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A REVIEW ON FALLOPIAN TUBE CARCINOMA

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Abstract:

Primary fallopian tube cancer is an uncommon condition that is challenging to treat. It is frequently included with primary ovarian and peritoneal carcinomas under the general heading of epithelial ovarian cancer. According to more recent data, ovarian epithelial malignancies may have their origins in a precursor in the fallopian tube. Platinum-based chemotherapy and surgical cytoreduction constitute the cornerstones of treatment. The ideal time for surgery and the best method for administering chemotherapy are hotly contested topics. Conventional intravenous regimens that are administered once every three weeks are contrasted with intraperitoneal and dose-dense regimens. Despite the ongoing discussions surrounding this topic, new targeted treatments have surfaced, such as bevacizumab and inhibitors of poly (adenosine diphosphate [ADP]-ribose) polymerase (PARP). Patients with BRCA1/2 gene mutations benefit most from PARP medicines, and using them has been demonstrated to increase patient survival. This article reviews the etiology, pathophysiology, symptoms, diagnosis and treatment of the disease.

Keywords: Fallopian tube carcinoma, high grade serous cancers, serous tubal intraepithelial carcinomas, Epithelial ovarian carcinoma (EOC).

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INTRODUCTION:

One in every ten female genital tract cancers , primary fallopian tube carcinoma (PFTC), is an extremely rare kind of cancer (1,2).Due to the uncommon nature of this illness, fewer than 2,500 instances have been documented in various studies conducted globally to date, the majority with relatively little study information of female genital cancers, primary fallopian tube carcinoma (PFTC) is a rare tumor that accounts for 0.14–1.8% of cases (3-8) Patients with PFTC frequently experience abdominal pain because tubal distension might induce this nonspecific symptom [9]. Patients with PFTC can be detected earlier since their symptom history is shorter than that of EOC [10]. Comparatively, PFTC exhibits a tendency toward tiny distant metastases. The four main histologic subtypes of ovarian epithelial cells are called clear cell carcinoma, mucinous, endometrioid, and serous cell carcinoma. Since ovarian epithelial cells are dichotomous, classification system has been proposed for cancers, which are a heterogeneous group of malignancies both molecularly and histologically. Type I tumors are restricted to the ovary, have a high level of chromosomal instability, and are genetically stable. They include low-grade serous, mucinous, endometrioid, and clear cell carcinomas; type II tumors present at advanced stages, are highly variable in terms of chromosomal stability, and are composed of high-grade serous, undifferentiated, and carcinosarcomas. More than 95% of cases of high-grade serous carcinomas (HGSC), TP53 mutations are present. While the inactivation of the BRCA1/2 genes by processes like hypermethylation can be observed in as many as 40% to 50% of cases of nonfamilial HGSC, no additional mutation is consistently observed in this population. The actual incidence of PFTC was most likely underreported in prior years due to the high number of advanced cases were incorrectly thought to be ovarian cancer. Patients with PFTC continue to have a low overall survival rate, which ranges from 22 to 57% (11). Most of the etiological factors remain unknown. This page highlights several novel elements of this uncommon cancer based on the most recent research.

ETIOLOGY:

