# 114 NEOPLASMS OF THE FALLOPIAN TUBE

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Malignant neoplasms of the fallopian tube are the rarest of the gynecologic cancers. While cancers metastatic to the tube occur frequently from ovarian, endometrial, or other sources, and while there are reports of transitional cell carcinoma, adenosquamous carcinomas, and sarcomas, almost all of the primary cancers of the fallopian tube are papillary adenocarcinomas.

The first gross description of fallopian tube cancer was attributed to Renaud in 1847. Rokitansky recorded the first microscopic description in 1861, and Orthman is generally credited with the first case report in 1888. Since that time, fewer than 1,800 patients with fallopian tube carcinomas have been reported, and the vast majority of these reports included fewer than 40 patients analyzed retrospectively over a period of more than 10 years in individual institutions. Only recently have collaborative groups or multi-institutional investigators begun to report their data. For example, Rosen and co-workers[1] reported on 143 women treated from 1980 to 1995 by the Austrian Cooperative Study Group for Fallopian Tube Carcinoma. While there have been reports from single institutions with experience in treating 40 or more patients[2–6] from which conclusions concerning pathophysiology, clinical presentation and course, and treatment patterns can be drawn, there have also been several attempts at definitive literature reviews,[7,8] the most recent one reported by Nordin.[9]

## INCIDENCE AND EPIDEMIOLOGY

Primary fallopian tube carcinoma comprises approximately 0.31 to 1.11% of cancers of the female genital tract.[7,10] The primary epidemiologic study of fallopian tube carcinoma was conducted in 1989 by Rosenblatt and colleagues[10] who reviewed the records of nine population-based cancer registries from the National Cancer Institute's Surveillance, Epidemiology, and End Results program (SEER). The annual incidence of epithelial fallopian tube carcinoma was 3.6 per million women per year, and this figure remained constant throughout the decade. Peak incidence occurred between the ages of 60 and 64 years; after age 65 years, the incidence plateaued to an age of 84 years. The incidence is significantly higher in Caucasian women (including Hispanics) than in African Americans. Etiologic factors for the development of primary fallopian tube carcinoma have not been clearly defined. Chronic tubal inflammation commonly coexists in fallopian tubes that contain carcinoma.[8,11] Whether there is an etiologic relationship remains unclear.

## CLINICAL PRESENTATION

The most frequently occurring symptoms are vaginal bleeding, unexplained vaginal discharge, and pelvic pain. The discharge may often be serosanguineous, is usually unexplained by microbiologic studies, and is unresponsive to antimicrobial therapy. The uterine bleeding is obviously pathologic, since the majority of patients are postmenopausal, and it is almost invariably unexplained by uterine curettage. Thus, primary fallopian tube carcinoma must be considered in the differential diagnosis when postmenopausal bleeding persists after a negative curettage.

The presence of pain is highly significant since cancers of the ovary, endometrium, and cervix do not cause pain until their diagnosis is all too obvious. The syndrome of "hydrops tubae profluens" described by Latzko in which a patient presents with pelvic mass, profuse watery or honey-colored vaginal discharge, and pelvic pain that is greatly relieved by the sudden disappearance of the mass, is rarely encountered but is almost pathognomonic. The pain is attributed to distension of the fallopian tube by trapped fluid, and when the fluid is emptied by traversing the tubal-uterine-vaginal channel, the patient is immediately relieved. Pelvic mass is the most common physical finding, occurring in approximately 65% of patients.[9] Ascites accompanies a mass in only 15%, and a variety of less frequent physical findings are attributable to widely metastatic disease at presentation.

## PREOPERATIVE DIAGNOSIS

Because of the infrequency of this disease, with the resultant low level of suspicion by the medical community, preoperative diagnosis is made in less than 3% of the patients described. The presence of a pelvic mass is rarely attributed to tubal carcinoma by the examining physician, and radiography of the urinary and gastrointestinal tracts is useless in making the diagnosis.

