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TESTICULAR CANCER

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INTRODUCTION

Testicular cancer is rare; it accounts for just 1% of all cancers in males. Despite this seemingly insignificant statistic, testicular cancer is a significant condition in men because in virtually all parts of the world it is the most common solid malignancy affecting young men.[1] Moreover, it is responsible for ten percent of all cancer deaths in men aged 15-35 years.

Based on 2003-2007 statistics, the National Cancer Institute calculated the age-adjusted incidence rate in the United States to have been 5.4 per 100,000 men per year.[2] The number of new cases in the United States is estimated to be 8400 per year, with annual deaths being approximately 380.[3]

Over the past quarter century, there has been a clear trend toward an increased incidence of testicular cancer in the majority of industrialized countries in North America, Europe, and Oceania.[4] In Canada, a 50% increase in the incidence rate of testicular cancer has been observed over the last 25 years.[5]

Mortality rates for testicular cancer are very low, with the five-year survival rate having increased from approximately 63% to more than 90% during the last 30 years.[6] This decline may be attributed to advances in treatment, such as platinum-based chemotherapy.

ETIOLOGY

Probably the best known risk factor for testicular cancer is cryptorchidism, (Figure 1) or undescended testis, which occurs in approximately 10% of cases.[7] A man diagnosed with undescended testis is four to five times more likely to be diagnosed with testicular cancer compared to a man with normally descended testes. Significantly, about one in ten men diagnosed with testicular cancer give a history of having had cryptorchidism.

The risk of testicular cancer is greater with bilateral cryptorchidism, and if the testes were located intra-abdominally rather in the inguinal region, the risk of cancer is even greater.

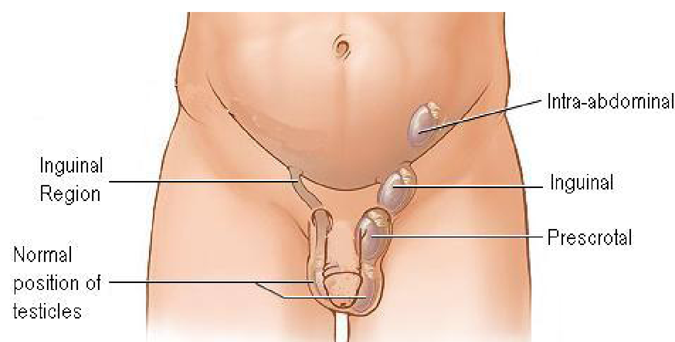

FIGURE 1. Cryptorchidism describes an undescended testicle that has not moved down into the scrotum.

In a study including 794 men and nine regions in England and Wales, the United Kingdom Testicular Cancer Study Group concluded that there was indeed an elimination of risk in men who underwent orchiopexy (surgery to mobilize the undescended testis and bring it down to be fixed in its rightful place within the scrotum) before the age of 10 years (Figure 2).[8] A 2007 study from Sweden identified 16,983 men in that country who underwent orchiopexy for undescended testis in the period between 1964 to 1999 and found that the increased risk of testicular cancer was almost halved if orchiopexy was performed before the boy reached the age of 13 years.[9]

Other studies done in Denmark demonstrated that subfertile men—especially those who had low sperm counts—had an increased risk of developing testicular cancer.[10] Men having low fertility were found to be twice as likely as normally fertile men to develop testicular cancer.

The associations demonstrated by all these research studies suggest that testicular cancers actually originate in fetal life. Studies also indicate that there is an appreciable genetic component in the etiology of testicular cancer, with a sixfold relative risk in men who have a first-degree relative diagnosed with cancer of the testis.

A definitely established major risk factor is prior testicular cancer. A man who has had cancer in one testis has a 25-fold increased risk of developing testicular cancer in the contralateral testis.

