New Developments in Upper and Lower Genital Tract Cancer

Unique to the gynecologic cancers are the direct elements of sexuality, fertility and femininity. The family physician is integral to helping patients navigate their journey through this difficult time of not only life and death, but identity.

By Catherine Popadiuk, MD

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This review will examine simple principles and guidelines to help the family physician integrate important concepts and heighten awareness regarding new developments and controversies in lower and upper genital tract cancer. We will focus on the concepts of screening and prevention, and clinical presentation, and provide guidance as to what to expect when the patient goes through the diagnosis and treatment...
of gynecologic malignancy.

Breast cancer, as the most prevalent cancer—affecting one in nine women—is a good point of comparison. In contrast, the most common gynecologic malignancies, such as endometrial, ovarian and cervical cancer, affect one in 37, one in 70, and one in 100 women, respectively.\(^1\) The remaining gynecologic cancers—vulva, vagina and fallopian tube—occur very infrequently (Table 1). Given the small number of women affected with these cancers, the literature addressing gynecologic oncology does not have many randomized controlled trials with the numbers required to generate statistically significant results. Much of what gynecologic oncology management is based upon are long-standing precepts and center-dependent preferences for management.

### Cervical Cancer

Cervical cancer is the eleventh most frequently diagnosed cancer among Canadian women and the second most common form of cancer in women worldwide. In 2000, 1,450 women in Canada developed it and 430 women died of it. These figures are thought to be underestimated, as death certificates often miscode cervical cancer as being uterine cancer.\(^2\) A classic case was Evita Peron, wife of the president of Argentina, who it was thought died of uterine cancer at age 33. In reality, she died from cervical cancer and had the classic risk factors (Table 2).

These include early age at first intercourse, a high number of sexual partners and exposure to high-risk males (men who have had multiple partners, arguably are uncircumsized or who are from certain geographic locations, such as Africa or Latin America).\(^3\) Prior to Evita’s death, Juan Peron had watched his first wife succumb to the same disease.\(^4\) The risk factors increase one’s chances of being exposed to the high-risk human papillomavirus (HPV) subtypes 16 and 18, arguably associated with all squamous cell and adenocarcinoma of the cervix. Other risk factors include human immunodeficiency virus (HIV) infection. The most important risk factor for cervical cancer is not having had a Papanicolaou’s (Pap) smear. With the advent of Pap smear screening, mortality has decreased 90\%.\(^5\)

In Canada, mortality and incidence from cervical cancer is highest in the elderly, with a sec-

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### Table 1

<table>
<thead>
<tr>
<th>Type</th>
<th>Prevalence</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breast</td>
<td>19,200</td>
<td>5,500</td>
</tr>
<tr>
<td>Lung</td>
<td>8,400</td>
<td>7,000</td>
</tr>
<tr>
<td>Colorectal</td>
<td>7,900</td>
<td>3,000</td>
</tr>
<tr>
<td>Uterus</td>
<td>3,500</td>
<td>670</td>
</tr>
<tr>
<td>Ovary</td>
<td>2,500</td>
<td>1,500</td>
</tr>
<tr>
<td>Pancreas</td>
<td>1,600</td>
<td>1,500</td>
</tr>
<tr>
<td>Cervix</td>
<td>1,450</td>
<td>430</td>
</tr>
</tbody>
</table>

### Table 2

<table>
<thead>
<tr>
<th>Cervical Cancer Risk Factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Not having a Pap smear</td>
</tr>
<tr>
<td>• Early onset of sexual activity</td>
</tr>
<tr>
<td>• Multiple sexual partners</td>
</tr>
<tr>
<td>• Exposure to high-risk male partners</td>
</tr>
<tr>
<td>• Human immunodeficiency virus infection</td>
</tr>
<tr>
<td>• Increased age</td>
</tr>
<tr>
<td>• Lower socioeconomic status</td>
</tr>
<tr>
<td>• Smoking</td>
</tr>
<tr>
<td>• Controversial dietary deficiencies—vitamin A</td>
</tr>
<tr>
<td>• Oral contraceptive pill</td>
</tr>
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</table>
ond peak in the 35- to 40-year-old range in females. In most cases, the patients did not have regular annual screening (up to 15% have never had a Pap smear). The Pap smear detects precancerous cells affordably and simply, so that treatment can be instituted to prevent cancer. The test is not meant to detect cancer cells. The Canadian task force on the periodic health examination recommends women should have annual Pap smears once they are sexually active or at age 18. If an organized screening program is in place, with quality control and information systems, this frequency may be reduced for women to three normal annual smears every three years until age 69. Only British Columbia, Nova Scotia and Prince Edward Island have adopted these suggestions for an organized central screening program (to varying extents).2