The effectiveness of cytologic diagnosis from cervical and/or vaginal pool samples is widely variable and has been reported as positive in 40 to 60% of women with tubal carcinoma.[7,12] Of greater importance is the presence of adenocarcinoma in samples from cervical or vaginal pools in the face of negative fractional dilatation and curettage of the uterus and absent palpable pathology. These patients may have early tubal carcinomas.

During the last decade, there have been increasing numbers of reports of successful diagnosis of fallopian tube carcinoma by ultrasonography.[13] The usual ultrasound finding included a sausage-shaped mass with internal projections into a fluid-filled lumen, giving a characteristic "cogwheel" appearance. The successful preoperative diagnosis of primary fallopian tube carcinoma using transvaginal color and pulsed Doppler ultrasound has recently been reported by Kurjak and co-workers.[14]

In 1984, Niloff and colleagues demonstrated the value of elevated CA-125 levels in monitoring four patients with recurrent fallopian tube carcinoma.[15] More recently, Rosen and co-workers[16] determined pre- and postoperative CA-125 values in 13 patients with fallopian tube carcinoma. The median preoperative value was 1,220 IU/mL, significantly higher in comparison to postoperative levels (median 194 IU/mL). Correlation analysis with stage and grading failed to achieve statistical significance; however, a trend for a positive correlation with stage and preoperative value could be observed.

While it is well established that serum levels of CA-125 can be significantly elevated in patients with pelvic inflammatory disease, endometriosis, and early pregnancy, and while neither CA-125 nor CA-19-9 is highly specific, the presence of an adnexal mass in a postmenopausal woman is an indication for serum marker studies preoperatively, at least to establish reference values for measuring therapeutic response.

## CLINICOPATHOLOGIC CLASSIFICATION AND STAGING

Although more than 90% of the fallopian tube cancers are papillary adenocarcinomas, the synchronous presentation of the same histology in multiple pelvic sites recommends the establishment of diagnostic criteria that will identify primary fallopian tube cancer. The criteria established by Hu and colleagues[17] and later modified slightly by Sedlis[7] have been widely accepted (Table 114.1). Hu and colleagues accepted the histologic classification originally proposed by Sanger and Barth[17] in which three grades of tumor were observed (papillary, papillary-alveolar, or alveolar-medullary). This classification is no longer used by many pathologists, and histologic grade is usually simply designated as well-, moderately, or poorly differentiated. The degree of differentiation of fallopian tube carcinomas has generally not been related to prognosis.[3,5,18,19]

Until 1991, there was no universally accepted staging system for patients with tubal carcinoma. In 1967, Erez and colleagues[20] proposed a clinical staging system based on prognostic observations. This was modified in 1970 by Dodson and colleagues[21] to conform with the International Federation of Gynecology and Obstetrics (FIGO) surgical staging system for ovarian cancer. The following year, Schiller and Silverberg[8] modified the Dodson system to emphasize the importance of disease confined to the tubal lumen and the prognostic importance of invasion through the tubal wall to spread beyond the serosa. These staging systems were prevalent in most of the reports during the last 20 years, and in 1991, FIGO officially promulgated a staging system for tubal carcinoma[22] (Table 114.2).

**Table 114.1. Criteria for Carcinoma of Fallopian Tube***

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| 1. Grossly: the main tumor is in the tube and arises from the endosalpinx |
| 2. Histologically: the pattern reproduces the epithelium of tubal mucosa (papillary pattern) |
| 3. Transition from benign to malignant tubal epithelium should be demonstrated |
| 4. The ovaries and endometrium are normal or have a much smaller tumor volume than that of the tube |

*From Sedlis A,[7] Hu et al. [17]

The new staging system takes into account the hollow viscus observations, the importance of ascites, and the impact of lymphatic spread. However, recently, Alvarado-Cabrero and co-workers[23] proposed expanding the staging system to permit staging of noninvasive tubal carcinomas and fimbrial carcinomas, and that substaging based on depth of invasion merits exploration in future studies. Their recommendations are based on the observation that there was a significant difference in the length of recurrence-free survival of patients with no invasion beyond the epithelium or invasion of the lamina propria versus invasion into the muscularis.

