

A Case of Invasive Mola Hydatidosa in the Uterine Tube

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We present a case of invasive hydatid mola as one of the forms of trophoblast proliferation of the chorionic villi that vary in invasiveness and are classified under the common entity trophoblastic disease.

The case presented deserves interest as a rare localization of invasive hydatid mola in the uterine tube in an ectopic pregnancy. For the last 30 years (1994-2004) only 40 cases have been reported in literature.

Key words: hydatid mola, trophoblastic disease, ectopic pregnancy.

Introduction

Gestational trophoblastic disease is a collective notion which unites morphologically similar, but clinically diverse proliferative and other disruptions of the structure of the trophoblast. A portion of these pathological conditions belong to the tumour-alikes, while others are real tumours. Nosologically, the gestational trophoblastic disease includes *Endometritis Syncytiale*, *Mola Hydatidosa*, *Mola hydatidosa invasiva*, *Chorionepithelioma*, and trophoblastic pseudotumour at the site of placental implantation. *Mola Hydatidosa* (MH) is a typical facultative precancerosis of the chorionepithelioma. The tubal localisation of MH is encountered extremely rarely and usually implies a diagnostic error. In this article we will describe one of our clinical cases to focus physicians' attention to this condition and to recall some clinical and non-clinical methods that assist in diagnosing it expediently and correctly.

Materials and Methods

A twenty-five-year-old single female admitted to the Clinic of Obstetrics and Gynecology at the University Hospital of Plovdiv with symptoms of acute abdomen. Status on admission: slightly swollen abdomen, peritoneal irritation in the right inguinal region, pale skin and mucosa. Gynecological status: uterus hardly palpable due to muscle defence. Uterine tubes and parametrium: without infiltrative changes, with palpatory tenderness on the

right. Cavum Douglasi: not swollen, painful. Ro: no free gas under the diaphragm dome; isolated hydroaeric shadows in the right abdominal half. Ultrasonography: uterus: no abnormality detected; dimensions of the right ovary: 23 mm/ 10 mm with doubts of amniotic sac in the area of the right uterine tube; left ovary and uterine tube without abnormalities. Presence of free liquid in cavum Douglasi. Puncture of the same: aspiration of blood and blood coagulates. The diagnosis - graviditas extrauterina, ruptura tuba dextra, haemoperitoneum — led to an emergency laparotomy. In situ: 800 ml free blood and blood coagulates in the abdominal cavity. Uterus: no abnormalities detected, left ovary and tube: no abnormalities detected, right uterine tube: ruptured in the isthmic section and necrotically altered. Classical salpingectomy was performed.

Results

Histopathological findings: the tissue material from the uterine tube was processed using a routine methodology and stained with haematoxylin and eosin (HE). The biopsy preparations (323-325/8.01.2001) provided histological verification: uterine tube with evidence of tubar pregnancy and tubar abortion — discrete decidual alterations in the mucose, presence of chorial villi in the lumen and wall. Haemorrhage and rupture of the uterine tube wall.

Due to the retention of high levels of human choriongonadotropin (HCG) in the serum, in the postoperative period after the tubectomy, the physician in charge requested a second review of the histological preparations. The meticulous examination of the sections established the presence of villi with significantly increased volume; swelling; a decreased number of cells and avascularisation of the villi stroma; growth of the trophoblastic epithelium with manifestations of significant trophoblastic dysplasia (Figs. 1, 2, 3). At places, trophoblastic nuclei cluster and exceed 5-8 rows. They also exhibit significant changes in size, form, and colouration (polymorphism and hyperchromasia). The cytotrophoblast and the syncytiumtrophoblast exhibit significant cellular polymorphism and vascularisation (Figs. 2, 3). The tube wall is heavily stratified. A large number of trophoblastic cells, some with atypical, abnormal shape, and whole chorial villi migrate through the mucose and the muscle layer to the serous lining (Fig. 4). A group of villi are situated paravasally in the nearby paratubar tissue (Fig. 5).

On the basis of the stromal and trophoblastic manifestaions described above, we decided on tubar pregnancy with characteristics of MH. Regarding the topology of the process, the idiosyncrasies of the morphological course of the tubar pregnancy, and lack of evidence of invasion of molar structures in other genital organs, in the pelvis or extragenitally, we abstained from diagnosing invasive MH.

The patient was discharged in good condition, with recommendations for clinical monitoring and periodical follow-up of the HCG titer.

Discussion

The incidence of MH is 1 per 1400 pregnancies in Europe [1], so it is a relatively rare form of gestational trophoblastic disease. Ectopic tubar MH has been described in only 40 cases in world literature [3].