Despite these recommendations, women in Canada are still dying from this preventable disease. Most provinces rely on opportunistic screening. Hence, most Pap smears are done frequently in younger women of childbearing age, and less so the older age group, where mortality and incidence are greatest (Figure 1).

We must do better. Public education can help address fears and anxiety about the test. Although the high-risk HPV is more likely to be acquired through multiple sex partners, it only takes one sexual partner to acquire the cancer later in life. A positive Pap smear does not mean cancer. It means abnormal cells have been found that can be treated, thereby preventing cancer. These are the messages to get across to our patients.

Case histories. One patient the author treated for cervical cancer who did not have a Pap smear said she thought she was saving the health-care system money by not being a burden to have the test. Had she been educated about the virtues of having a Pap smear done, she would have come in.

Another tragic case represents the priorities our patients now maintain. A 38-year-old Gravida 2 Para 2 (G2P2) homemaker presented to the emergency department with vaginal bleeding and palpitations. Her hemoglobin was 72 g/l and, following transfusion, she felt better. On history, she reported vaginal bleeding intermenstrually and post-coitally for two years. Over the last two months, the bleeding worsened and was associated with profuse foul-smelling discharge. The patient had experienced trouble urinating for the past year and crampy abdominal pain for the last month. She had not had a Pap smear for 10 years, since the birth of her second child. She had been very busy caring for her children and mother-in-law, who suffered from Alzheimer’s and lived with the family. The patient had no time for herself until she arrived in the hospital, wearing incontinence pads because of leaking urine from a vesicovaginal fistula.

She went on to be staged and was found to have a necrotic tumor extending into the bladder on

Figure 1. Incidence of Pap smears.
cystoscopy. The tumor was eroding out towards both pelvic sidewalls on clinical examination, and in combination with lung metastases that showed up on chest x-ray, made her Stage 4B. Staging does not include computed tomography (CT) scans or the use of expensive high-tech instruments, as the World Health Organization (WHO) recommends simple testing. The reason for this is patients in developing countries, where cervical cancer is the number two cancer, cannot afford expensive staging techniques.

The patient went on to be treated with high-dose “palliative” radiation. Since her radiation two years ago, she has had a good quality of life maintained by a regular, increasing dose of morphine for the pain; oral flagyl to combat the odor from the necrotic tumor; and the essential support of family and her primary-care physician.

More recently, the tumor blocked her femoral veins, causing blood clots and lymphedema of her lower limbs. She was started on low molecular weight heparin, which works better in this cancer situation than warfarin. The anticoagulation caused profuse bleeding from the central tumor. This was successfully treated with vaginal packing and arterial embolization. She then presented with pleural effusions and renal failure. After much thoughtful discussion with her family, she proceeded with percutaneous nephrostomy tube placement and drainage of the effusion. She was not ready to die of renal failure at the age of 40. She is currently stable at home, trying to live to another birthday. The tumor in her vagina and the computed axial tomography (CAT) scan performed on her are shown in Figures 2 and 3.

Cervical cancer, except for the earliest of stages up to 2A, for which radical hysterectomy is offered (sparing the ovaries in pre-menopausal women), is treated with radiation therapy. The most profound development in treatment has been an improvement in local control and survival with the addition of chemotherapy, often cisplatin, as a radiation sensitizer. Given once weekly with the radiation, there are few side effects: no alopecia, nor the renal and neurotoxicity seen with cisplatin.

