

Mini-Review

Diseases Manifesting in the Upper Genital Tract in Children and Adolescents: A Review

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Introduction

Some of the gynecologic conditions affecting children and adolescents are rarely encountered by providers who have not developed a specific expertise in the area, and hence may pose diagnostic difficulty when encountered. In a previous review,¹ conditions affecting the lower genital tract of children and adolescent females were discussed. The purpose of this review is to provide an overview of conditions of the upper genital tract affecting female children and adolescents, some of which may be encountered only rarely by the more generalized practitioner. Gynecological examination techniques must be tailored to the age of the patient. Several review articles deal with examination techniques for both prepubertal children and adolescents.^{2–5}

Adnexal Masses

The risk of malignancy in any female under age 20 with an adnexal mass is low, and so ovarian preservation should be achieved whenever possible.⁶ In Cass' series⁶ of 106 procedures on 102 patients ranging in age from 2 days to 20 years, 49 adnexal masses were nonneoplastic and diagnoses included torsion, functional cyst, paraovarian cyst, hemo/hydrosalpinx, and ovotestis. Forty-six cases were benign neoplasms, mainly mature cystic teratomas. The remaining 11 were a variety of germ cell and stromal neoplasms.

Ovarian torsion can occur with or without the presence of a neoplasm. In Mordehai's series,⁷ there were 8 cases of postnatal torsion with additional pathology, and 6 cases with normal adnexa. This group suggested performing fixation of the contralateral ovary in cases of torsion of a normal adnexum. Detorsion has also

been recommended in some circumstances, but remains controversial.

Pediatric ovarian masses may be considered in three separate groups, fetal/neonatal, prepubertal, and postpubertal.

Fetal and Neonatal Adnexal Masses

With the increased use of prenatal ultrasound, adnexal masses in the female fetus are being detected more frequently. Most of these masses are maternal hormonally-related follicular cysts that will regress; however, some persist. Rarely, a follicle cyst in the newborn may become large enough to present as an abdominal mass, and rare giant follicle cysts in the newborn have been encountered. Persistent cysts, particularly if over 5 cm, may possibly torsion. Surgery, if undertaken, should maximize ovarian preservation.⁸ In some cases, aspiration is performed. The differential diagnosis of fetal intra-abdominal cystic masses includes a wide variety of gynecological and nongynecological conditions including genitourinary disorders, gastrointestinal disorders, and miscellaneous conditions.⁹

Adnexal Masses in Premenarchal Girls

In Quint's series¹⁰ of 52 premenarchal girls who underwent ovarian surgery, the most common finding was torsion (18 cases), often unsuspected; mass without torsion (16 cases) was the second most common finding. Other final diagnoses included 5 hernias, 8 chromosomal abnormalities, and 5 malignancies. Pathology reports confirmed 19 hemorrhagic infarctions, 8 dysgenetic gonads, 7 simple cysts, 6 teratomas, 4 theca lutein cysts, and 1 each fibroma, stromal tumor, mucinous cystadenoma, granulosa cell tumor, uterine neuroblastoma, mixed germ cell tumor, metastatic Wilms tumor, and gonadoblastoma. Functional ovarian cysts were noted to be rare in this prepubertal

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cohort. Prepubertal ovarian cysts may be associated with premature thelarche or precocious puberty.¹¹

Cass et al⁶ evaluated 102 girls with ovarian pathology treated at their institution. The mean age was 9.8 years. Of 59 cases presenting with acute abdominal pain, 42% had a torsion, of which a third were associated with benign cystic teratomas. Only one case was associated with malignancy. Of those who presented with a mass, 26% had malignancies, with germ cell tumors most common, and sex-cord stromal neoplasms second in frequency in this group.

Although rare, metastatic disease to ovaries may also present in children. Signs are similar to those seen in adults, including bilaterality, large masses, ascites, peritoneal implants, matted bowel, adenopathy, and/or pleural effusion.¹² Malignancies metastatic to the ovary in McCarville's series¹² included predominantly neoplasms seen in the pediatric age group, except for colonic adenocarcinoma, and also included Burkitt lymphoma, alveolar rhabdomyosarcoma, Wilms tumor, neuroblastoma, and retinoblastoma.