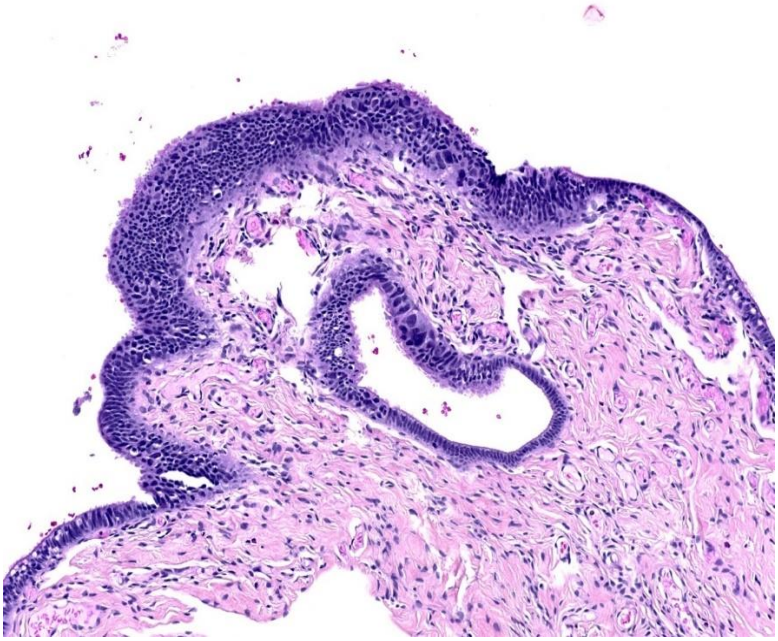
Although the exact etiology of PFTC is yet uncertain, epithelial ovarian cancer has been linked to comparable etiological variables. Previous reports deemed chronic tubal infection as PFTC's driving force (12) identified a link between tuberculous salpingitis and PFTC, and a high incidence of histological and/or gross signs of ancient pelvic inflammatory illness was documented (13). However, no other specific infectious agent has been linked to PFTC. Age, race, weight, education level, infertility, endometriosis, lactose intolerance, smoking, previous hysterectomy, and pelvic inflammatory disease have all been found to have no statistically significant link with PFTC [14, 15, 16]. Families at high risk for breast and ovarian cancer that have germ-line BRCA-1 and BRCA-2 mutations have been linked to PFTC [17-20]. A shared molecular etiology was postulated because to certain studies' findings that the frequency and structure of the chromosomal alterations (BRCA-1 or BRCA-2 mutations) seen in PFTC were comparable to those identified in serous ovarian, breast, and uterine carcinomas [21–25]. In bearers of the BRCA1 mutation, occult PFTC has occasionally been found during prophylactic salpingo-oophorectomy [26]. Therefore, when performing preventative surgery in such high-risk women, the risk for malignant malignancy should be considered [27].

PATHOGENESIS:

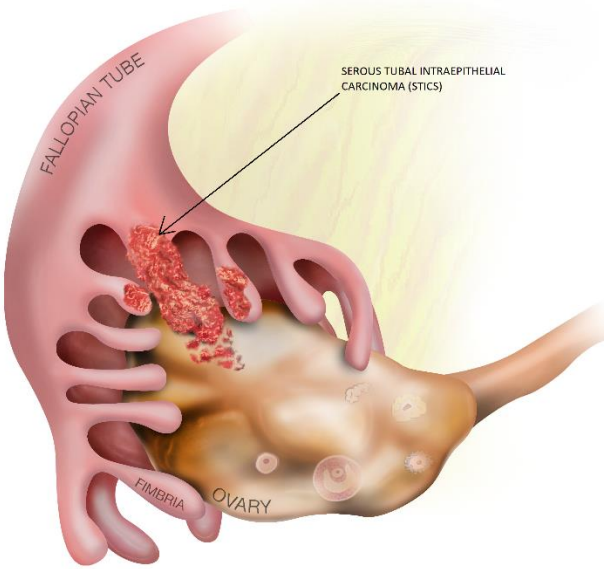
It was formerly believed that the neoplastic transformation of cells in the ovary's cortical inclusion cysts was the cause of epithelial ovarian cancer (EOC).28-29 According to the incessant ovulation idea, ovarian epithelial lining genetic abnormalities gradually accumulate due to repetitive ovulation-related damage and repair, ultimately resulting in endometrial cancer (EOC).30-32. But it has never been proven beyond a reasonable doubt that there is an ovarian precursor lesion that develops into high-grade serous carcinoma (HGSC).
Another theory holds that the epithelial layer of the nearby fimbrial end of the fallopian tube is where HGSC starts rather than the ovary's surface.33-35 More recent information indicates that the pathophysiology and molecular makeup of ovarian HGSC, primary peritoneal carcinoma (PPC), and FTC are comparable. A shared etiology of ovarian, fallopian, and primary peritoneal malignancies is also supported by the epidemiological data. These three malignancies have comparable incidence rates across racial, ethnic, and geographic groups additionally, the greater prevalence of all three malignancies in non-Hispanic White women raises the possibility of a shared etiology.
Most epithelial malignancies in the body typically develop from benign epithelium to invasive cancer because of a sequence of events.

Underreporting of FTCs has been exacerbated by the traditional pathologic designation of pelvic serious malignancies primarily as ovarian cancer. Diagnosing FTC only occurs when there is neither an endometrial nor an ovarian mass.36 It is necessary to have both a

dominant tubal mass and a precursor lesion in the fallopian tube to classify a tumor as having its genesis there; however, an ovarian tumor can be diagnosed without a precursor lesion.37 Likewise, PPC is only identified in cases where no endometrial, fallopian tube, or ovarian tumor is discovered. When the origin is unknown, serous tumors in the pelvis are traditionally classified as ovarian cancer.