Chemotherapy is not used to eradicate possible metastases throughout the body, but strictly to “sensitize” the tumor cells to the radiation locally and regionally within the pelvis. Occasionally, after a radical hysterectomy, radiation is given to prevent recurrence if the patient’s features suggest aggressive tumor behavior. Chemotherapy, in a
similar adjuvant setting to prevent distant recurrence, has not been shown to be effective in previous trials. Also, it has not been shown in randomized controlled trials to be effective in a neoadjuvant setting—before definitive treatment with surgery or radiation, in an attempt to shrink the tumor and prevent distant metastatic spread.5

Chemotherapy was not used in the aforementioned patient. As with most cervical cancer patients, she suffers and will die from the central tumor effects. Systemic chemotherapy, a platinum agent, can be used to attempt to decrease lymph node involvement or the lung metastases. With chemotherapy in such a setting, morbidity must be considered, particularly if the patient is not symptomatic in these areas.

Vulvar and Vaginal Cancer

Abnormalities found in Pap smears and a history of prior treatment for cervical dysplasia necessitates vigilant follow-up of the entire lower genital tract, which is predisposed to the same ill effects by HPV. Vaginal cancer is extremely uncommon, particularly without a prior history of other vulvar or cervical dysplasia. Hence, Pap smear screening should be continued after a hysterectomy if there is any question regarding the patient’s past Pap smear history. If dysplasia is found on Pap smear, and confirmed on colposcopic biopsy, treatment with surgical excision, laser or fluorouracil 5FU intravaginal chemotherapy cream is used to eradicate it. For recalcitrant dysplasia or invasive cancer, radiation therapy is the treatment.

Vulvar cancer accounts for 5% of all gynecologic malignancies, with 90% being squamous in origin. Like cervical cancer, vulvar cancer is most frequently found in women 65 to 75 years old, but 15% occur in women under 40.5 In this latter group, vulvar cancer is thought to result from dysplasia secondary to HPV.8 This is less the case in the older woman. There is little data to support the concept that these neoplasms develop from vulvar dystrophies, such as lichen sclerosis or squamous cell hyperplasia. They are, however, often seen within such areas.

Vulvar pruritis is the primary complaint (Table 3). In more than 50% of patients, long-term pruritis or a lump on the vulva is seen on presentation. In most reported series, patients delay seeking medical advice for up to 16 months. Furthermore, treatment is initiated with steroid creams for up to 12 months or longer prior to biopsy for definitive diagnosis.5 All suspicious lesions on the vulva, regardless of pruritis, should be biopsied prior to treatment. Eutectic mixture of local anesthetics (EMLA) cream and xylocaine injected with a tiny caliber needle is necessary prior to a punch biopsy in this sensitive area. Seventy per cent of vulvar cancers occur on the labia, most commonly the labia majora.

Once a diagnosis of vulvar cancer is made, staging and treatment involves surgery with possible radiation. In the past, treatment included radical surgery in the form of an extensive vulvectomy and bilateral inguinal femoral and pelvic lymph node dissection. More recently, the role of mutilating surgery has diminished, and radiation has taken on a greater role.9 For well-circumscribed lesions away from the rectum or urethra, a minimum 1-cm margin of tissue is removed around

Table 3
Symptoms of Vulvar Disease

- Pruritus
- Burning
- Lump/mass
- Ulceration
- Pigmented areas
the tumor, and inguinal femoral lymph node dissection performed if the tumor is more than 1 mm in depth. If the lymph nodes are involved, adjuvant radiation treatment is offered to prevent pelvic lymph node metastases and regional recurrence. Survival has been shown to be improved with bilateral radiation to the pelvis.10

Similarly, if the tumor involves vital structures, such as the anus or rectum, radiation is now being used as an initial modality to shrink the tumor and avoid extensive surgery. Again, except as a radiation sensitizer, chemotherapy plays a limited role in the treatment of vulvar cancer.