The most prominent risk factor discussed in the literature is the presence of a husband older than 40. The age of the mother is also important: women over 45 almost never develop MH. If the patient has had a previous MH, the risk increases by more than 25% [2]. Other risk factors are: previous abortions (regardless of whether they are spontaneous or induced), surgical intervention, oral contraceptives that have been taken for 2.5 to 10

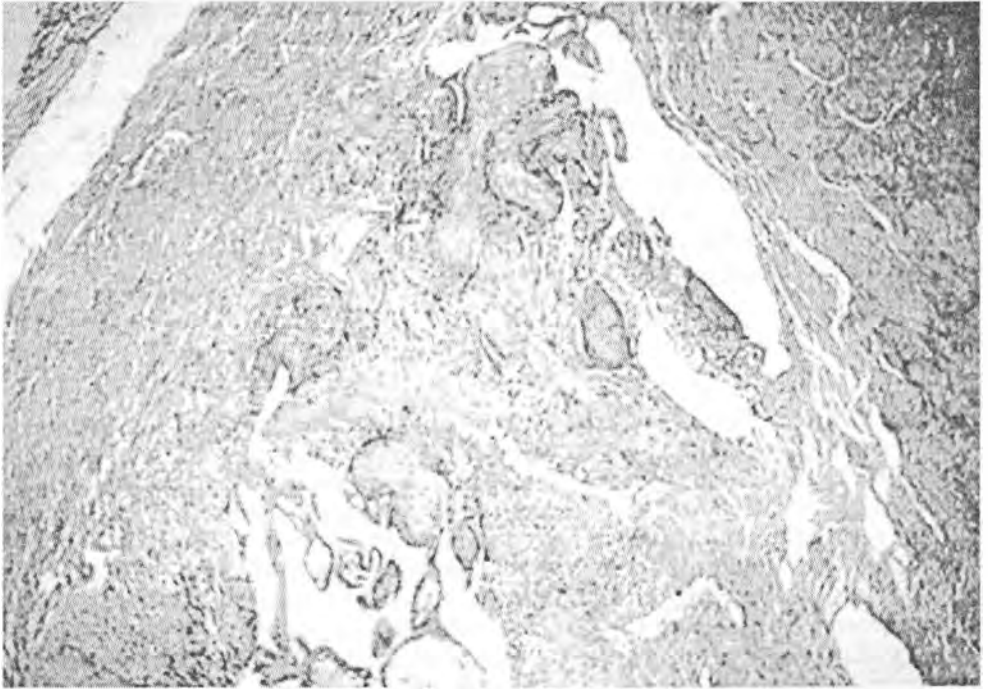


Fig. 1. Chorial villi with swelling and avascularisation of the stroma; trophoblast proliferation with considerable trophoblast dysplasia. HE staining ($\times 10$)

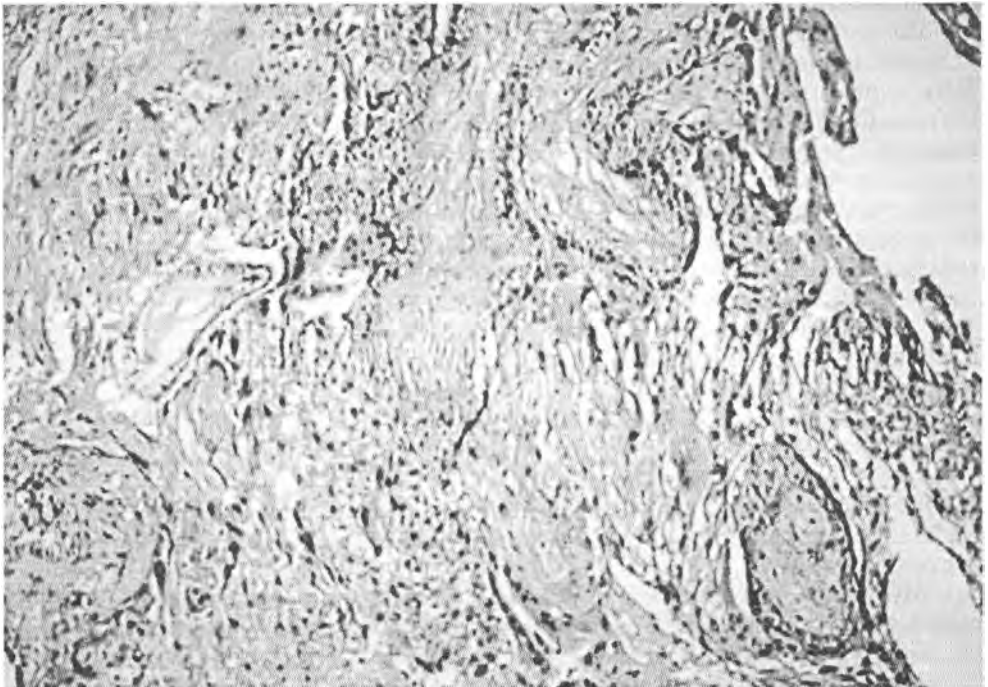


Fig. 2. Chorial villi with swelling, cell deprivation and avascularisation of the stroma; trophoblast proliferation. HE staining ($\times 40$)