## PATTERNS OF SPREAD

Studies from the literature indicate that the pattern of spread of fallopian tube carcinoma is similar to ovarian carcinoma with both intraperitoneal and lymphatic spread commonly encountered.[3,7] However, because older staging systems did not mandate lymphadenectomy, there are few data on the incidence of lymph node metastases at the time of presentation. Tamimi and Figge[11] reviewed 15 patients treated over a 12-year period in their institution. Lymph node sampling was not routine at the time of initial surgery, and yet 4 of their patients had positive para-aortic nodes at the time of presentation and, overall, 8 of their patients had lymph node involvement, either at the time of presentation or at the time of recurrence shortly after treatment. Semrad and colleagues,[24] studying patterns of recurrent disease, noted a 71% incidence of extraperitoneal metastases suggesting a strong probability of unrecognized lymphatic invasion at the time of initial therapy. This observation is strengthened by reports of other investigators.[2,3,18] More recently, di Re and co-workers,[25] studying the lymphatic spread of fallopian tube carcinoma in 17 patients undergoing surgical staging, observed an increase in nodal metastases rates with stage of disease and grade. Of note, the percentage of patients with positive nodes was 33%, 66%, and 80% for stage I, II, and III-IV disease, respectively. Overall, patients with negative nodes had a median survival of 76 months, compared with only 33 months if nodal metastases were found.

**Table 114.2. Staging of Carcinoma of Fallopian Tube**

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| Stage 0 | Carcinoma in situ (limited to tubal mucosa) |
| Stage I | Growth limited to the fallopian tubes |
| Stage IA | Growth limited to one tube with extension into the submucosa and/or muscularis but not penetrating the serosal surface, no ascites |
| Stage IB | Growth limited to both tubes with extension into the submucosa and/or muscularis but not penetrating the serosal surface, no ascites |
| Stage 1C | Tumor either stage 1A or 1B with tumor extension through or onto the tubal serosa or with ascites present containing malignant cells or with positive peritoneal washings |
| Stage II | Growth involving one or both fallopian tubes with pelvic extension |
| Stage IIA | Extension and/or metastasis to the uterus and/or ovaries |
| Stage IIB | Extension to other pelvic tissues |
| Stage IIC | Tumor either stage IIA or IIB and with ascites present containing malignant cells or with positive peritoneal washing. |
| Stage III | Tumor involving one or both fallopian tubes with peritoneal implants outside of the pelvis and/or positive retroperitoneal or inguinal nodes. Superficial liver metastases equals stage III. Tumor seems limited to the true pelvis with negative nodes but with histologically proved malignant extension to the small bowel or omentum |
| Stage IIIA | Tumor grossly limited to the true pelvis with negative nodes but with histologically confirmed microscopic seeding of abdominal peritoneal surfaces |
| Stage IIIB | Tumor involving one or both tubes with histologically confirmed implants of abdominal peritoneal surfaces, none exceeding 2 cm in diameter. Lymph nodes negative |
| Stage IIIC | Abdominal implants greater than 2 cm in diameter and/or positive retroperitoneal or inguinal nodes |
| Stage IV | Growth invading one or both fallopian tubes with distant metastases. If pleural effusion is present, there must be positive cytology to be stage IV. Parenchymal liver metastases equal stage IV |

## PROGNOSTIC FACTORS

In view of the inherent difficulties of studying such a rare disease, the role of prognostic factors assumes a greater importance. Stage is the most consistent prognostic factor associated with survival.[1–3,18,26] Although the initial tumor burden does not have predictive significance regarding survival, residual disease after cytoreduction is a strong prognostic factor of survival.[2,3,26,27] Other clinical prognostic factors include the presence of ascites.[3] More recently, Rosen and co-workers[1] reported on prognostic factors in 143 women with primary fallopian tube carcinoma. FIGO stage, histologic grade, and presence of residual tumor had an independent prognostic impact in multivariate analysis. Several histologic factors are prognostic of survival, most notably the extent of tubal invasion. The observation that extent of tubal invasion was associated with a poorer prognosis was first reported by Schiller and Silverberg.[8] Peters and colleagues,[26] analyzing stage I disease, observed a statistically significant increase in the risk of death with invasion of more than 50% of the tubal muscularis. No association between grade and survival was observed by several investigators.[3,5,18]