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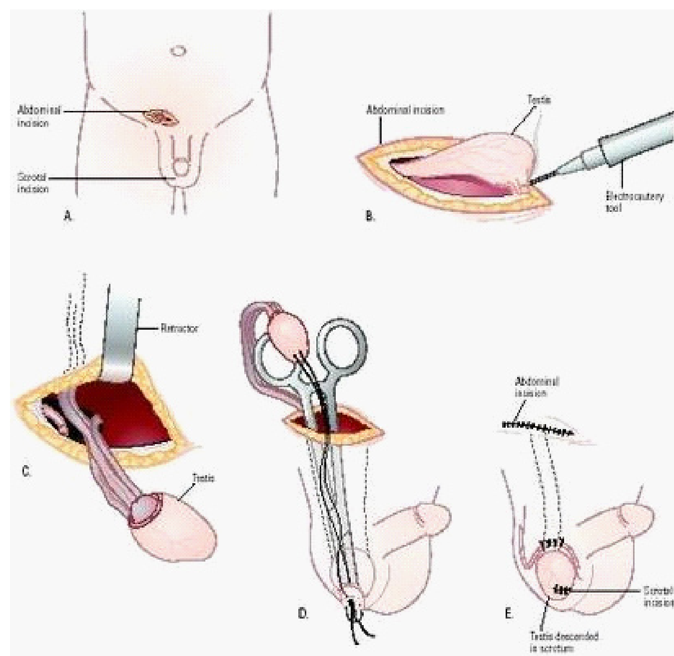

FIGURE 2. Orchiopexy is a surgical procedure to move an undescended testicle into the scrotum and permanently fix it there. Incisions are made in the abdomen at the site of the undescended testicle and in the scrotum (A). The testis is detached from surrounding tissues (B) and pulled out of the abdominal incision attached to the spermatic cord (C). The testis is then pulled down into the scrotum (D) and stitched into place (E). Source: Encyclopedia of Surgery

PATHOPHYSIOLOGY

The majority of testicular cancers arise from the germ cells of the testis and occur in two main forms:

* Seminomas—cancers that grow slowly and are sensitive to radiation therapy
* Nonseminomas—tumors that contain different cell types and grow more quickly than seminomas

Testicular cancer has a typical age distribution—a small peak in incidence around two years of age followed by low rates until round 15 years. After this the incidence climbs rapidly. The tumors seen during childhood and early adult life usually show germ cell histology, while the tumors seen in older men (after the age of 65 years) are mainly of the non-germ cell type, principally lymphomas.

PATHOLOGY

When given a specimen of a testicular cancer, the pathologist's first task is to identify the type of cell from which the tumor originates. Non-germ cell tumors account for 5 to 10% of all testicular cancers—four-fifths of these are lymphoma, and the remainder are rare tumors such as Sertoli cell tumors, interstitial (Leydig cell) tumors and paratesticular embryonal sarcomas. The other 90 to 95% are germ cell tumors, which for clinical purposes, as mentioned, above are classified into

* Seminomas
* Nonseminoma germ cell tumors (NSGCTs)

Differentiating these histologically is relatively easy. Approximately 40% of testicular cancers are seminomas, 35% are NSGCTs, and 15% are mixed seminomas and NSGCT—while the remaining 10% or less, as indicated above, constitute the non-germ cell tumors.

Seminomas on gross examination are irregularly nodular or lobulated, with the tumor compressing the normal testicular tissue. On section, they are usually firm and grayish white to yellow in color with occasional foci of hemorrhage.

The NSGCTs are made up of a collection of different germ cell tumors that often coexist within the single tumor. Embryonic carcinoma is composed of cells corresponding to undifferentiated early embryonic cells presenting as a soft irregular mass, poorly demarcated from the surrounding tissue and often containing areas of hemorrhage and irregular areas of necrosis. Differentiation into either yolk sac tumor, which produces high quantities of alpha fetoprotein (αFP), or choriocarcinoma, which produces high quantities of beta-human chorionic gonadotropin (βhCG), is common.

SPREAD OF TESTICULAR CANCER

Testicular tumors spread via the lymphatics and the bloodstream. A common misconception is that testicular cancers metastasize to the nodes in the groin. These inguinal lymph glands in fact drain only the scrotal skin—while the testes drain along four to eight efferent lymphatic channels accompanying the testicular arteries, passing from the testis through the inguinal canal and joining up to form major lymphatic channels that enter the retroperitoneal lymph nodes. Cancer cells from the testes therefore metastasize to the nodes around the aorta and inferior vena cava (paraaortic, preaortic, paracaval, and retrocaval nodes).