Endometrial Cancer

Endometrial cancer is the most likely gynecologic malignancy family physicians will encounter. Seventy-five per cent of patients are pre-menopausal, and abnormalities in vaginal bleeding cycles are more problematic to discern from normalities. Vigilance on the part of the physician in these instances is essential. Given the hallmark sign of abnormal bleeding, 75% of endometrial cancers are diagnosed as Stage 1, confined to the uterus.

Associated factors for endometrial cancer include unopposed estrogen production (Table 4). The unchecked estrogen promotes hyperplasia in the endometrium. If atypia is seen, the risk for endometrial cancer progression rises to 29%. Progesterone treatment may be tried in younger women hoping to achieve fertility, but a hysterectomy is preferable, as 20% of patients with endometrial hyperplasia and atypia harbor cancer not detected on initial diagnostic biopsy.11

Although a diagnosis can be made relatively easily with endometrial biopsy, this is not seen as a screening modality to be done in all women on an annual basis. The cost effectiveness of screening asymptomatic women for endometrial cancer and its precursor lesion is very low. Endometrial sampling is not required prior to, or during, estrogen-progesterone hormone replacement therapy (HRT) unless unexpected bleeding occurs (i.e., outside monthly withdrawal bleeding on cyclical regimen, or sporadically after six months on continuous regimen).5

The Pap test has insufficient sensitivity for endometrial cancer. Endometrial cells present on a smear of a post-menopausal woman, however, merit investigation with biopsy. When a biopsy cannot be successfully completed in the office due to cervical stenosis, transvaginal ultrasound may discern an atrophic endometrium (endometrial thickness < 4 mm) from a thickened lining. The interpretation can be problematic, as cancers have been seen with endometrial linings less than 5 mm. Definitive investigation with a dilation

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Table 4
Endometrial Cancer Risk Factors

<table>
<thead>
<tr>
<th>Risk Factor</th>
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<tbody>
<tr>
<td>Unopposed estrogen</td>
</tr>
<tr>
<td>Obesity</td>
</tr>
<tr>
<td>Nulligravid</td>
</tr>
<tr>
<td>Early menarche</td>
</tr>
<tr>
<td>Late menopause</td>
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<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
</tr>
<tr>
<td>Anovulatory cycles</td>
</tr>
<tr>
<td>Polycystic ovarian disease</td>
</tr>
<tr>
<td>Hereditary non-polyposis colon cancer</td>
</tr>
<tr>
<td>Tamoxifen?</td>
</tr>
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and curettage (D and C) may be necessary. As well, persistent bleeding despite a normal pipelle biopsy (with 95% sensitivity) should be investigated further with D and C.

Recently, the question of whether or not tamoxifen causes endometrial cancer has been suggested, following a number of case reports and series. The National Surgical Adjuvant Breast Project (NSABP) results have put the risk into perspective. Patients who were found to be node negative following surgery for breast cancer were randomized to placebo or tamoxifen. The benefits of tamoxifen to prevent 121 breast cancers favorably outweighed the risk of 6.3 endometrial cancers in the tamoxifen-treated group. The stage and grade of the endometrial cancers associated with tamoxifen were no worse than those with sporadic endometrial cancer. Hence, for women on tamoxifen, the endometrium should be evaluated if the patient is symptomatic. The cost of screening these women annually for adenocarcinoma precursors would be prohibitive.12

The staging and treatment of endometrial cancer is surgical: a total abdominal hysterectomy, bilateral salpingo-oophorectomy and pelvic and/or para-aortic lymph node dissection at the discretion of the surgeon.13 The decision to use adjuvant treatment in the form of radiation therapy to the pelvis is now based more frequently on consideration of tumor factors seen in the hysterectomy specimen, and less so on lymph node status (although this is a part of staging). Prognosis depends on stage, grade, myometrial invasion and histology (clear cell and papillary serous subtypes being very poor).

Again, chemotherapy has not yet been conclusively shown to improve survival in an adjuvant setting. For advanced stages or poor histologic subtypes, chemotherapy may be incorporated into treatment with limited response rates. There is questionable impact on survival, given low patient numbers to discern statistically significant benefits.

