diagnosis of diffuse peritoneal gliomatosis in presence of an ovarian teratoma. The patient therefore undergoes a laparotomy with removal of the mass and right adnexiectomy and with biopsy of the nodules.

The anatomical and hystopathological diagnosis of the specimen is: trifillic cystic teratoma of the ovary, multilocular, containing sebaceus

material mixed with cutaneous structures and hair, areas like gelatine, cartilagine and bone. Microscopical examination of the soft areas shows cerebral and cerebellar tissues with the ectasy of the vessel and ventricular and coroideus plexus rudiments. Moreover in a limited area is present a diffuse proliferation of glial cells like astrocites immersed in a fibrillar matrix. These cells have a round hyperchromic nucleus which is not atypical, and so they are classified as 1st degree astrocitoma originating from heterotopic tissues, according to the classification of Turlbeck and Scully. The peritoneal nodules appear to be composed of well differentiated glial cells without an atipical appearance or mitotic activity and so are diagnosed as 0 degree peritoneal glial implantation (fig. 1).

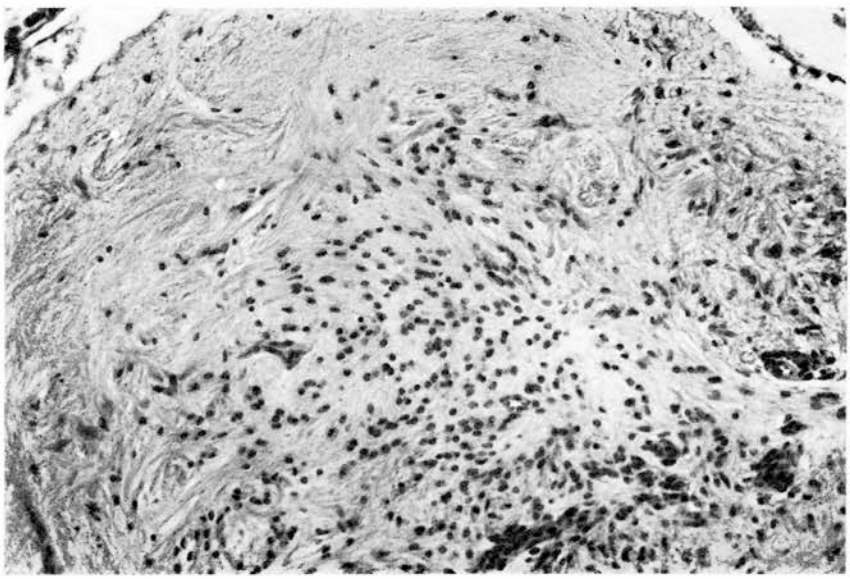

Fig. 1.

The patient dismissed after a regular postoperative period declares to be well now presenting, two years after the exeresis, normal and regular cycles.

Here we report the criteria of Turlbeck and Scully in order to define the degree of maturity of primitive teratomas and or associated peritoneal gliomatosis:

— Degree 0: all tissues mature; no mitotic activity.
— Degree 1: minor foci of abnormally cellular or embrional tissues mixed with mature elements; rare mitoses.
— Degree 2: moderate quantities of embrional tissue mixed with mature elements; moderate mitotic activity.
— Degree 3: large quantities of embryonal tissue present.

DISCUSSION

The ovarian teratoma, not a rare pathology in fertile females, are composed tumors which develop from hypotetic totipotential cells able to differentiate themselves in ecto - meso - endodermal tissues. The chaotic growth of these cells produce a mixture of tissues varying in nature and morphology and with selfgoverning.

In a high percent of cases ovarian teratoma are benign and cystic, but they have various anatomopathological findings. The degeneration of an ovarian teratoma is very rare and is generally rappresented by squamous carcinoma. The association of ovarian teratoma with miliariform peritoneal gliomatosis is instead of rare finding. The Authors [3] who have best

studied this rare pathologic association do not point out, on the basis of their experience and on an accurate revision of the cases reported in literature, the substantial differences between clinical, macroscopical and hystological findings of an isolated ovarian teratoma and of those associated with peritoneal gliomatosis, excepting the age at wich it appears 19 years for the first and 12 for the latter. Physical examination and diagnosis are substantially similar to those observed in abdominal masses with slow growth which dislocates contigous organs. Peritoneal gliomatosis has also a silent growth, not producing reactive peritoneal signs as in our case.