Adnexal Masses in the Adolescent Female

The majority of adnexal masses in adolescents are functional ovarian cysts, which usually resolve. The most common neoplasms are germ cell neoplasms, with stromal neoplasms second, and epithelial neoplasms last in frequency. This is in distinction to adult women who have predominantly epithelial neoplasms. Most germ cell neoplasms in the adolescent are benign cystic teratomas. These may be asymptomatic, or may present with pain, a mass, or gastrointestinal symptoms.¹³ Due to the contents, dermoids are heavy for their size, and are prone to torsion. Antecedent or intraoperative spillage of the contents can cause a chemical peritonitis. The lesions are usually treated by cystectomy. Caution must be exercised in cases of suspected benign cystic teratomas, particularly if large or solid, as immature teratomas may present similarly.

The remainder of ovarian germ cell tumors are malignant. With advances in chemotherapy, prognosis is significantly improved for these neoplasms (see section below on ovarian malignancy).

Ovarian Malignancies

The majority of genital tract malignancies in the adolescent are of ovarian origin, and are usually germ cell neoplasms in this age group. The most common is a dysgerminoma. A small percentage of these arise in intersex individuals, who may also have gonadoblastomas,¹³ which are essentially in situ malignancies. Other malignant germ cell neoplasms that may occur include yolk sac (endodermal sinus) tumor, embryonal carcinoma, immature teratoma, mixed germ

cell tumors, and the exceedingly rare polyembryoma and choriocarcinoma. Preservation of hormonal function and future fertility are of particular importance in this age group. Newer chemotherapy has considerably improved the prognosis of malignant germ cell tumors of the ovary. Many of the malignant germ cell tumors express markers, which can aid in diagnosis and follow-up of the patient.

The second most common malignant ovarian neoplasms in the adolescent, representing 10% of adolescent ovarian malignancies,¹³ are sex cord stromal tumors. These include granulosa cell tumors, many of the juvenile histology, as well as Sertoli-Leydig or Sertoli tumors. These lesions may produce estrogen, leading to menstrual irregularities, or may lead to virilization due to androgen production. Juvenile granulosa cell tumors in prepubertal children may present as precocious pseudopuberty. These neoplasms may also be part of several syndromes, including Ollier's disease (multiple enchondromas), and Maffucci's syndrome (multiple enchondromas and hemangiomas).

Malignant epithelial ovarian neoplasms in adolescents almost exclusively occur after puberty, with an age range of 14–19 years.¹³ Many are of low malignant potential ("borderline"). Preservation of fertility by leaving the uterus and contralateral ovary in low stage disease, both borderline and invasive, is a major consideration. Pregnancies conceived by IVF have been carried in uteri of patients with surgically absent ovaries. Frozen section may be utilized in the planning of the extent of procedure, but it should be noted that a diagnosis of borderline tumor on a frozen section does not rule out invasive carcinoma being detected on permanent sections with additional sampling. Similarly, large mucinous neoplasms may have large areas that are histologically benign admixed with borderline areas, and hence frozen sections on these neoplasms are unreliable in establishing the final diagnosis due to sampling issues.

An excellent review of the pathology of ovarian neoplasms in children and adolescents was published by Lack et al.¹⁴

Menstrual Disorders in the Adolescent

Menstrual problems affect 75% of adolescent females,¹⁵ and include amenorrhea, dysmenorrhea, and abnormal uterine bleeding. Most abnormal vaginal bleeding in the prepubertal child is nonuterine, and related to the lower genital tract. This has been covered in the prior review.¹ Vaginal bleeding in the newborn may be associated with withdrawal of maternal hormones.

Polycystic Ovarian Disease (PCO)

PCO is the most common female endocrinopathy, occurring in approximately 6% of reproductive-age

women.¹⁶ The condition often has its first manifestation in adolescence. While the ovaries may be slightly enlarged, issues relating to the endocrinopathy, including menstrual irregularities, are the most common presenting symptoms. The constellation includes hyperandrogenism/hyperandrogenemia, oligoanovulation, and insulin resistance. It is unclear which comes first, but there is increased gonadotropin releasing hormone (GnRH), increased luteinizing hormone (LH; persistent steady state rather than normal surge), and increased insulin, leading to increased ovarian production of androgens. The androgens are converted in peripheral fat to estrone, and the ovaries also make increased estradiol. This increased estrogen leads to the risk of endometrial hyperplasia and carcinoma. Increased androgens lead to masculinization, and ovarian follicular atresia.