The first site of hematogenous spread is usually the lungs. Multiple small metastases in a peripheral location are typical features of NSGCT, but in seminoma, pulmonary metastases tend to be larger. Other common sites of disseminated disease include the brain, bone, and liver—although virtually any organ may be involved by secondary spread.

TUMOR MARKERS

Human chorionic gonadotropin (hCG) is a glycoprotein made up of α and β subunits that is normally secreted by the placenta during pregnancy. The β subunit is produced by syncytiotrophoblastic cells. Elevated values are sometimes seen in patients with pure seminoma, although they are more common in those with nonseminomas. Prognosis is generally poorer if the βhCG level is greater than 10,000 ng/mL (50,000 IU/L).

Alpha fetoprotein (αFP) is a glycoprotein produced in the liver, gastrointestinal tract, and yolk sac of the fetus. Production is usually restricted to embryonal carcinoma and its derivatives (such as yolk sac tumor) although elevated levels of αFP can occur in some patients who have regenerating hepatocellular injury.

This marker can be used for prognosticating in patients with testicular cancer. Levels less than 100 ng/mL suggest a good prognosis, while levels over 10,000 ng/mL indicate that the prognosis is poor.

CLINICAL PRESENTATION

Typically, testicular cancer presents as a painless hard swelling of the testis. In a few instances (between 10 to 20%), the patient may complain of pain or discomfort in the testis—often the result of a bleed into that organ. Unfortunately, symptoms of pain and local tenderness can act as a red herring resulting in a mistaken diagnosis of epididymo-orchitis, which can delay the initiation of appropriate therapy.

If a patient presents with a complaint of painless swelling of the testis, it is prudent at that consultation to examine the patient for evidence of cancer spread. Testicular cancer typically spreads along the lymphatic drainage from these organs to the para-aortic lymph nodes—enlargement of which can cause abdominal or back discomfort. Very large glands could even result in obstruction of a ureter.

Other symptoms associated with metastases include hemoptysis and dyspnoea due to lung metastases, and gynecomastia and galactorrhea resulting from exceptionally high levels of beta-human chorionic gonadotropin.

Diagnosis is based on a careful clinical examination. Not only should both testes be examined, but so should the abdomen and lungs. Following clinical examination, the next step is to arrange an ultrasound scan of the testes and blood tests to measure the level of serum tumor markers.

DIFFERENTIAL DIAGNOSIS

A patient with a hard, painless testicular swelling could be having, if not a testicular cancer, a testis affected by syphilis or tuberculosis. Both conditions are relatively uncommon in the United States, so a hard painless testicular swelling should be considered malignant until proved otherwise.

The differential diagnosis of a mass within the scrotum includes the following:

- Epidermoid cysts of the testis are spherical, sharply demarcated lesions that can be clearly distinguished from solid tumors on ultrasonography.
- Testicular abscesses are almost always associated with fever, pain, marked epididymitis, and redness and swelling of the scrotum.
- A testicular hematoma is usually associated with a history of trauma to the scrotum. It is important when dealing with a scrotal hematoma to make sure that the lesion resolves completely, because incidental trauma is often the presenting symptom for an asymptomatic testicular mass.

Tumor markers—βhCG and αFP—are not always elevated, but, if they are present in increased quantities, this provides a useful marker of recurrent disease. Levels should normalize after orchidectomy, but if they persist in remaining raised, this usually indicates the need for further therapy such as chemotherapy.

If metastases are present, it is vital that the disease is correctly staged. Because enlarged retroperitoneal nodes are not clinically palpable, obtaining a CT scan of the abdomen and chest is essential.

Patients suspected to have a testicular cancer should be referred to a urologist for further assessment. This assessment will usually involve surgical exploration through an inguinal incision, which will allow a specimen to be taken for histology and a decision to be made as to whether orchidectomy is indicated. Prior to undertaking surgery, the question of whether the patient would like to have a testicular prosthesis inserted (if histology makes orchidectomy mandatory) should be discussed.