## TREATMENT

Initial management of carcinoma of the fallopian tube is surgical. No consensus exists regarding the best adjunctive therapy or whether adjuvant therapy has any value. The absence of surgical staging has probably inflated the number of patients assigned to stage I and thereby obscured the subset that might not require adjuvant treatment. However, by analogy with ovarian cancer, it is possible that patients with grade I and stage I tumors require no further therapy. For the remainder, with 5-year survival rates for patients with stage II disease in the range of 50 to 60% and for patients with stage III and stage IV disease in the range of 10 to 20%,[2,3,7,18] consideration and investigation of postsurgical therapy are warranted.

**Surgical Therapy** Since fallopian tube cancer is rarely diagnosed preoperatively, the surgeon is usually confronted with the diagnosis intraoperatively. The new FIGO staging system requires a surgical exercise similar to that mandated for ovarian carcinoma. This includes cytologic analysis of either ascitic fluid or pelvic and abdominal washings, abdominal hysterectomy and bilateral salpingo-oophorectomy, omentectomy, and selective (or therapeutic) pelvic and para-aortic lymphadenectomy with selective peritoneal biopsies. In apparent early stage disease, there may be up to a 33% incidence of nodal metastases,[25] highlighting the importance of performing a selective pelvic and para-aortic lymphadenectomy. For advanced disease, the same principles of cytoreductive surgery for ovarian carcinoma apply to fallopian tube carcinoma as well. The importance of cytoreductive surgery is supported by the observations of Eddy and co-workers,[2] who found the median survival of patients with no gross residual disease was 30 months, significantly longer compared with patients whose largest tumor diameter was greater than 2 cm (17 months).

Several studies examining the role of second-look laparotomy in fallopian tube carcinoma have been performed.[2,5,6,28] Barakat and co-workers[28] reported the largest series of patients undergoing second-look laparotomy. They observed only 19% of patients had a recurrence

after negative second-look laparotomy with a mean follow-up of 50 months. This contrasts with advanced-stage ovarian carcinoma patients treated with platinum-based chemotherapy, among whom approximately 50% will experience recurrence following a negative second-look procedure, with a median interval of 14 months to recurrence.[29] However, more recently, Cormio and co-workers[30] observed that 31% of patients with a negative second look had recurrences, with a mean follow-up of 49 months.

**Radiation Therapy** Because of the transcelomic pattern of dissemination, the use of whole abdominopelvic irradiation, as employed for intermediate-risk ovarian cancer, has been explored as postoperative treatment for patients with fallopian tube carcinoma.[3,4,18] This approach has produced better survival than radiation therapy confined to the pelvis.[31,32] A nationwide retrospective study demonstrated no significant difference in survival between patients treated postoperatively with chemotherapy (cisplatin-based) and those treated with radiation therapy.[1] However, patients in this study were assigned nonrandomly to treatment and there was no stratification for amount of residual disease among the two groups of patients.

There are no data from randomized trials comparing the efficacy of abdominopelvic radiotherapy and cisplatin-containing chemotherapy for the adjuvant treatment of early fallopian tube cancer.

While abdominal irradiation has been used in treating patients with more advanced disease and varying amounts of postsurgical tumor residuum, it does not appear to be of curative benefit.[3,31] Comparable studies in ovarian cancer suggest that adjuvant whole abdominal irradiation can cure only when the postoperative residual tumor is less than 1 to 2 cm in size in the pelvis and there is no macroscopic disease in the upper abdomen. While fallopian tube cancer is sensitive to radiation therapy, the dose that may be delivered safely to the upper abdomen using conventional fractionation is less than that required to eradicate macroscopic disease. Thus, if postoperative abdominopelvic radiation therapy is to be recommended for more advanced disease, it should be reserved for those with microscopic or no residual disease in the upper abdomen and less than a 1-cm residuum in the pelvis.