Recent studies suggest a prenatal connection to PCO, with low birth weight being associated with PCO.¹⁷ PCO is now considered a life-long disease.¹⁸ Long-term sequelae of PCO include cardiovascular disease (hypertension, dyslipidemia), endometrial hyperplasia/carcinoma, infertility, and type 2 diabetes development. Therapy includes oral contraceptives (progestins decrease ovarian androgenicity and cycle endometrium; the estrogen in oral contraceptive pills increases liver sex hormone binding globulin), antiandrogens, topical hair suppression, GnRH agonists (for severe hair-AN syndrome [hyperandrogenism, insulin resistance, and acanthosis nigricans (AN)]), ovarian induction of ovulation if pregnancy is desired, ovarian drilling (there is no adolescent data), and insulin sensitizing drugs such as metformin. Long term complications of therapy are unknown. Wedge resection of the ovary, once routine, is no longer performed. The polycystic ovary is covered by a dense tunica, under which numerous primordial and developing follicles may be seen. While there are usually no stigmata of ovulation (corpora lutea or albicans), an occasional such structure may be seen.

Amenorrhea

The classical definition of primary amenorrhea is the absence of menses by age 16; however, a child who is being seen for possible primary amenorrhea needs to be evaluated as to when development started and how it progressed to make a true determination as to whether the condition is present. Secondary amenorrhea is the cessation of menses after onset of menarche.

Hypogonadism may result in either primary or secondary amenorrhea, depending on the condition. Hypogonadism may be hypergonadotropic, hypogonadotropic, or eugonadotropic. Evaluation involves assessment of growth, development, including body mass index and pubertal stage. Follicle-stimulating

hormone (FSH) and LH levels are helpful in steering further investigation.¹⁵ Causes of primary amenorrhea include hypogonadism, and some Müllerian anomalies. Causes of secondary amenorrhea in an adolescent include pregnancy, medications such as oral contraceptives, hypogonadism, ovarian-related conditions, outflow tract obstruction such as imperforate hymen or transverse vaginal septum, some Müllerian anomalies, Asherman's syndrome (uterine synechiae), conditions associated with androgen excess (polycystic ovarian disease, adrenal neoplasm or hyperplasia, ovarian neoplasms) and other endocrine conditions including thyroid disease and Cushing's syndrome.¹⁹

Hypogonadotropic Hypogonadism (low to normal FSH/LH)

Hypogonadotropic hypogonadism includes a wide spectrum of conditions including anorexia and other systemic illnesses or chronic disease, stress, GnRH deficiency, hyperprolactinemia, and hypopituitarism. Other etiologies include marked athletic activity, Kallman's syndrome, central nervous system tumors and their therapy, some syndromes such as Laurence-Moon-Biedl or Prader-Willi. Pituitary causes include neoplasms such as craniopharyngioma, infiltrative conditions such as sarcoid, or tuberculosis, hemochromatosis, head injuries, postpartum necrosis, and empty sella. Endocrinopathies, depression, drugs, and physiologic delay may also result in hypogonadotropic hypogonadism.

Hypergonadotropic Hypogonadism (high FSH/LH)

Amenorrhea, both primary and secondary, may be associated with hypergonadotropic hypogonadism. Causes include premature ovarian failure (autoimmune oophoritis, resistant ovary syndrome), radiation, chemotherapy, surgery, torsion, 17 hydroxylase deficiency, and some hereditary conditions (galactosemia, trisomy 21, ataxia telangiectasia, and myotonia dystrophica). A number of the intersex conditions, including Turner's Syndrome and XY gonadal dysgenesis, fall under the category of hypergonadotropic hypogonadism.¹

Eugonadotropic Hypogonadism

Causes of eugonadotropic hypogonadism that may present as secondary amenorrhea include polycystic ovarian syndrome, illness, stress, weight loss (here there is some overlap with the hypogonadotropic category), and neoplasia. Eugonadotropic causes of primary amenorrhea include conditions of mechanical obstruction to outflow, Müllerian agenesis, Asherman's syndrome, and polycystic ovarian syndrome.¹⁵

Most of the workup for amenorrhea is clinical, including history, growth charts, diet, examination, FSH/LH/prolactin, bone density. If androgen excess is present, additional studies such as testosterone, free testosterone, dehydroepiandrosterone, 17-OH progesterone, and a morning DHA may also be performed. Assessment of estrogen levels can be done clinically (progesterin challenge, estradiol levels), but can also be done by vaginal smear for maturation index. The maturation index is evaluated on a pap smear slide prepared specifically from the lateral vaginal wall. It is performed by counting 100 cells, and assessing the ratio of parabasal cells to intermediate cells to superficial cells. A prepubertal child might have an atrophic ratio such as 90/10/0, while a pubertal girl might have one of 0/100/0.²⁰