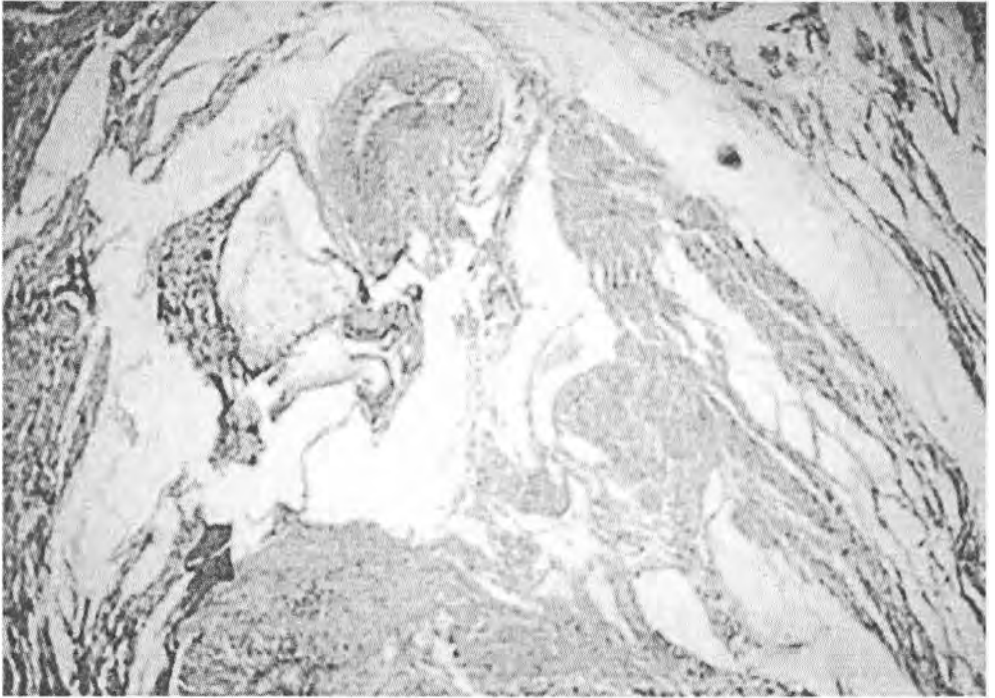


Fig. 3. Segment of a chorial villus with expressed swelling of the stroma, expressed trophoblast proliferation with dysplasia. HE staining ($\times 40$)