In our report the preoperative demonstration of peritoneal gliomatosis has been raised possible by the laparoscopic exam and by a means of a biopsy which has allowed us to make an accurate differential diagnosis between malignant dissemination and miliariform tubercolar peritonites. Still unknown are the modalities of implantation and growth of peritoneal nodules only composed by mature glia. Less convincing ([3]) is the hypotesis of lymphatic dissemination because, with the exception of a case reported in literature, only one case belonged to the 1st degree ([3]), never have the glial cells been found in the peritoneal lymphatic collectors. More probable seems the hypotesis of a diffusion of glial cells through a laceration of the teratoma's wall perhaps by trauma ([2, 3, 5]) because if the nodules are few, they are located all near the teratoma's wall. In our case there was a diffuse peritoneal implantation and the wall of teratoma appeared intact. It is also very difficult to explain the different degree of maturation of the peritoneal dissemination with respect to the primitive teratoma. It is already ardous to understand how a benign 1st degree teratoma can produce a peritoneal dissemination; so it is necessary to believe that the teratoma has a less degree of maturity when it scatters the glia, or that the cells of a mature teratoma go back into immaturity and return mature after peritoneal implantation. It is more difficult to explain a neuroglial dissemination always with 0 degree with respect to the 1st, 2nd or 3rd degree primitive teratoma. It is here possible to suppose the existence of maturative peritoneal stimulation on the neuroglial cells.

The prognosis is strictly referred to maturity of teratoma and peritoneal implantation, whose growth is never malignant as deduced from a laparotomic look up of several patients. So the prognosis is generally good when the mass, possible source of malignancy, is removed.

Besides there isn't a substantial variation in the prognosis of patients undergoing different treatment: that is with only an exeresis of adnexial mass or with simultaneous controlateral adnelectomy or also with antiblastic chemiotherapy, that doesn't modify the evolution. In our case only right adneiectomy was performed.

We may come to the conclusion, from what has been described above, that many aspects of this rare evolution of ovarian teratoma are unknown and are in need of a valid explanation: in particular, regarding the origin of implantation and the biological significance attributed to the dissemination that several Authors have defined, perhaps not quite inappropiately, « metastasis ».

BIBLIOGRAPHY

1) Garden A. S.: *Journal Obst. Gyn., 79*, 1139, 1972.
2) Noguchi T., Lonser E.: *Am. J. Obst Gyn.*, 381, 1961.
3) Robboy S., Scully R.: *Human Pathology,* vol. I, *4,* 643, 1970.
4) Nogales F., Oliva H.: *Obst. Gyn., 43*, 915, 1974.
5) Favara B., Franciosi R.: *Cancer, 31,* 678, 1973.
6) Willis R. A.: *Pathology of tumors,* 4th ed., Butterworth, 987, London, 1967.

7) Neuhauser H.: *Archiv. fur Gynak., 79,* 696, 1906.
8) Barnard W. G.: *Journ. of Pathol. & Bacter., 34,* 389, 1931.
9) Dempster K. R., Pinniger D. M.: *J. Obst. Gyn. Brith. Emp., 63,* 58.
10) Benirschke K., Easterday C.: *Obst. Gyn., 15,* 512, 1960.
11) Woodruff I. D., Peterson W. F.: *Am. J. Obst. Gyn., 102,* 702, 1968.
12) Luse S. A., Vietti T.: *Cancer, 21,* 38, 1968.
13) Berger N.: *Am. J. Roent., 107,* 647, 1969.
14) Ackerman L. V.: *Surgical Pathol.,* 2th edit., 658, St. Louis 1959, Mosby Company.
15) Anderson W. A.: *Pathology,* 3th edit., St. Louis, 1080, Mosby Company, 1957.
16) Boyd W.: *Pathology for the Surgeon,* Ed. 5th, 393, Saunders Company London, 1955.
17) Blackwell W. J., Dockerty M.B.: *Am. J. Obst. Gyn., 51,* 151, 1946.
18) Ewing J.: *Neoplastic Diseases,* Ed. 4 Saunders Company, Phil. and London, 673, 1957.