A possible future direction for treating patients with advanced disease is the exploitation of the cytotoxic activity of both chemotherapy and radiation by using both modalities either sequentially or concurrently. The latter strategy may also take advantage of enhancement of radiation cell kill, resulting in additivity or supra-additivity of effect. If initial chemotherapy can reduce the tumor burden to a microscopic residuum, it may be worthwhile to follow this with whole abdominopelvic irradiation. One report of sequential therapy from Toronto[33] suggested that it was superior to whole abdominopelvic radiotherapy alone. Overall survivals for the sequential therapy were 54% for stage I, 68% for stage II, 28% for stage III, and 25% for stage IV. The overall survival was significantly influenced by treatment, with 5-year survival of 84% following sequential therapy compared with 37% for the historic cohort treated with radiation alone ($p = .0006$). The patient groups were comparable with respect to at least two recognized prognostic factors, stage and residual disease. The overall toxicity was deemed acceptable. The authors concluded that a policy of platinum-based chemotherapy followed by abdominopelvic irradiation led to an improvement in overall survival, and they recommended sequential treatment as their current postoperative management.

**Chemotherapy** Because of similarities in the appearance of papillary carcinomas of the tube and ovary, it was logical to apply to patients with tubal carcinoma the cytotoxic agents known to be active against ovarian carcinoma. Cyclophosphamide, melphalan, and thiotepa were among the frequently used single agents in the early cytotoxic treatment of tubal carcinoma.[5,6,34] Response rates to single alkylating agents were generally less than 20% in small series of patients with disparate stages and prognostic features. With the introduction of cisplatin-containing regimens,[35] complete clinical responses were noted in patients with advanced disease and were confirmed by second-look surgery. Peters and colleagues[6] demonstrated that multi-agent chemotherapy with cisplatin achieved an 81% objective response rate, whereas multi-agent therapy without cisplatin achieved a 29% response rate, and single-agent therapy (other than cisplatin) achieved a 9% response rate. In the cisplatin group, there were 12 complete surgical responses in 20 patients.

Barakat and colleagues[27] reported the treatment of 38 patients with cisplatin-based chemotherapy. While the overall survival of the group was 51% at 5 years, patients with bulky disease who had complete cytoreductive surgery had a 5-year survival of 83% as compared with 28% if there was residual disease postoperatively. Of 21 patients with advanced-stage disease who came to second-look surgery, 11 were found to be without evidence of disease, there was 1 recurrence. Similarly, Cormio and co-workers[36] treated 32 patients with cisplatin, doxorubicin, and cyclophosphamide after primary cytoreductive surgery. The overall clinical response rate was 80%. Ten of 14 patients who underwent second-look laparotomy had a pathologic complete response, but 3 relapsed. The median survival for the entire group was 38 months and the 5-year survival rate was 35%. Other investigators have reported similar experience with platinum-based regimens in the treatment of fallopian tube carcinoma.[37]

During the past decade, as experience increased, it is obvious from the literature that fallopian tube carcinoma responds well to cytotoxic chemotherapy; multi-drug regimens containing cisplatin seem to be more active than nonplatinum single agents or multi-drug regimens without cisplatin. There is no information about the effectiveness of platinum compounds as single agents or paclitaxel alone or in combination with cisplatin. The role of hormonal therapy in the treatment of tubal carcinoma is unclear; however, medroxyprogesterone acetate and megestrol acetate have been employed in the treatment, usually in combination with cytotoxic agents.[3,5,35] There is no evidence that progestational therapy alone is effective in this disease.

A proposed algorithm for the treatment of such patients is given in Fig. 114.1.