IMAGING IN THE DIAGNOSIS OF TESTICULAR CANCER

The standard imaging technique utilized to identify testicular cancer is ultrasonography, which has a high sensitivity. Ultrasonography is, however, more specific in the presence of a palpable mass, and so must always be combined with physical examination to achieve the best specificity.

Intratesticular lesions that are not palpable and are seen as incidental findings on ultrasound scanning, even if larger than 1 cm, are unlikely to be malignant. Ultrasonographic screening for testicular cancer is not currently recommended, because the disease does not meet the criteria for an acceptable screening program wherein early treatment must have a success rate superior to that of the treatment initiated when the patient is clinically symptomatic.

Other imaging techniques play a pivotal role in the management of testicular tumors by establishing the presence and extent of metastatic disease at the time of diagnosis, by assessing response to treatment in those with demonstrable metastatic disease, and by evaluating suitability for resection of residual masses. It is also valuable for detecting sites of relapse in patients with early disease who are being managed by surveillance and in those who have previously been treated.[11]

The most important imaging techniques utilized in ascertaining the presence of metastases from testicular tumors are computed tomography (CT) and plain chest radiographs; magnetic resonance imaging (MRI) and ultrasound also have a place in certain clinical situations.

MANAGEMENT OF PRIMARY TESTICULAR TUMOR

Before commencing treatment for primary testicular cancer, as mentioned above, it is imperative that the tumor be clinically staged.

Stage I tumor is defined as a tumor confined to the testis, while stage II is a tumor that has spread to the retroperitoneal lymph nodes. Stage II tumors are often subdivided into

* IIa—nodes less than 1 cm in diameter
* IIb—nodes between 1 and 5 cm in diameter
* IIc—nodes greater than 5 cm in diameter

Stage III disease indicates that the cancer has spread beyond the retroperitoneal lymph nodes.

STAGE I NONSEMINOMA GERM CELL TUMORS (NSGCT)

A Clinical stage I NSGCT is, by definition, a germ cell tumor that is confined to the testis without evidence of metastatic disease. If serum levels of αFP and βhCG are normal and CT scans of the abdomen, pelvis, and chest following inguinal orchidectomy are normal, it can be assumed that the disease has not metastasized.

Approximately 70% of clinical stage I patients have a good prognosis and do not relapse. Among those who do relapse, in the vast majority tumor recurrence first manifests itself in the retroperitoneal lymph nodes.

A large retrospective trial of clinical stage I NSGCT patients by the UK Medical Research Council determined that retroperitoneal recurrence could be predicted by[12]

* The presence of vascular and or lymphatic invasion by the primary tumor
* The presence of embryonal carcinoma
* The absence of yolk sac elements

This high-risk group had approximately 50% chance of recurrence, compared to a risk of 2% in the low-risk group.

The cure rate in clinical stage I NSGCT should approach 100%. Treatment needs to be individualized and should be undertaken in a center that has experience in treating testicular cancer. Primary retroperitoneal lymph node dissection is the therapeutic option usually employed in the United States. However, in other countries (for example, Australia) the approach that is currently in vogue is surveillance. The rationale for the latter approach is to prevent over-treatment of patients with stage I NSGCT, because about 70% of such patients who undergo retroperitoneal lymph node dissection have no evidence of metastases in these nodes—and so are subject to the morbidity of a major operation without any benefits.

If patients on surveillance protocols suffer a recurrence, most do so within the first two years. Surveillance usually requires regular physical examination together with CT scans of the abdomen, chest, and pelvis every one to two months for the first year. One drawback to adopting a surveillance approach is the uncertainty associated with this approach, which can have a psychological impact on young patients. The key requirement for success with surveillance is patient compliance, which requires that these patients understand the need to conscientiously adhere to the follow-up protocol and remain motivated to comply with therapeutic advice.