Picture of serous tubal carcinoma:



picture of fallopian tube carcinoma

CLINICAL MANIFESTATION AND SYMPTOMS:

Postmenopausal individuals make up two thirds of PFTC cases.

PFTC patients had significant rates of nulliparity and infertility—13–45% and 71%, respectively (38). Patients with PFTC range in age from 50 to 69 years old.

While PFTC has been observed in young girls ages 17–20 (39), the average age of the participants in these investigations was 55 years (40). Table III provides a summary of the clinical characteristics and symptoms of PFTC (40). The most distinctive symptoms of PFTC are aberrant cytological results and serosanguinous hemorrhage accompanied by a pelvic tumor.In the past six months, we have seen one typical finding: a huge, enlarged uterus that appeared to be filled with serosanguinous fluid, along with a sausage-shaped fallopian tube packed with fluid. The conclusive histopathological diagnosis was PFTC. The fourth and sixth decades of life are when PFTC most commonly manifests itself [41,42]. The median age of occurrence for PFTC is 55 years old (range, 17–88 years). Nevertheless, PFTC has been among young girls between the ages of 17 and 19 [43]. Compared to patients with EOC, individuals with PFTC seem to have a shorter symptom history [44]. Table 1 displays the clinical symptoms and indicators of PFTC [45]. There is no pattern to these symptoms. Fifteen percent of cases have been documented to exhibit Latzko's triad of symptoms, which include intermittent profuse serosanguinous vaginal discharge, colicky pain alleviated by discharge, and abdominal or pelvic mass [46].

IMAGING AND DIAGNOSIS:

For suspected gynecologic cancers, imaging tests such as computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound are frequently performed stomach. Imaging methods, of course, cannot securely rule in or out the likelihood of malignancy, and their results cannot alter the way PFTC is managed. When diagnosing individuals who may have tubal disease, transvaginal and transabdominal ultrasonography is a crucial imaging modality.

Timor-Tritsch and Rottem, provided evidence about the advantages of transvaginal ultrasound over transabdominal ultrasound for fallopian tube imaging. Fallopian tubes have a general echo graphic look that can be mistaken for ovarian tumors, tubo-ovarian abscesses, and ectopic pregnancies, among other pelvic illnesses.

On an MRI or CT scan, the lesion may show up as a tiny, solid, lobulated mass. The lesion's attenuation on a CT scan is comparable to that of other soft tissue. Not as much bulk and enhancement as the myometrium. The tumor is typically hypointense on T1-weighted MR images and homogeneously hyperintense on T2-weighted MR imaging. Most of the time, imaging can identify solid and cystic components with papillary projections; these can be markedly enhanced on MRI by the addition of gadolinium. Peritumoral ascites, intrauterine fluid accumulation, and hydrosalpinx are findings that are associated with this condition. When it comes to identifying tumor infiltration of the bladder, vagina, pelvic sidewalls, pelvic fat, and rectum, MRI appears to be superior to CT scan or ultrasound.

TREATMENT:

There aren't any sizable, prospective, randomized clinical trials that just assess treatment for FTC or PPC because of the conditions' alleged rarity. According to Cancer Network standards, EOC, FTC, and PPC should all be treated similarly and with the same regimens. Although they are regarded as separate clinical entities, patients with FTC and PPC have actually participated in more recent and current ovarian cancer clinical studies than in the earlier ones. Given the present evidence linking ovarian cancer, FTC, and PPC to a single etiology, treating these cancers similarly is probably the best course of action for managing all of these tumors.

FIRST LINE TREATMENT:

A platinum agent plus a taxane is the first-line treatment for ovarian cancer, FTC, and PPC.47,48 The Gynecologic Cancer Research, the combination of carboplatin and paclitaxel was contrasted with the combination of cisplatin and paclitaxel. Trial 158 of the Oncology Group (COG). When comparing carboplatin plus paclitaxel to cisplatin and paclitaxel, there was no statistically significant difference in either progression-free survival (PFS) or overall survival (OS); yet, the carboplatin arm had fewer nonhematologic side effects and was better tolerated.(49)The conventional treatment of both paclitaxel and carboplatin every three weeks was compared with the combination of dose-dense weekly paclitaxel and carboplatin every three weeks, in a study conducted by the Gynecologic Oncology Group in Japan. In the dose-dense arm, the median PFS was longer (28.0 months compared [vs] 17.2 months)

