

Impact of Barrett's Esophagus on Inpatient Outcomes in Patients Hospitalized with Atrial Fibrillation

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Introduction

Barrett's esophagus (BE) is a metaplastic change of the distal esophageal lining resulting from chronic gastroesophageal reflux disease (GERD). Previous studies have suggested that GERD plays a pivotal role in triggering atrial fibrillation (Afib). This study aimed to exam the impact of BE on the outcomes of patients hospitalized with Afib.

Methods

The National Inpatient Sample (NIS) database was used to identify patients admitted for Afib with BE in 2010- 2014. The primary outcomes were hospital mortality, length of stay, disposition status and total hospital charges. Secondary outcomes measured include acute stroke/transient ischemic attack (TIA) and acute heart failure.

Results

11,953 patients were admitted for Afib with BE. The average age of patients having Afib with BE was 72.8 years old, 60.8% were male and 87.1% were Caucasian. Compared to Afib patients without BE, Afib with BE patients had a greater prevalence of concomitant dyslipidemia, aortic atherosclerosis, prior PCI, tobacco use, chronic liver disease (CLD) and chronic pulmonary disease (CPD) ($p < 0.05$). There was no difference in the Elixhauser comorbidity index score, CHA2DS2-VASC Score between the two groups. After adjusting for confounders, patients with Afib and BE had less incidence of acute heart failure (aOR 0.72, $p < 0.001$, 95% CI 0.59-0.87), less hospital mortality (aOR 0.63, $p < 0.01$, 95% CI 0.46-0.87), and were more likely to have routine disposition (aOR 1.30, $p < 0.0001$, 95% CI 1.23-1.38). No significant difference in acute stroke/TIA events, length of stay, and total hospital charges were seen between the two groups.

Discussion

Despite patients with Afib and BE having greater prevalence of concomitant dyslipidemia, aortic atherosclerosis, prior PCI, tobacco use, CLD and CPD, hospitalized patients develop less acute stroke/TIA and acute heart failure leading to favorable hospital outcomes. This can suggest that the autonomic activity and local inflammatory responses seen with GERD are hindered by these metaplastic changes in BE.