Primary chemotherapy avoids the morbidity of invasive surgical procedures and is useful for patients who are at high risk of recurrence. Standard regimes usually utilize two cycles of bleomycin, etoposide, and cisplatin. This regime is associated with low morbidity, but it is important to note that this may still over treat half the patient population, because only 50% of the high-risk patients are destined to relapse. Nonetheless, recent studies have shown that if all patients are treated with a regime of chemotherapy, the risk of disease relapse decreases from 50% to less than 10%.

STAGE I SEMINOMA

As with the stage I NSGCTs, about a third of patients with clinical stage I seminomas have occult retroperitoneal disease at the time of diagnosis. However, the characteristic of seminomas is that as a rule they are exquisitely radio-sensitive and can usually be cured with adjuvant radiation therapy.

Disease control is obtained in up to 98% of patients by using moderate-dose radiation. Because the lymphatic drainage of the testes goes directly to the paraaortic lymph nodes, most radiation oncologists focus radiation on these nodes. There has been a recent tendency to omit radiation to the pelvic lymph nodes because metastases in these nodes are relatively uncommon.

Five-year survival rates for stage I seminoma are between 95 and 100% in the majority of studies. Patients who are destined to relapse usually do so within the first 18 months after being diagnosed with primary cancer.

Although most men will recover spermatogenesis in time, patients recommended radiotherapy should always be offered the option of sperm storage if they wish to optimize their chances of producing offspring.

While for many years adjuvant radiotherapy has been the standard therapy for stage I seminomas, there has been a recent interest in therapy with a single course of carboplatin that deals with microscopic disease in the lymph nodes.

Surveillance is also of value for those patients who do not have any evidence of metastatc disease on CT scanning. Studies have demonstrated an actuarial five-year relapse-free rate of 82% for surveillance regimes. The important predictors of relapse are

* Tumors greater than 4 cm diameter in size
* Invasion of the rete testis by cancer cells

About 20% of patients develop evidence of recurrent disease and are treated with chemotherapy at the time the recurrence is discovered. This treatment gives excellent cancer-specific five-year survival rates of 97 to 100%.

MANAGEMENT OF METASTATIC TESTICULAR CANCER

The advent of the chemotherapeutic agent cisplatin revolutionized the treatment of metastatic testicular cancer. Today, testicular cancer has become the epitome of a curable neoplasm. While in the early 1970s metastatic testicular cancer was associated with only 5% survival, with today's chemotherapy and surgery techniques, 80% of patients will survive their disease.[13]

In a recent Australian study of 633 patients with testicular cancer treated during the six-year period 1988 to 1993,[14] the five-year relative survival proportion for all men with testicular cancer was found to be 95%. Taking all these patients (stages I, II, and III) into account, the relative survival at five years for patients with seminoma was 99%, while, for patients with nonseminoma, relative survival at five years was 91%. The overall five-year survival for all patients was:

* Stage I: 99%
* Stage II: 96%
* Stage III: 73%

The younger the patient was at the time of diagnosis and treatment, the better was the prognosis. Some of the aspects of management peculiar to this malignancy include

- A strong reliance on serum tumor markers for management
- The use of surveillance following initial therapy in patients with stage I disease, with cure by salvage therapy available for those who relapse
- The expectation of cure of metastatic disease with a brief, intensive course of chemotherapy
- The use of surgery to resect residual masses that develop at metastatic sites after chemotherapy

Currently, the most efficacious drug regime uses four cycles of bleomycin and etoposide (BEP) in combination. A relatively recent technique, reinfusing autologous hemopoietic stem cells, allows much higher doses of myelosuppressive radiation to be used—and this holds great promise for the future.

Surgical resection of all residual cancer is recommended in patients with NSCGT who have normal tumor markers following chemotherapy. Nonetheless, in a large retrospective research study, 25% of patients experienced disease progression following resection of residual masses.

Because the majority of relapses in patients with low and high stage germ cell tumors of the testis occur within two years of treatment, in the past a two-year disease-free period was believed to represent complete cure. Unfortunately, the incidence of late relapse for germ cell tumors appears to be increasing, with the most common site for recurrent tumor being the retroperitoneum. Late relapse of germ cell tumors can occur at any time after treatment. Many patients are asymptomatic and present with either elevated tumor markers or with an incidental abnormality on radiological examination. αFP is usually the most common tumor marker found in patients who suffer late relapse of cancer.