Abnormal Uterine Bleeding

Abnormal vaginal bleeding may not always be of uterine origin, and it may be divided into nongenital (ie bladder/rectal), nonuterine genital (vulva, vagina, cervix), secondary to systemic disease, pregnancy-related, intrinsic uterine disease, dysfunctional (hormonal imbalance), or hyperplasia/neoplasia.

In an adolescent, abnormal uterine bleeding may be pregnancy related, related to medication, due to hypothalamic or pituitary conditions, relating to outflow tract issues such as trauma, foreign body, tumor, or partially obstructing Müllerian anomaly, or due to androgen excess as in PCO, adrenal or ovarian tumor, or adrenal hyperplasia. It may also be secondary to other endocrine causes such as thyroid or adrenal disease, may be hematologically related, or secondary to infectious conditions such as pelvic inflammatory disease or cervicitis.¹⁹ Anatomic (organic) causes of abnormal bleeding are not always of uterine origin, and may include lesions of the cervix (cervicitis, condyloma, polyps, sarcoma botryoides), or ovary (PCO, estrogen-producing tumors such as thecoma, juvenile granulosa cell tumor, Sertoli-Leydig cell tumor, yolk sac tumor), or vulvovaginal conditions such as inflammation, trauma, infectious, or malignancy (sarcoma botryoides, clear cell adenocarcinoma). In addition to Müllerian anomalies and pregnancy related causes, uterine etiologies include endometritis, endometrial hyperplasia or malignancy (particularly in the setting of PCO), endometrial polyps, and leiomyomata.²¹

Abnormal uterine bleeding secondary to an organic etiology is the least common in the adolescent.

Dysfunctional Uterine Bleeding (DUB)

After thorough evaluation, most adolescent abnormal uterine bleeding is found to be dysfunctional uterine bleeding, defined as bleeding in the absence of

intrinsic uterine disease, systemic disease, or pregnancy, and hence it reflects hormonal imbalance. This is common at both ends of reproductive life, and usually, although not always, relates to anovulation and unopposed estrogenic stimulation of the endometrium. This results in a buildup of a thick but unstable endometrium that sheds dyssynchronously, leading to prolonged periods of bleeding, as this shedding interferes with the normal mechanism of spiral artery constriction and thrombosis. Most DUB in the adolescent is treated medically; however, occasionally more invasive intervention is necessary.

Endometrial Biopsies in Adolescents

Endometrial biopsies are rarely performed on adolescents, but a rare one will be received by the laboratory for intractable problems not ameliorated by medical therapy. Endometrial biopsies can be difficult to interpret, due to technical issues, hormonal changes (endogenous and exogenous), and lack of clinical history supplied to the pathologist. Endometrial sampling is frequently performed in the clinician's office, due to simplicity and cost issues, and has been shown to be accurate under most circumstances, but may provide insufficient tissue, or may miss a focal lesion such as a localized carcinoma or polyp. Clinicians may be under the impression that the endometrial samples are abundant; however, they may be composed mostly of blood. Abundant tissue can be expected in the late proliferative and secretory phase, with pregnancy, after estrogen exposure, or when there is hyperplasia or neoplasia. Scant tissue is present when there is atrophy, prolonged bleeding, or recent menses, Asherman's syndrome, exposure to drugs that lead to an atrophic endometrium such as GnRH, danocrine, or prolonged progestins or oral contraceptives. Technical issues such as cervical stenosis or operator inexperience can also lead to a scant specimen. Endometrial biopsies are often problematic for the pathologist, who encounters problems including insufficient tissue, autolysis, a variety of tissue artifacts peculiar to endometrium, and inactive fragments of basalis, or lower uterine segment. Superimposed breakdown of tissue is often a confounding factor in interpreting DUB biopsies. Lack of history provided with the specimen is also problematic on many occasions. The history submitted with endometrial biopsies is often "DUB" or menometrorrhagia. These terms may be loosely applied, so they are not particularly helpful. In addition, an undisclosed history of exogenous hormonal administration currently or recently may add to diagnostic difficulty. DUB is usually anovulatory, but may be ovulatory as well. As such, endometrial patterns may be either non-secretory or secretory.²²

If estrogen is unopposed by progesterone for prolonged periods of time, the earliest indication histologically is disordered proliferation, and this is one of the commonest patterns seen in an adolescent with DUB. Here there is no increase in gland to stroma ratio, but some of the glands show cystic dilatation, and occasional outpouchings.²² This is not considered severe enough to be called a hyperplasia, and is not considered pre-neoplastic. With ongoing unopposed estrogenic stimulation, hyperplasia and carcinoma may develop, particularly in girls with PCO.