## PROGNOSIS

Although most of the larger series have reported recurrent disease later than 5 years after therapy, more than 80% of the recurrences will have happened by the third year from the onset of treatment.[24] If one surveys the results published in the larger series of the last decade, which reported staging similar to the current FIGO system, the 5-year survival for 228 patients is 61% for stage I, 40% for stage II, and 17% for stage III.[3,6,18,32] In two reports, there were no 5-year survivors in stage IV; two series did not mention survivorship for stage IV; and in one, the survival was 25% through the application of extremely aggressive combinations of therapy.[3] In selected series in which patients were treated more recently, Muntz and colleagues[38] reported 5-year survival rates for 35 patients of 100% for stage I, 65% for stage II, 40% for stage III, and 25% for stage IV. In Barakat and colleagues' report,[27] the 5-year survival rate for stages III and IV was 51%. Rosen and co-workers[1] in a retrospective analysis of 143 women, reported a 5-year survival rate of 59% for stages I and II and 19% for stages III and IV. The 5-year survival rate for all stages was 43%.

In an attempt to identify prognostic features, the Austrian Cooperative Study Group for fallopian tube carcinoma analyzed the 66 patients treated in Austria between 1980 and 1990.[19] The group studied the role of inflammatory reaction, nuclear anaplasia, mitotic activity, and progesterone and estrogen receptor concentrations. The distribution of patients was 35% stage I, 20% stage II, 29% stage III, and 16% stage IV. Ascites was found in 24% of patients, but only in FIGO stages III and IV. The overall 5-year survival rate for stages I and II was 50%, and for stages III and IV it was 14%. The authors found no prognostic impact of degree of differentiation or the presence, absence, or concentration of estrogen and progesterone receptors. Lymphocyte infiltration in the tumor was confirmed as a prognostic factor by multivariate analysis, as was stage of disease. In a separate review of the Austrian experience, 61 specimens of fallopian tube carcinoma were examined by image cytometry for ploidy determination. Forty-eight of the tumors showed an aneuploid pattern. While patients with a euploid pattern had a median survival of 33.8 months as compared with 24.5 months for patients with aneuploid tumors, significance was not reached, nor was there a correlation between ploidy status and stage or grade of tumor.[39]