The overall prognosis for patients who have late relapse of germ cell tumors is poor, the five-year survival rate being around 30%. As late relapse can occur at any time, patients should be followed up regularly (at least once a year) for the duration of their lives.

SUMMARY

Testicular cancer is the most common cancer in men aged 20–40 years, and accounts for approximately 1% of all cancers in men. The majority of tumors are derived from testicular germ cells—seminomas and nonseminoma germ cell testicular (NSGCT) cancer. In more than 70% of patients that have testicular cancer today, the diagnosis is made early (when the disease is confined to the testis—stage I disease). Testicular tumors show excellent cure rates of over 95%, mainly due to their extreme sensitivity to radiotherapy and chemotherapy. With advances in the management of the disease over the past 25 years,

most men with metastatic testicular cancer can be cured with combination chemotherapy.

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TESTICULAR CANCER POST TEST

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1. Risk factors associated with testicular cancer include all the following except
- a. undescended testis.
- b. having a first-degree relative with the disease.
- c. excessive alcohol consumption.
- d. low sperm count.

2 Which of the following is not true about testicular cancer?
- a. Testicular cancer is increasing in incidence in industrialized countries.
- b. Testicular cancer is the most common form of cancer affecting men aged 15-35 years.
- c. Testicular cancer is more common in men born with bilateral cryptorchidism.
- d. Testicular cancer has been proven to be more common in men of Southeast Asian ancestry compared to those of African-American ancestry.

3. A boy found to have an undescended testis at the age of one year
- a. has a lesser risk of testicular cancer compared to a boy with normally-descended testes.
- b. will reduce his risk of testicular cancer if orchiopexy is done before he is ten years old.
- c. will have the same risk of testicular cancer whether he undergoes orchiopexy or not.
- d. has a greater than average risk of developing prostate cancer.

4. What is the first step in the investigation when a hard painless testicular lump is discovered during physical examination?
- a. Ultrasound scan
- b. Percutaneous needle biopsy of the testis under local anesthesia
- c. CT scan of the genitalia
- d. MRI scan of the chest, abdomen, and pelvis

5. If testicular cancer is suspected after physical evaluation, which are the most useful imaging investigations?
- a. Ultrasonography of the testis plus CT scanning of the pelvis
- b. CT scanning of chest, abdomen, and pelvis plus x-ray of abdomen to include kidneys, ureters, and bladder
- c. X-ray of abdomen to include kidneys, ureters, and bladder in addition to lymphangiography of the inguinal lymph nodes
- d. Ultrasonography of the testis plus lymphangiography of the inguinal lymph nodes

6. Which of the following is a correct statement about testicular seminomas?
- a. Testicular seminomas are resistant to radio therapy.
- b. Testicular seminomas are categorized as germ cell tumors.

c. Testicular seminomas constitute 90% of testicular cancers.
d. Testicular seminomas have a relative overall five-year survival of 55%.

7. Which the following statements are true regarding ultrasound scanning of the testis?
a. Ultrasonographic screening of asymptomatic patients to detect testicular cancer is recommended.
b. Nonpalpable intratesticular lesions incidentally detected on ultrasound scanning are likely to be malignant.
c. Ultrasonography has a low degree of sensitivity in identifying testicular cancer.
d. Ultrasonographic screening is the first choice of imaging techniques in investigating a painless swelling of the testis.

8. Which of the following is not a true statement regarding testicular tumor staging?
a. Stage I tumors are defined as those confined to the testis.
b. Stage II tumors are defined as those that have spread to the retroperitoneal lymph nodes.
c. Stage II tumors include those that have spread to the inguinal lymph nodes.
d. Stage III tumors include those that have metastasized to the lung.