The question of resuming or starting HRT following treatment for endometrial cancer is a sensitive issue. In the past, HRT was withheld, as it was thought to be integral to the initiation and propagation of disease.

The question of resuming or starting HRT following treatment for endometrial cancer is a sensitive issue. In the past, HRT was withheld, as it was thought to be integral to the initiation and propagation of disease. Current thought is that if there are no remaining cancer cells in the body, HRT will not initiate the process. Hence, HRT is now being considered in endometrial cancer patients. With recurrence or advanced stage dis-
ease, high-dose progesterone is used to control disease progression and treatment. A response can be seen in 15% of patients, particularly in estrogen receptor/progesterone receptor (ER/PR)-positive patients. It is a bit of a conundrum that, in the past, estrogen would be given only to patients with Grade 1 tumors with minimal myometrial invasion, and withheld from patients with Grade 3 disease, which was less likely to be hormone responsive. The former patients were more likely to be estrogen receptor positive and responsive to the hormone if tumor cells were present.

Case history. This case history will show the peculiarities of this disease. The patient is an 84-year-old G9P9 female, who is obese, diabetic and hypertensive. She presented with an episode of post-menopausal bleeding. She was not on HRT. An endometrial biopsy showed Grade 2 endometrial adenocarcinoma. She had a total abdominal hysterectomy and bilateral salpingo-oophorectomy. The final pathology showed Grade 2 endometrial cancer, with invasion into the outer half of the myometrium (Stage 1CG2).

Given her high risk of recurrence in the vaginal vault area and pelvis, she was given adjuvant radiation therapy to the pelvis and upper vaginal vault. This was started two months after her surgery, and was completed over a six-week treatment plan. She was not offered HRT, as she never took it in the past. Four months later, she presented with vaginal pruritis and discharge. She was treated with antibiotics and then with flagyl to no avail. Finally, after another two months, a 3-cm recurrence of endometrial cancer was seen and palpated in the posterior lower third of the vagina, just above the anal sphincter and outside the radiation field. Metastatic work-up showed three lung nodules of new onset.

The patient was started on progesterone therapy, megestrol acetate 80 mg three times daily, and treated with more radiation beyond the initial field. Fortunately, she did not develop a recto-vaginal fistula from the radiation, although she did suffer severe erythema and local skin desquamation. Her lung lesions disappeared with the progesterone, and she continues to do well on her progesterone two years post-recurrence. She has not received chemotherapy, although this may be considered in some centers.

Ovarian Cancer

Ovarian cancer is the most lethal among the gynecologic malignancies. It affects one in 70

Gilda Radner was diagnosed with hereditary ovarian cancer at the age of 39. She initially responded to treatment, however, succumbed to her disease three years later at the age of 42.
women, and is the fifth most diagnosed and fifth leading cause of cancer deaths. Ninety per cent of ovarian cancers are from the surface epithelium, similar to the mesothelium lining the fallopian tubes and peritoneal cavity. The non-invasive low malignant potential (borderline) category has an excellent prognosis and is not considered a precursor lesion for invasive epithelial ovarian cancer.

Risk factors and protective factors relate to fertility and ovulation. The use of oral contraceptives, term pregnancy and breastfeeding are protective. The use of fertility agents, such as clomiphene citrate, is controversial, as it is unclear if an inherent abnormality leading to infertility is the problem, as opposed to the fertility agents. Five per cent to 10% of ovarian cancer is thought to be hereditary or familial. The BRCA1 gene predisposes an individual to a 50% to 70% lifetime risk of breast cancer and a 20% to 40% risk of ovarian cancer. The BRCA2 gene confers a 55% to 80% risk of breast cancer and 10% to 20% risk of ovarian cancer. The HNPCC gene, in addition to endometrial cancer risk, is associated with ovarian cancer. Studies regarding differences between hereditary and sporadic ovarian cancers are inconclusive as to any differences between them, except for the incidence of the hereditary cancers occurring at a younger age. Genetic counseling is essential in these patients concerning potential oophorectomy and ongoing ovarian evaluation. The largest proportion of sporadic ovarian cancer occurs in the older woman, 65 to 79 years old.