Dysmenorrhea

Dysmenorrhea is common in adolescents. It can be associated with lack of identifiable pelvic pathology, and is then termed primary, or can be associated with pelvic conditions such as endometriosis or pelvic inflammatory disease, in which case it is secondary. Primary dysmenorrhea can be attributed to the release of prostaglandins by the endometrium. While the prostaglandins lead to vasoconstriction and uterine contractions essential for passage of tissue and controlling of blood loss, this also leads to the cramps so many adolescents experience. NSAIDs are first line treatment for the problem.¹⁵

Congenital Anomalies of the Upper Genital Tract

Depending on whether there is total or partial obstruction, anomalies of the Müllerian system are potential causes of amenorrhea or abnormal bleeding. These may be divided into disorders of canalization, disorders of fusion, agenesis (dysgenesis, hypoplasia), and embryonic rests. During embryogenesis, the two Müllerian ducts fuse, and are destined to become the fallopian tubes, uterus, and upper two thirds of the vagina. These structures are met by upward extension of the urogenital sinus, which forms the lower vagina. After fusion, there is canalization of the vagina, and resorption of the longitudinal septum. Any failures of these three important steps in embryogenesis; fusion, canalization, or resorption, can lead to genital tract anomalies. Among the Müllerian anomalies that may be encountered are hymenal abnormalities: imperforate, microperforate and variants—vaginal agenesis: (Mayer-Rokitansky-Küster-Hauser syndrome), transverse vaginal septum (high, medium, or low), longitudinal septum (obstructive or nonobstructive)—cervical agenesis or hypoplasia, unicornuate uterus with or without rudimentary horn, which may have a communicating or noncommunicating endometrial cavity or no cavity—and fusion or resorption defects such as uterus didelphys, bicornuate uterus (complete or partial), septate uterus (complete or partial, one or two cervixes), and DES-related T-shaped uterus.

Müllerian anomalies may be associated with urinary tract anomalies, skeletal anomalies, inguinal hernia of adnexa, and occasionally congenital heart anomalies. A partially obstructing longitudinal septum is often associated with a uterus didelphys, and absent kidney on the side of the obstruction.²³

Segmental agenesis/hypoplasia presents with primary amenorrhea. If there is some communication, there may be abnormal menses due to slow prolonged flow of obstructed blood. Also, increased rates of endometriosis occur, related to obstruction. Total obstruction can present at birth with mucocolpos, or at menarche with hematocolpos, pain, urinary retention, bowel symptoms and primary amenorrhea. Fertility issues related to endometriosis can arise. If endometriosis does not interfere, there is no decreased conception with uterine anomalies, but patients are prone to abnormal fetal presentation, preterm labor, and cervical malfunction (see following section).

Surgical specimens received during corrective surgery for a Müllerian anomaly will not generally be diagnostically difficult. Of note, when an obstructing transverse vaginal septum is resected, the lower surface can be lined by squamous epithelium; however, the more cephalad portion may be lined by mucous secreting glandular epithelium.

Uterine Anomalies

Uterine anomalies are probably more common than suspected. In an analysis of multiple studies, Nahum²⁴ found that uterine anomalies were present in 1 out of 594 fertile women and 1 out of 29 infertile women. In this analysis, there was an overall prevalence of 1 in 201 women in the general population, of which 7% were arcuate, 34% septate, 39% bicornuate, 11% didelphic, 5% unicornuate, and 4% hypoplastic, aplastic, solid, or other forms. Most uterine anomalies are asymptomatic, with reproductive problems arising in only about 25%; hence they may never be diagnosed.²⁴ However, in addition to future fertility issues and potential pregnancy wastage, the issue for the adolescent may be amenorrhea if there is a total obstruction, or dysmenorrhea, menstrual irregularity, mass, and risk of endometriosis if there is partial obstruction. Absence of one of the Müllerian ducts leads to a unicornuate uterus.²⁵ Failure of fusion of the two ducts leads to varying degrees of duplication, from bicornuate uterus to uterus didelphys. Failure of resorption of the septum leads to a septate uterus, which can be distinguished externally from a bicornuate uterus by the lack of indentation in the fundus. Failure of canalization leads to transverse vaginal septum. Varying degrees of Müllerian agenesis or hypoplasia may also occur. Approximately 10% of individuals with uterine anomalies have associated genitourinary