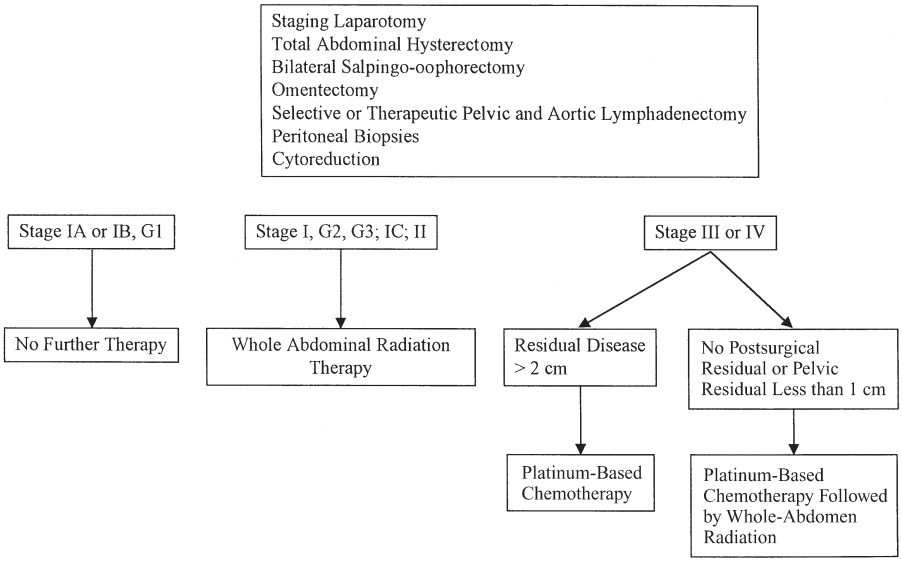


**Figure 114.1.** Treatment algorithn of primary fallopian tube carcinoma.

## OTHER MALIGNANT FALLOPIAN TUBE NEOPLASMS

Fewer than 40 malignant müllerian mixed tumors of the fallopian tube have been reported, as well as rare primary leiomyosarcomas, rare examples of immature teratomas, and 58 occurrences of trophoblastic disease, primarily choriocarcinomas.[40] The diagnostic approach for any of these tumors is the same as that for primary adenocarcinoma of the fallopian tube, except that in the case of trophoblastic disease, human chorionic gonadotropin beta (β-HCG) serum levels and, in the case of malignant teratoma, alpha-fetoprotein levels, are useful. Surgical therapy should be followed by individualized treatment with cytotoxic chemotherapy appropriate to the histology or radiation therapy to postoperative fields according to the primary tumor classification and stage of disease.