Despite major technologic advances and decades of research, the mortality from ovarian cancer has not changed. More than 60% of patients present with advanced disease—Stages 3 and 4. Childbearing age. There are ongoing randomized controlled trials in England, which are evaluating the effectiveness of screening in postmenopausal women who, by virtue of age, are at higher risk. Results are expected by 2004. A new potential tumor marker is being investigated. Lysophosphatidic acid (LPA) is a phospholipid, implicated as a growth factor present in the ascites of ovarian cancer patients. It is now being evaluated in trials as a biomarker for ovarian and other gynecologic malignancies. These tests are promising, but are not yet avail-
able to the general population. Pelvic examinations are encouraged, however, it is difficult to discern subtle abnormalities using this method. By the time changes are felt, the disease has spread.

With respect to prognosis, the following factors have been assessed: stage, surgical resectability, platin chemotherapy response, grade, histology, ploidy analysis and performance status. Higher grade, stage, chemo resistance and poor performance status are associated with poorer survival.15

Once the ovarian cancer patient recurs, she is incurable. When she recurs—be it two months, six months or a year after completing treatment—it dictates her potential to respond to further chemotherapy and to be salvaged for a while longer.

There are no differentiating features in the tumor pathology to suggest which patients will do “well” and respond to treatment for some duration, or which will not and die within months of diagnosis. Dr. Robert E. Scully, a leading gynecologic pathologist, summarized this conundrum as follows:

“Knowledge of the pathology of ovarian tumors is essential to understanding their behavior and selecting the optimal therapy. The ovary is the site of a wider variety of tumors than any other organ, and the oft-repeated precepts that ovarian neoplasia is not one, but many, diseases is fully justified by the range of its biological manifestations.”18

What is the patient to expect following a diagnosis of ovarian cancer? In the uncommon event that cancer was found incidentally during a hysterectomy or oophorectomy, the gynecologic oncologist may suggest further surgery to complete a hysterectomy and lymph node analysis for staging and debulking. Generally, adjuvant chemotherapy, including cisplatin or carboplatin, and paclitaxel is recommended for six cycles every three weeks to patients with more than Stage 1A/B G1/2 ovarian cancer. This may prevent recurrence. The chemotherapy is now well tolerated with modern anti-emetics. The most disturbing side effect is the total body alopecia and muscle aches from the paclitaxel. The myelosuppression is less of a problem.

When a patient presents with obvious advanced disease (i.e., ascites, pelvic masses, pleural effusion, omental caking and elevated CA125 levels), she should be referred to a gynecologic oncologist. In the past, aggressive surgery was undertaken and chemotherapy started. Survival correlated with the ability to debulk the patient optimally. If any residual tumors were left, the patient’s survival was compromised.

Currently, the gynecologic oncology community is grappling to understand the role surgery plays in a patient’s survival. The biologic behavior of the tumor is being looked at critically within the context of surgical resectability.19 In 1995, the Van Berg group made a major breakthrough in suggesting that the ability to perform optimal interval debulking after three cycles of chemotherapy in previously unresectable patients improved survival.20

Many centers in Canada are now participating in the National Cancer Institute of Canada (NCIC) OV13 trial, randomizing patients to up-front surgical debulking versus chemotherapy first for three cycles and then debulking surgery; to see if there is any difference in survival, depending on when the surgery is performed. This is a major shift in thought from when the traditional management was surgery, first and foremost. As well, the NCIC is also running OV14, where patients are randomized to carboplatin and paclitaxel, the current standard chemotherapy treatment, to carbo-
platin, paclitaxel and epirubicin. Median survival for advanced Stage 3C disease approached three years with the advent of paclitaxel in 1996.

Improvements are being sought with new innovative combinations of chemotherapy. Treatment is usually limited to six cycles, as further cycles have not been shown to improve survival. A second-look laparotomy upon completing treatment also is no longer done, except in research settings. Even in patients with no residual disease, one half would experience recurrence and ultimately succumb to their disease. Once the patient fails to respond initially to treatment, or has a recurrence, she is incurable.