anomalies as well, including fused pelvic kidney, pelvic kidney, absent or atrophic kidney, crossed renal ectopia, hydronephrosis, and collecting system duplication.²⁵ A variety of surgical procedures are available to correct uterine anomalies if necessary.

The Mayer-Rokitansky-Küster-Hauser syndrome consists of congenital absence of the uterus and vagina. A vaginal length satisfactory for intercourse is often achievable by nonsurgical dilatation, but some cases may come to surgical intervention.²⁶ Surgical corrections of genital tract anomalies should be performed by those with sufficient expertise to assure a successful outcome. There have been case reports of neoplasia arising in neovaginas.^{27,28}

Fallopian Tube Anomalies

Isolated fallopian tube anomalies are rare. Absence of a fallopian tube may be congenital, or related to torsion due to abnormally long mesosalpinx.²⁹ A case of torsion of a Fallopian tube with abnormal blood supply has been described.³⁰

Ovarian Anomalies

Ovarian anomalies are uncommon, and include absence, hypoplasia, accessory, and supernumerary ovaries. Neoplasms can arise in ectopic ovaries, with potentially unusual presentations.³¹ In addition, abnormal ovarian differentiation such as streak ovaries or ovotestis may be seen in some of the intersex conditions, and individuals with ovarian anomalies should have chromosomal analysis.

Upper Genital Tract Malignancies in Adolescents

Ovarian Malignancies

See previous section on adnexal masses.

Uterine Malignancies in the Adolescent

Uterine malignancies are rare in this age group. Uterine sarcomas, particularly carcinosarcomas, have been rarely reported.¹³ Endometrial adenocarcinoma and hyperplasia are exceedingly rare in the pediatric population, and are usually associated with morbid obesity, as seen in girls with PCO. Girls with Turner's Syndrome, if on unopposed estrogen, are also at risk. About half the cases of adolescents with endometrial carcinoma that are treated with high dose progestins will have reversal of the condition, with preservation of their fertility.³² As the underlying condition, ie PCO, is still present, long term follow-up is indicated.

Pregnancy-Related Conditions

Aside from the issues relating to teenage pregnancy, which are beyond the scope of this discussion, adolescents are also at risk for spontaneous abortions, and for tubal ectopic pregnancies, which must be considered in the differential of abdominal pain and abnormal bleeding in a sexually active adolescent.

Gestational trophoblastic disease is more frequent at both ends of the reproductive spectrum, and hence the diagnosis of hydatidiform mole and malignant gestational trophoblastic disease must be considered in a postpubertal adolescent with bleeding abnormalities and a positive B-hCG (human chorionic gonadotropin). A case of hydatidiform mole associated with hyperthyroidism has been reported in a 13-year-old, and hence gestational trophoblastic disease is in the differential of an adolescent with hyperthyroidism.³³

Hydatidiform moles are either complete or partial. Complete moles are more likely to present with uterine size greater than dates, while partial moles are more likely to present as missed abortions. Complete moles are diploid, with both components paternally derived, while partial moles are triploid with biparental contribution. There are histological differences between the two, and they may be distinguished from each other by p57^{kip} immunostaining as well.³⁴ While both types of moles are treated the same way, with evacuation followed by follow-up hCG levels, and possible chemotherapy for levels that plateau or rise, there is a significant difference in the risk of subsequent malignant gestational trophoblastic disease, which is about 5% after complete hydatidiform mole, but <5% after a partial mole.³⁵

Summary

Gynecologic conditions that affect the upper genital tract of children and adolescents are often unique to the age group. Conditions that can be seen in the adult woman may also be seen in the adolescent, and must be considered, particularly if the adolescent is sexually active. A familiarity with the range of conditions that can occur in these younger patients is critical to considering an appropriate differential diagnosis in a patient presenting with symptomatology.

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