(hazard ratio [HR] 0.71; P = 0.0015). Additionally, the dose dense regimen group's OS at three years was greater (72.1%) than the conventional therapy group's (65.1%) (HR 0.75; P = 0.03). While other effects were similar in both arms, there were higher hematologic toxicities and treatment discontinuations with the dose-dense paclitaxel. (50) With more than six years of follow-up, the trial's most recent update revealed a median overall survival of more than 100 months in the dose-dense. The co-administration of carboplatin with docetaxel (51) is a viable initial treatment strategy. Because to its convenience, lengthy history, good side-effect profile, and ease of administration. The combination of paclitaxel and carboplatin has become the global standard of therapy for patients receiving first-line treatment for advanced ovarian cancer, FTC, and PPC.7,4

SECOND LINE TREATMENT:

The amount of time that has passed since the last regimen influences the second-line treatment. One significant indicator of the response to second-line therapy is the platinum-free interval.60 Individuals cancer was under control after the last treatment for more than six months are regarded as platinum sensitive.

Depending on their performance level, history of toxicities, and comorbidities, these individuals receive a platinum doublet as a new course of treatment. It has been demonstrated that carboplatin is a good combination to use with paclitaxel (ICON4/Arbeit gemeinschaft Gynecologist Oncology-Ovarian [AGO-OVAR]-2.2), gemcitabine (Intergroup trial), or PEGylated liposomal doxorubicin (PLD) (Calyx in Platinum Sensitive Ovarian [CALYPSO]). The combination of carboplatin and PLD was compared to the combination of carboplatin and paclitaxel in the CALYPSO study. Platinum-resistant patients are those who experience a recurrence within six months of starting platinum treatment. Patients are deemed platinum refractory if their tumors worsen while they are receiving platinum treatment, and the prognosis is not good. For these individuals, platinum drugs are not advised; instead, future care is determined by underlying renal, hepatic, and other comorbidities.

Instead of receiving combination therapy, patients typically receive sequential therapy with single medicines. In resistant ovarian cancer, a Phase III trial using weekly paclitaxel was found to be as effective as a combination therapy using paclitaxel plus carboplatin or topotecan.66 PLD was found to extend the platinum-free interval in a retrospective analysis, allowing patients to become resensitized to a platinum agent.

HARMONALTHERAPY:

The use of hormonal drugs in PFTC has increased. The reasoning behind this is that during the menstrual cycle, hormonal fluctuations cause changes in tubal epithelia. Both histologically and embryologically, the tubal and endometrial epithelium originate from the same source. Since pregestational agents are known to have cyclic reaction of the normal tube to menstrual cycle hormonal fluctuations. It is impossible to make definitive conclusions about the efficacy of pregestational medications because there are no randomized trials and almost all patients receiving them are also receiving combination chemotherapy. Although they have been discovered in a few cases, steroid receptors' clinical significance is unknown.

CONCLUSION:

Less than 1% of all female genital tract tumors are PFTCs, an uncommon malignancy. It is like EOC both clinically and histologically. Rarely is the diagnosis of PFTC considered prior to surgery; instead, it is typically discovered during the procedure or by a pathologist. The age distribution of both carcinomas is comparable, and nulliparous women are more likely to get them.

which frequently have a serous papillary histology. Complete abdominal hysterectomy, bilateral salpingooophorectomy, omentectomy, and dissection of pelvic and para-aortic lymph nodes should all be part of the surgical procedure. Patients with advanced illness should be considered candidates for aggressive debulking surgery. Comparable to EOC in surgical staging is PFTC. There are two distinctions between the two illnesses, though: regular lymphadenectomy has a well-established role, and PFTC is more frequently detected at an earlier stage. and is required in the PFTC. The two most significant predictive markers for outcome are stage and residual tumor. Postoperative treatment may not be provided to patients with stage I low-risk illness who are submitted to appropriate surgical staging. On the other hand, patients with stage I high-risk illness or stage IIA disease, as well as those with stage I low-risk disease who have not had full surgical staging, ought to get 3-6 rounds of adjuvant carboplatin + paclitaxel. As with EOC, patients with advanced illness should get treatment with paclitaxel with carboplatin. For chronic or recurrent illness, second-line therapy should be determined by the platinum-free interval.

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