



EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

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Case Study
ISSN 2394-3211
EJPMR

HISTOLOGY AS A GOLD STANDARD IN DIAGNOSING GLIOMATOSIS PERITONEI ASSOCIATED WITH IMMATURE TERATOMA OF OVARY

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Article Received on 01/12/2019

Article Revised on 22/12/2019

Article Accepted on 12/01/2020

ABSTRACT

Gliomatosis peritonei (GP) is an infrequent occurrence, exclusively associated with mature or immature teratoma of the ovary. GP is defined as miliary implantation of glial tissues on the surface of the visceral or parietal peritoneum with secondary maturation into glial nodules. A 19 year-old female presented with rapidly enlarging lump in right lower abdomen over the last 2 months. Her past and family history were unremarkable. Local examination revealed a huge mass in abdominopelvic region. Rest of the general and systemic examination were within normal limits. Contrast enhanced computed tomography (CECT) whole abdomen revealed a mass with altered signal intensity lesion in abdomen and pelvis extending into adjacent bowel loops measuring 17.8×17.1×10 cm showing areas of calcification. Serum tumor markers were elevated. Patient underwent laparotomy followed by right salpingo-oophorectomy and excision of nodules on peritoneal surface, omentum and pouch of Douglas. On histopathology examination, a diagnosis of immature teratoma (grade 3) with GP was rendered. GP with an immature teratoma of the ovary is associated with favourable prognosis, although more frequent recurrence is noted. Extensive sampling and multiple biopsies followed by histopathology is the gold standard because it can be mistaken for intra-abdominal carcinomatosis or tuberculosis, and a close follow-up is required to monitor recurrence and rare malignant transformation.

KEYWORDS: Gliomatosis peritonei, Histopathology, glial, implants.

INTRODUCTION

Gliomatosis peritonei (GP) is an infrequent occurrence, exclusively associated with a mature or immature teratoma of the ovary. GP is defined as miliary implantation of glial tissue on the surface of the visceral or parietal peritoneum with secondary glial nodule formation. [1] Surgery and chemotherapy gives longer survival even in recurrent disease.

Grossly, gliomatosis peritonei can resemble ovarian carcinoma associated with adherent capsule or capsular defect. Etiology of GP is largely unknown. There are two theories about the development of GP. One being capsular defects of the primary teratoma or dissemination via angiolymphatic channels.^[2]

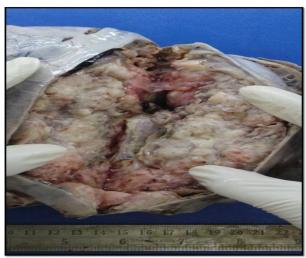
CASE SUMMARY

A 19 yr old female presented to Gynaecology OPD with complaints of rapidly enlarging lump in right lower abdomen associated with pain, non-bilious vomiting and progressive ascites for last 2 months. There was no history of fever, loss of weight, tuberculosis contact, or any other significant past medical history. Her family history was unremarkable.

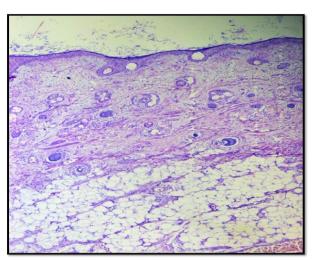
PATHOLOGICAL FINDINGS

Grossly received right ovarian mass measuring $18 \times 18 \times$ 8.5 cms with attached fallopian tube measuring 7 cm in length. Capsule appeared smooth glistening white and intact. On serial slicing, mass showed variegated appearance with solid and cystic areas with bony hard cartilage, yellowish fatty areas and hair. Areas of hemorrhage and necrosis were noted. Histologically, multiple sections examined showed variety of mature and immature tissue derived from all the three germ layers, including stratified squamous epithelium, adnexal structure, glial tissue, ganglion cells, nerve bundles, fibroadipose tissue and muscle bundles. Areas of hemorrhage and necrosis were also seen. Immature neuroepithelium in the form of rosettes and tubules occupying >3 foci of low power field. Bone, cartilage, blood vessels, respiratory epithelium with goblet cells were also seen. Contralateral ovary showed corpus luteum cyst. No teratoma tissue could be identified. Peritoneal biopsy showed features of gliomatosis peritonei. Histopathological diagnosis of immature teratoma grade 3 with gliomatosis peritonei was given.

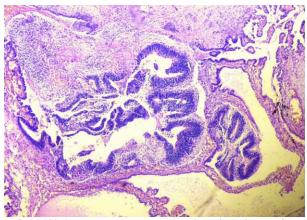
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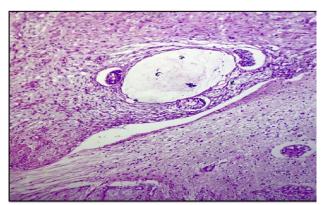
Gross:- Cut section of right ovarian mass showing variegated appearance with solid and cystic areas.



H & E (10x):- Multiple sections examined showed variety of mature and immature tissue derived from all the three germ layers, including stratified squamous epithelium, adnexal structure, glial tissue, ganglion cells, nerve bundles, fibroadipose tissue and muscle bundles.



H & E (40x):- Immature neuroepithelium in the form of rosettes and tubules occupying >3 foci of low power field.



H & E (10x):- Other elements like bone, cartilage, blood vessels, respiratory epithelium with goblet cells also seen.

DISCUSSION

The metastatic implantation of mature glial tissue on the surface of peritoneum, omentum and abdominal lymph nodes is defined as gliomatosis peritonei. Finding of these peritoneal nodules in a clinical setting may be misdiagnosed as ovarian carcinoma or peritoneal tuberculosis intraoperatively.[3] In our case, GP was found in first surgery only; however in some reports, GP was only found in the second surgery. [4] In a literature review, there were three cases of nodal gliomatosis presenting as grades 1 to 3.^[5] If the nodal implantation contained immature elements, these should not be considered as nodal gliomatosis but as metastasis of the immature teratoma. The stage and grade of the primary teratoma and the grade of its metastatic tumor are related to the prognosis of teratoma. [6] Robboy and Scully in a review of case series, found that the prognosis in metastasized ovarian teratomas or glial implants in peritoneum are favorable when composed of fully mature glial tissue as in our case.^[7]

Müller in a review of literature of cases showing adverse outcomes found that the recurrence of disease was associated with lack of extensive histological sampling at the first surgery.^[8]

Thus, all specimens need to be adequately sampled and multiple biopsies should be taken to exclude immature glial tissue or teratoma elements. Once the presence of immature teratoma is confirmed in the metastatic tissue, the treatment scheme and prognosis may change.

CONCLUSION

GP with an immature teratoma of the ovary is associated with favorable prognosis. Histopathology is the gold standard for diagnosis. Because GP is always present with massive peritoneal implantation, optimal resection is difficult. A close clinical follow-up is required to monitor recurrence and rare malignant transformation. Although residual peritoneal disease can be totally quiescent over a long period, long-term follow-up is needed for patients with residual disease.

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Conflicts of Interest/disclosures

The authors declare that they have no financial or other conflicts of interest in relation to its publication.

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