followed with careful annual mammography and semiannual physical examinations. Additional molecular analysis of these lesions may make it possible to discriminate between patients who are at risk of further progression and require additional therapy and those in whom simple follow-up is adequate.

### MALE BREAST CANCER

Breast cancer is about 1/150th as frequent in men as in women; 1720 men developed breast cancer in 2006. It usually presents as a unilateral lump in the breast and is frequently not diagnosed promptly. Given the small amount of soft tissue and the unexpected nature of the problem, locally advanced presentations are somewhat more common. When male breast cancer is matched to female breast cancer by age and stage, its overall prognosis is identical. Although gynecomastia may initially be unilateral or asymmetric, any unilateral mass in a man older than age 40 years should receive a careful workup including biopsy. On the other hand, bilateral symmetric breast development rarely represents breast cancer and is almost invariably due to endocrine disease or a drug effect. It should be kept in mind, nevertheless, that the risk of cancer is much greater in men with gynecomastia; in such men, gross asymmetry of the breasts should arouse suspicion of cancer. Male breast cancer is best managed by mastectomy and axillary lymph node dissection or SLNB. Patients with locally advanced disease or positive nodes should also be treated with irradiation. Approximately 90% of male breast cancers contain estrogen receptors, and approximately 60% of cases with metastatic disease respond to endocrine therapy. No randomized studies have evaluated adjuvant therapy for male breast cancer. Two historic experiences suggest that the disease responds well to adjuvant systemic therapy, and, if not medically contraindicated, the same criteria for the use of adjuvant therapy in women should be applied to men.

The sites of relapse and spectrum of response to chemotherapeutic drugs are virtually identical for breast cancers in either sex.

### FOLLOW-UP OF BREAST CANCER PATIENTS

Despite the availability of sophisticated and expensive imaging techniques and a wide range of serum tumor marker tests, survival is not influenced by early diagnosis of relapse. Surveillance guidelines are given in Table 108-5. Despite pressure from patients and their families, routine computed tomography scans (or other imaging) are not recommended.

**TABLE 108-5 BREAST CANCER SURVEILLANCE GUIDELINES**

| Test | Frequency |
|---|---|
| **Recommended** | |
| History; eliciting symptoms; physical examination | q3–6 months × 3 years; q6–12 months × 2 years; then annually |
| Breast self-examination | Monthly |
| Mammography | Annually |
| Pelvic examination | Annually (particularly for patients on SERMs) |
| Patient education about symptoms of recurrence | Ongoing |
| Coordination of care | Ongoing |
| **Not Recommended** | |
| Complete blood count | |
| Serum chemistry studies | |
| Chest radiographs | |
| Bone scans | |
| Ultrasound examination of the liver | |
| Computed tomography of chest, abdomen, or pelvis | |
| Tumor markers CA 15-3, CA 27-29, CEA | |

***Abbreviations:*** CEA, carcinoembryonic antigen; SERM, selective estrogen receptor modulator.

***Source:*** Recommended Breast Cancer Surveillance Guidelines, ASCO Education Book, Fall, 1997.



# 109 Upper Gastrointestinal Tract Cancers

Robert J. Mayer

Upper gastrointestinal cancers include malignancies arising in the esophagus, stomach, and small intestine.

## ESOPHAGEAL CANCER

### INCIDENCE AND ETIOLOGY

Cancer of the esophagus is an increasingly common and extremely lethal malignancy. The diagnosis was made in 18,170 Americans in 2014 and led to 15,450 deaths. Almost all esophageal cancers are either squamous cell carcinomas or adenocarcinomas; the two histologic subtypes have a similar clinical presentation but different causative factors.

Worldwide, squamous cell carcinoma is the more common cell type, having an incidence that rises strikingly in association with geographic location. It occurs frequently within a region extending from the southern shore of the Caspian Sea on the west to northern China on the east, encompassing parts of Iran, central Asia, Afghanistan, Siberia, and Mongolia. Familial increased risk has been observed in regions with high incidence, although gene associations are not yet defined. High-incidence "pockets" of the disease are also present in such disparate locations as Finland, Iceland, Curaçao, southeastern Africa, and northwestern France. In North America and western Europe, the disease is more common in blacks than whites and in males than females; it appears most often after age 50 and seems to be associated with a lower socioeconomic status. Such cancers generally arise in the cervical and thoracic portions of the esophagus.

A variety of causative factors have been implicated in the development of squamous cell cancers of the esophagus (Table 109-1). In the United States, the etiology of such cancers is primarily related to excess alcohol consumption and/or cigarette smoking. The relative risk increases with the amount of tobacco smoked or alcohol consumed, with these factors acting synergistically. The consumption of whiskey is linked to a higher incidence than the consumption of wine or beer. Squamous cell esophageal carcinoma has also been associated with the ingestion of nitrates, smoked opiates, and fungal toxins in pickled vegetables, as well as mucosal damage caused by such physical insults as long-term exposure to extremely hot tea, the ingestion of lye, radiation-induced strictures, and chronic achalasia. The presence of an esophageal web in association with glossitis and iron deficiency (i.e., Plummer-Vinson or Paterson-Kelly syndrome) and congenital

**TABLE 109-1 SOME ETIOLOGIC FACTORS ASSOCIATED WITH SQUAMOUS CELL CANCER OF THE ESOPHAGUS**

| |
|---|
| Excess alcohol consumption |
| Cigarette smoking |
| Other ingested carcinogens |
| Nitrates (converted to nitrites) |
| Smoked opiates |
| Fungal toxins in pickled vegetables |
| Mucosal damage from physical agents |
| Hot tea |
| Lye ingestion |
| Radiation-induced strictures |
| Chronic achalasia |
| Host susceptibility |
| Esophageal web with glossitis and iron deficiency (i.e., Plummer-Vinson or Paterson-Kelly syndrome) |
| Congenital hyperkeratosis and pitting of the palms and soles (i.e., tylosis palmaris et plantaris) |
| ? Dietary deficiencies of selenium, molybdenum, zinc, and vitamin A |