This raises the question of initiating follow-up to detect a potential recurrence. Monitoring CA125 levels was thought to be helpful, but this elevation has been shown to occur approximately five months prior to the presentation of symptoms. Starting chemotherapy earlier in an asymptomatic patient has not been shown to improve survival. Hence, many centers no longer follow CA125 levels in asymptomatic patients, after the completion of initial treatment. When a patient has a recurrence—be it two months, six months or a year after treatment completion—dictates her potential to respond to further chemotherapy and to be salvaged for a while longer. The later the recurrence, the more likely the patient will respond to treatment again.

For example, Gilda Radner was diagnosed with hereditary ovarian cancer at the age of 39. She initially responded to treatment, but succumbed to her disease three years later, at the age of 42. Liz Tilberis, editor of Harper’s Bazaar, was diagnosed at 44 years of age. Although she had multiple recurrences and treatments, she died after six years at the age of 51. In contrast to these women, actress Madeline Kahn had very aggressive disease. She died at the age of 57, only one year after being diagnosed. All these women had advanced ovarian cancer. What was the inherent difference in the biology of their tumors to account for such variable outcomes in spite of similar initial treatment? We have yet to answer this question.

Irrespective of the type of gynecologic malignancy the family physician may encounter, there are special issues that need to be considered, particularly those regarding survival, identity and sexuality. It is estimated that 50% of patients treated for gynecologic cancer suffer from some type of sexual dysfunction. Unfavorable changes to sexual desire and fulfillment have been attributed, not only to the psychologic duress a diagnosis of cancer has on a patient’s mind, but to the physical changes resulting from the cancer treatments themselves. The vagina may be shortened from a radical hysterectomy or vulvar reconstruction. Irradiation affects tissue elasticity and the ability for lubrication. This is further exacerbated by induced menopause because of surgical removal or radiation of the ovaries.

Furthermore, if the patient presented with some form of vaginal bleeding, the trauma of intercourse on atrophic friable vaginal mucosa is frightening to both partners. Although couples shy away from sharing intimacy during the cancer experience, communication and closeness are integral to the healing process and should be encouraged. There is no greater advocate to help the patient and partner through this ordeal than the family physician. Suggestions for polycarbophil vaginal moisturizer lubricant and advice about the importance of keeping the vagina patent with dilators if the patient is not sexually active after radiation come best from a familiar face. Patients appear to regain their sexual identity with the treatment of their disease and the passage of time.

The bottom line in our gynecologic oncology...
pot pourri is that the greatest obstacle following such a diagnosis is not only the fear of death, but the enhanced fear of the unknown and the sense of isolation, as there are few active large-scale support groups to share these particularly intimate feelings. Unique to the gynecologic cancers are the direct elements of sexuality, fertility and femininity. Cancer of the vulva, vagina and cervix directly impact on one’s self esteem. For endometrial and ovarian cancer, and any disease treated with radiation therapy to the pelvis in premenopausal women, the resulting castration from ovarian loss is an important consideration as well. The family physician has an integral role in helping this patient navigate her journey through this difficult time of not only life and death, but identity.

References

Suggested Readings
4. LoCoco S, Covens A, Carney M, et al: Does aggressive therapy improve survival in suboptimal stage III/IV ovarian...
low grade squamous intraepithelial lesion. HPV 6 and 11 is the cause of. condyloma acuminatum -recurrent respiratory papillomatosis. high risk HPV strands. 16 and 18. HPV 16 and 18 cancers. condyloma acuminatum aka. low grade squamous intraepithelial lesion or vulvar intraepithelial neoplasia. micro of condyloma acuminatum. branching -koilocytosis -hyperkeratosis/parakeratosis -acanthosis. high grade vulvar intraepithelial neoplasia. squamous dysplasia of the vulva affecting most layers of epithelium, but without stromal invasion. high grade VIN precedes the development of invasive cancer and may be found in separate foci or may coexist with the lesion. usual VIN cause. hpv 15 -coexist with squamous dysplasia in cervix, vagina, and anus.