Methods

SEER*stat version 7.0.4 was accessed to identify trends in esophageal cancer from 1975-2008.1 For incidence and mortality statistics the following 9 SEER registries were used: San Francisco-Oakland, Connecticut, Detroit, Hawaii, Iowa, New Mexico, Seattle, Utah and Atlanta. This represents approximately 10% of the US population. Rates and figures 1-5 are age-adjusted to the 2000 U.S. population.

Histology definitions - ICD-O-3 histology codes:

- Squamous cell carcinoma = 8050 - 8089
- Adenocarcinoma = 8140 - 8389

Subsite definitions:

- Upper Esophagus C15.0 (cervical esophagus)
- Middle Esophagus C15.1 (thoracic esophagus)
- Lower Esophagus C15.2 (abdominal esophagus)
- Middle third of esophagus C15.4
- Lower third of esophagus C15.5

Chi-square tests were performed on rate ratios for blacks and whites in 11-year increments. The ‘other race’ category was excluded from analysis since there was a small number of these cases in this population from 1975-2008.

Results

From 1975 to 2008 there has been a reversal in frequency of esophageal squamous cell carcinoma (ESSC) and esophageal adenocarcinoma (EAC) when considering all races (Figure 1). The increase of EAC has increased nearly 6-fold during this time, a rate greater than that of any other cancer in the United States.1 This increase is seen almost exclusively in the white male population (Figure 2). In 1975, ESSC was dominant in the male black population. The marked increase in adenocarcinoma in white males makes these incidence rates nearly the same (Figure 2 and 3). The increase in EAC in white males is almost exclusively located in the lower esophagus (figure 4).

Comparing the periods 1975-1985 to 1986-1996 and 1986-1996 to 1997-2007 there was an increase in the incidence of EAC in white males (p<0.001 for both) and in white females (p<0.001 for both). Incidence of EAC in black males and black females did not see an increase when comparing 75-85 to 86-96 (p=0.15 and p=0.068 respectively) but did see an increase when comparing 86-96 to 97-07 (p=0.0009 and p=0.022 respectively). Figure 5a and 5b demonstrate the rate changes over these time periods.

Obesity has also been associated with an increased risk of EAC. It is thought that central obesity may increase intragastric esophageal pressure gradient, thereby facilitating gastroesophageal reflux disease (GERD).3 The increase in incidence of lower EAC for white males and females corresponds to increased ambulatory care visits and hospital discharge rates for GERD (Figure 6). Normally the esophagus is lined with stratified epithelium. GERD causes stomach acid to back up into the lower esophagus, causing chronic irritation. GERD is a major predisposing factor for Barrett's esophagus. Barrett's esophagus is characterized by metaplasia of the distal esophagus resulting in columnar epithelium.3 EAC subsequently develops in the columnar epithelium. The overall prevalence of Barrett's is low in the general population making it unfavorable for routine screening.3 Interestingly, studies have shown there is no difference in frequency of GERD symptoms between white and black populations2 (Table 1). However, there is a striking difference in rates of Barrett's esophagus and EAC.4-5

Conclusions

Over the past 30 years, rates of EA in white males are increasing faster than any other cancer. Rates of GERD are similar between white males and black males; however rates of Barrett's esophagus and EA are markedly different, indicating an environmental or genetic difference exists. Further investigation and clinical studies of these differences will help to better understand esophageal cancer, identify its risk factors, and decrease mortality.

References