9. Which of the following tumor markers have proven useful in the management of testicular cancer?
a. Alpha fetoprotein (αFP), and carcino embryonic antigen (CEA)
b. Alpha fetoprotein (αFP), and beta-human chorionic gonadotropin (βhCG)
c. Carcino embryonic antigen (CEA), and cancer antigen 125 (CA 125)
d. Cancer antigen 125 (CA 125), and beta-human chorionic gonadotropin (βhCG)

10. The lymphatic drainage of the testis goes
a. directly to the inguinal nodes on the same side.
b. bilaterally, together with the lymphatics of the scrotal skin, to the inguinal nodes.
c. principally to the para-aortic lymph nodes.
d. principally to the liver.

11. A large multicenter study from the UK (Lancet 1985) showed that retroperitoneal recurrence could be predicted by all of the following except the
a. absence of yolk sac elements.
b. presence of inguinal metastases at the time of diagnosis.
c. presence of vascular or lymphatic invasion by the primary tumor.
d. presence of embryonal carcinoma.

12. Patients that relapse after initial therapy
a. have a poor prognosis, with an average five-year survival of less than 20%.
b. usually present with hepatic metastases.
c. usually do so many years after diagnosis and treatment.
d. commonly develop recurrent tumor in the retroperitoneal region.

13. Chemotherapeutic agents that are commonly used to treat testicular cancer include all of the following EXCEPT
a. etoposide.
b. docatexel.
c. cisplatin.
d. bleomycin.

14. In the management of testicular cancer, imaging techniques are useful to accomplish all of the following EXCEPT
a. establishing the grade and tissue type of the tumor.
b. establishing the presence and extent of metastatic disease at the time of diagnosis.
c. assessing the response to treatment in those with demonstrable metastatic disease.
d. evaluating whether residual masses left after radiotherapy can be resected.

15. Regarding primary retroperitoneal lymph node dissection, all of the following are true EXCEPT
a. it should only be performed for stage II tumors.
b. it requires the patient to undergo monthly physical examination and CT scanning following surgery.
c. it may over-treat the majority of patients with stage I NSGCT.
d. it is considered a therapeutic option in the United States.

16. Regarding primary chemotherapy for testicular cancer, all of the following are true EXCEPT
a. it avoids the morbidity of an invasive surgical procedure.
b. it is useful for patients who are at low risk of recurrence.
c. it is usually done using seven weekly cycles of vincristine, bleomycin, and cyclophosphamide.
d. it should be initiated after a patient has shown non-compliance using the surveillance approach.

17. Regarding stage I seminomas,
a. these tumors respond poorly to radiotherapy.
b. metastases are often found in the pelvic lymph nodes.
c. occult retroperitoneal disease is present at the time of diagnosis in about one-third of patients.
d. five-year survival rates are about 50% in the majority of studies.

18. Late relapse of testicular germ cell tumors
a. is usually asymptomatic, with most patients presenting with elevated tumor markers or an incidental abnormality detected on imaging studies.
b. usually presents as a solitary metastasis in the lung.
c. has a five-year survival rate of about 90%.
d. appears to be decreasing in incidence over the past ten years.

19. Which of the following statements is true with regard to testicular cancer?

a. Blood tests for tumor markers are of no practical use in management.
b. Surveillance following initial therapy, with cure by salvage therapy available for those who relapse, is an acceptable treatment policy for patients with Stage I disease.
c. The older the patient at the time of diagnosis, the better the prognosis.
d. Testicular cancer accounts for approximately 10% of cancers in men.

20. Which of the following statements is true?

a. Compared to fifty years ago, the incidence of testicular cancer has decreased.
b. Compared to fifty years ago, testicular cancer has increased in the United States in contrast to Canada and Mexico.
c. Compared to fifty years ago, the mortality rate has doubled in young men aged 15–35 years.
d. Compared to fifty years ago, platinum-based chemotherapy has become a feasible and efficacious treatment option.

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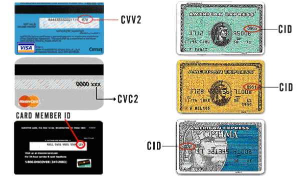

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Article Title: Testicular Cancer

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2. a b c d	7. a b c d	12. a b c d	17. a b c d
3. a b c d	8. a b c d	13. a b c d	18. a b c d
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