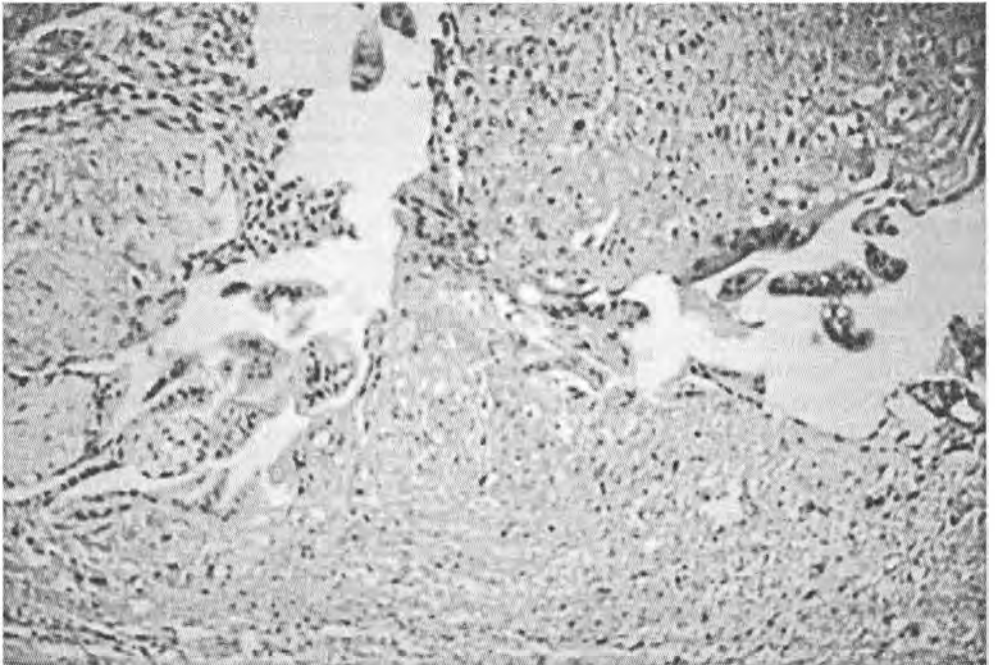


Fig. 4. A cluster of chorial villi resembling molar villi with perivascular localization in the adjacent paratubal tissue. HE staining ($\times 40$)

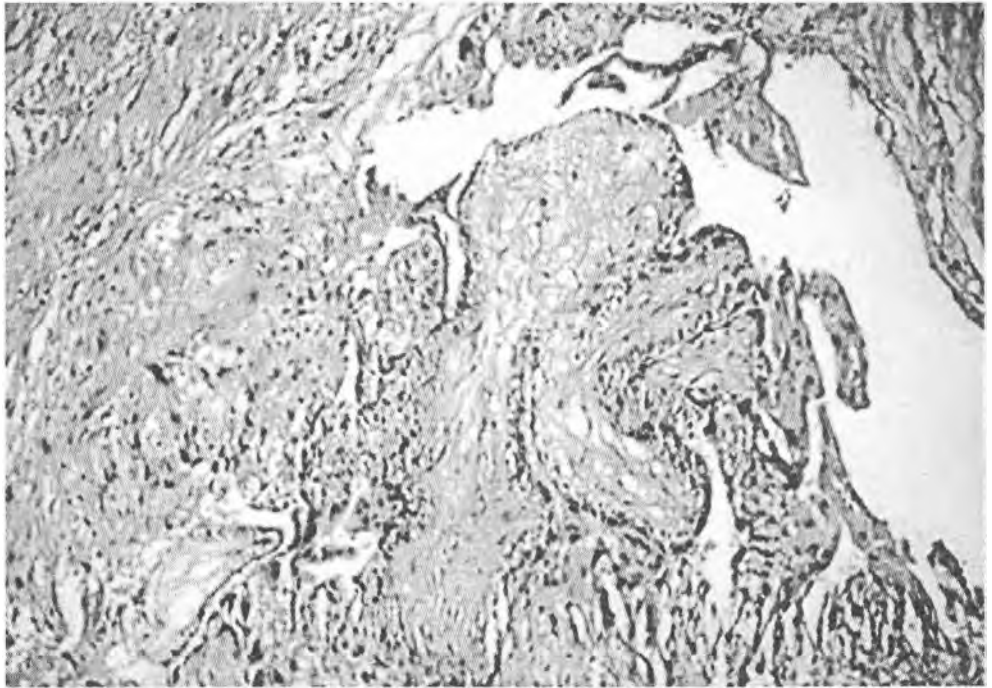


Fig. 5. Migration of a large number of atypical, abnormal cytotrophoblastic and syncytial cells in the uterine tube wall. HE staining ($\times 40$)

years. Potential risk factors are: smoking, alcohol, dieting, socio-economic factors, infectious agents like the papilloma virus, and exposure to pesticides and herbicides [2].

Tubar MH can be expressed with the symptoms of acute abdomen, as in our case. To avoid misdiagnosis, the physician should consider anamnestic data of previous conditions, gynecological status, ultrasonographic findings, etc. An important characteristic of MH is the possibility of producing large quantities of HCG, although there are cases of ectopic tubar MH with low HCG levels [3]. As a noninvasive and repeatable method, a colour doppler examination can lead to a precise diagnosis in such unclear cases.

One DNA analysis shows that MH can become invasive [1].

MH and invasive MH should not be confused with choriocarcinoma despite the significant proliferation of the cytotrophoblast and the syncytiotrophoblast. There are no villi in the case of choriocarcinoma [2]. Invasive MH also exhibits metastatic foci in the vagina, lung, ligamentum latum uteri several weeks after its removal [4].

We found in the literature that mola hydatisosa forms as a result of fertilisation of an abnormal (anuclear) egg cell with a haploid 23X sperm cell, which then doubles (a complete MH has a 46XY or a 46XX karyotype).

Conclusion

The contribution of our research work is to demonstrate that MH developing in an ectopic tubar pregnancy is difficult to diagnose **clinically**. In such a case, the correct histopathological diagnosis is of primary importance. The case we described corroborates the challenges related to the diagnosis and the course of the disorder.

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