## REFERENCES


1. Rosen AC, Klein M, Hafner E, et al. Management and prognosis of primary fallopian tube carcinoma. Gynecol Obstet Invest 1999;47:45–51.
2. Eddy GL, Copeland LJ, Gershenson DM, et al. Fallopian tube carcinoma. Obstet Gynecol 1984;64:546–552.
3. Podratz KC, Podczaski ES, Gaffey TA, et al. Primary carcinoma of the fallopian tube. Am J Obstet Gynecol 1986;154:1319–1326.
4. Benedet JL, White GW, Fairey RN, Boyes DA. Adenocarcinoma of the fallopian tube. Experience with 41 patients. Obstet Gynecol 1977;50:654–657.
5. Rose PG, Piver MS, Tsukada Y. Fallopian tube cancer, the Roswell Park experience. Cancer 1990;66:2661–2667.
6. Peters WA, Anderson WA, Hopkins MA. Results of chemotherapy in advanced carcinoma of the fallopian tube. Cancer 1989;63:836–838.
7. Sedlis A. Primary carcinoma of the fallopian tube. Obstet Gynecol Surv 1961;16: 209–226.
8. Schiller HM, Silverberg SG. Staging and prognosis in primary carcinoma of the fallopian tube. Cancer 1971;28:389–395.
9. Nordin AJ. Primary carcinoma of the fallopian tube: a 20 year literature review. Obstet Gynecol Surv 1994;49:349–361.
10. Rosenblatt KA, Weiss NS, Schwartz SM. Incidence of malignant fallopian tube tumors. Gynecol Oncol 1989;35:236–239.
11. Tamimi HK, Figge DC. Adenocarcinoma of the uterine tube: potential for lymph node metastases. Am J Obstet Gynecol 1981;141:132–137.
12. Johnson GA. Primary malignancy of the fallopian tube: a clinical review of 13 cases. J Surg Oncol 1983;24:304.
13. Ajjimakorn S, Bhamarapravati Y. Transvaginal ultrasound and the diagnosis of fallopian tube carcinoma. J Clin Ultrasound 1991;19:116–119.
14. Kurjak A, Kupesic S, Ilijas M, et al. Preoperative diagnosis of primary fallopian tube carcinoma. 1998;68:29.
15. Niloff JM, Klug TL, Schaetzl E, et al. Elevation of serum CA-125 in carcinomas of the fallopian tube, endometrium and endocervix. Am J Obstet Gynecol 1984;148:1057–1058.
16. Rosen AC, Klein M, Rosen HR, et al. Preoperative and postoperative CA-125 serum levels in primary fallopian tube carcinoma. Arch Gynecol Obstet 1994;255:65.
17. Hu CY, Taymor ML, Hertig AT. Primary carcinoma of the fallopian tube. Am J Obstet Gynecol 1950;59:58–67.
18. McMurray EH, Jacobs AJ, Perez CA, et al. Carcinoma of the fallopian tube management and sites of failure. Cancer 1986;58:2070–2075.
19. Rosen AC, Klein M, Lahousen M, et al. Primary fallopian tube carcinoma in Austria—a retrospective multicenter study. Br J Cancer 1993;68:605–609.
20. Erez S, Kaplan AL, Wall JA. Clinical staging of carcinoma of the uterine tube. Obstet Gynecol 1967;30:547–550.
21. Dodson MG, Ford JF, Averette HE. Clinical aspects of fallopian tube carcinoma. Obstet Gynecol 1970;36:935–939.
22. Petterson F. Staging rules for gestational trophoblastic tumors and fallopian tube cancer. Acta Obstet Gynecol Scand 1992;71:244–245.
23. Alvarado-Cabrero I, Young RH, Vamvakas EC, et al. Carcinoma of the fallopian tube: a clinicopathological study of 105 cases with observations on staging and prognostic factors. Gynecol Oncol 1999;72:367–379.
24. Semrad N, Watring W, Fu Y-S, et al. Fallopian tube carcinoma: common extraperitoneal recurrence. Gynecol Oncol 1986;24:230–235.
25. di Re E, Grosso G, Raspagliesi F, Baiocchi G. Fallopian tube cancer: incidence and role of lymphatic spread. Gynecol Oncol 1996;62:199–202.
26. Peters WA, Andersen WA, Hopkins MP, et al. Prognostic features of carcinoma of the fallopian tube. Obstet Gynecol 1988;71:757–762.
27. Barakat RR, Rubin SC, Saigo PE, et al. Cisplatin based combination chemotherapy in carcinoma of the fallopian tube. Gynecol Oncol 1991;42:156–160.
28. Barakat RR, Rubin SC, Saigo PE, et al. Second-look laparotomy in carcinoma of the fallopian tube. Obstet Gynecol 1993;82:748–751.
29. Rubin SC, Hoskins WJ, Saigo PE, et al. Prognostic factors for recurrence following negative second-look laparotomy for ovarian cancer patients treated with platinum-based chemotherapy. Gynecol Oncol 1991;42:137–141.
30. Cormio G, Gabriele A, Maneo A, et al. Second-look laparotomy in the management of fallopian tube carcinoma. Acta Obstet Gynecol Scand 1997;76:369–372.
31. Sehray MF, Podratz KC, Malkasian GD. Fallopian tube cancer: the role of radiation therapy. Radiother Oncol 1987;10:267–275.
32. Brown MD, Kohorn EI, Kapp DS, et al. Fallopian tube carcinoma. Int J Radiat Oncol Biol Phys 1985;11:483–490.
33. Rawlings G, Bush R, Dembo A, et al. Fallopian tube cancer: survival following radiation and chemotherapy. International Proceedings, Gynecologic Cancer Society 3rd Biennial Meeting; 1991; Cairns, Australia.
34. Boronow RC. Chemotherapy for disseminated tubal cancer. Obstet Gynecol 1973; 42:62–66.
35. Deppe G, Bruckner HW, Cohen CJ. Combination chemotherapy for advanced carcinoma of the fallopian tube. Obstet Gynecol 1980;56:530–532.
36. Cormio G, Manco A, Gabriele A, et al. Treatment of fallopian tube carcinoma with cyclophosphamide, adriamycin, and cisplatin. Am J Clin Oncol 1997;20:143–144.
37. Jacobs AJ, McMurray EH, Partiama J, et al. Treatment of carcinoma of the fallopian tube using cisplatin, doxorubicin, cyclophosphamide. Am J Clin Oncol 1986;9: 436–439.
38. Muntz HG, Tarraza HM, Granai CO, Fuller AF Jr. Primary adenocarcinoma of the fallopian tube. Eur J Gynaecol Oncol 1989;4:239–249.
39. Rosen AC, Graf AH, Klein M, et al. DNA ploidy in primary fallopian-tube carcinoma using image cytometry. Int J Cancer 1994;58:362–365.
40. Young RH, Scully RE. Pathology of tumors of fallopian tube and broad ligament. In: Coppleson M, editor. Gynecologic oncology. Melbourne: Churchill Livingstone; 1992. p. 843–852.