Background Esophageal cancer is the sixth leading cause of cancer-related mortality worldwide. The risk factors are variable and are based on ethnicity, geographic location and pathological type. The well-established factors include alcohol, tobacco, dietary factors, nutritional deficiencies, environmental carcinogen exposure and chronic irritation of the esophagus. Although Plummer Vinson syndrome (PVS) and Lichen planus (LP) are known independent risk factors, they have never been reported together to cause esophageal squamous cell cancer (ESCC). Hence this association was studied in our cohort of PVS.

Methods We reviewed patients with ESCC and PVS from 2013 to 2020 to study their demographic and clinico-pathological characteristics. All these patients underwent dermatological examination for the presence of lichen planus.

Results A total of 170 patients were diagnosed with esophageal webs during this period. Nine patients with PVS were diagnosed with ESCC. Six patients had cancer along with webs at the time of diagnosis while three developed ESCC during follow up after endoscopic dilation of webs. There was a female preponderance (Male: Female=1:8) with a mean age of 50 years. ESCC was located in the upper esophagus in two, mid esophagus in three, and lower esophagus in four patients. There was no history of alcohol consumption or tobacco use in any of the patients. Histologically, ESCC was well-differentiated in one and moderately differentiated in 8 patients. Oral LP was observed in four patients, genital LP in one patient, oral and genital LP in one patient. Histological evidence of esophageal lichen planus was observed in one out of four patients who underwent mucosal biopsies.

Conclusions ESCC occurred in 5.3% of patients with PVS, more than half of whom had associated oral lichen planus. The coexistence of PVS and mucosal LP can increase the predisposition to ESCC, especially in women without conventional risk factors. However, this association needs to be proven in larger prospective studies so as to develop surveillance strategies in regions where PVS, LP and ESCC are highly prevalent.
Clinical manifestation and bacteria of secondary sepsis from hepatic disease and cholestasis

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Background Biliary stasis and increased intraductal pressure are central to the pathogenesis of acute cholangitis. Biliary stasis inhibits the continuous flushing activity of bile and the bacteriostatic effect of bile salts, which help maintain bile sterility. Elevated intraductal pressure favors translocation of bacteria and toxins out of the ducts and into the systemic circulation, which can result in sepsis. Sepsis is among the most common causes of mortality for hospitalized patients worldwide, and its incidence is steadily increasing.

Aims To characterize clinical manifestation and identify bacteria of secondary sepsis from hepatic disease and cholestasis.

Methods We conducted a prospective study of 38 patients with secondary sepsis from hepatic disease and cholestasis at the 108 Military Central Hospital from January 2018 to August 2019. Contributions of the history, physical examination, laboratory investigation and subclinical in making medical diagnoses. PCR-based Sepsis@Quick test is for identification of sepsis-causative pathogens.

Results On examination: fever (100%); abdominal pain (39.5%) and jaundice (100%). Laboratory investigation: White blood cell (16.98 ± 8.46 G/L); Neutrophil (85.3 ± 10.82%); Procalcitonin (21.18 ± 27.16 ng/ml). Procalcitonin values demonstrated a statistically significant and directly proportionate relationship to severity of sepsis. Secondary sepsis from cholestasis: biliary tract cancer 28.9%; gallstone 28.9%; pancreatic cancer 10.5%. Secondary sepsis from hepatic diseases: hepatocellular carcinoma 13.2%; cirrhosis 10.5%; liver abscess 7.9%. Sepsis-causative pathogens: Escherichia coli 55.3%; Klebsiella pneumoniae 31.6%; Citrobacter koseri 2.6%; Strep-tococcus anginosus 2.6%; Enterococcus faecalis 2.6%; Citrobacter freundii 2.6%; Klebsiella oxytoca 2.6%; Pseudomonas aeruginosa 2.6%.

Conclusions Etiology of secondary sepsis from hepatic disease and cholestasis the most is Escherichia coli, second is Klebsiella pneumoniae, of these we find Escherichia coli most commonly in gallstones, Klebsiella pneumonia most commonly in cholangiocarcinoma.