Comorbidities such as diabetes (45.55% vs 25.95%; compared to African Americans (12.79% vs 13.49%) and Hispanics (11.25% vs 7.97%); older (mean age 58.72 years vs 55.02 years; IBD who also had a co-diagnosis of VTE. Compared with non-obese controls, obese patients were increased risk of venous thromboembolism (VTE). Limited data exists related to the e

RESULTS: During the fi

FIGURE 2. Forest Plot showing overall clinical remission rate of Tococitinib.

INTRODUCTION: Obesity and inflammatory bowel disease (IBD) have been associated with an increased risk of venous thromboembolism (VTE). Limited data exists related to the effect of obesity on IBD-related complications, including VTE. The aim of our study was to assess the effect of obesity on VTE-related outcomes among patients with IBD.

METHODS: All adults hospitalized with IBD and VTE during the years 2010 to 2014, were identified using the Nationwide Inpatient Sample (NIS). These patients were divided into two groups: patients who were overweight, obese or morbidly obese (study group) and those with a normal BMI (control group). We compared demographics, inpatient mortality, length of stay and total charges among both these groups.

RESULTS: During the five-year study period, there were 295,592 hospitalizations for patients with IBD who also had a co-diagnosis of VTE. Compared with non-obese controls, obese patients were older (mean age 58.72 years vs 55.02 years; P < 0.001) and more likely to be females (53.24% vs 44.94%; P = 0.001). Patients in both groups were predominantly White (71.14% vs 73.17%) compared to African Americans (12.79% vs 13.49%) and Hispanics (11.25% vs 7.97%); P = 0.001. Comorbidities such as diabetes (45.55% vs 25.95%; P = 0.001) and hypertension (67.49% vs 55.97%; P = 0.001) were more prevalent in the obese group. Rates of colectomy were higher (3.98% vs 0.94%; P = 0.003) among obese patients. Complications associated with pulmonary embolism (PE) such as cardiogenic shock (1.16 vs 0.11; P = 0.07), right heart failure (9.83% vs 5.45%; P = 0.002) and respiratory failure (19.06% vs 14.66%; P = 0.001) were also higher in the obese group of patients. Length of stay (9.72 days vs 5.18 days; P < 0.001), inpatient mortality (2.32% vs 1.11%; P < 0.001) as well as total cost of hospitalization ($21,469 vs $17,291; P = 0.003) among obese patients. Complications associated with pulmonary embolism (PE) such as cardiogenic shock (1.16 vs 0.11; P = 0.07), right heart failure (9.83% vs 5.45%; P = 0.002) and respiratory failure (19.06% vs 14.66%; P = 0.001) were also higher in the obese group of patients. Length of stay (9.72 days vs 5.18 days; P < 0.001), inpatient mortality (2.32% vs 1.11%; P < 0.001) as well as total cost of hospitalization ($21,469 vs $17,291; P = 0.003) among obese patients vs VTE as compared to non-obese controls. On multivariate logistic regression, obesity was an independent predictor of length of stay (OR 0.13 [0.10-0.16]; P < 0.001) and inpatient mortality (OR 1.78 [1.61-1.99]; P < 0.001) among hospitalized IBD patients with VTE.

CONCLUSION: Obesity is associated with a significantly higher rate of VTE-related morbidity and mortality among patients with IBD. The total cost of hospitalization among these patients is also higher compared to non-obese individuals. Our study identifies obesity as a risk factor leading to poorer outcomes among IBD patients who are hospitalized with VTE.
third of situations in patients with mild-moderate UC. These results could be used to guide the design of clinical trials for these patient groups in order to guide best practice.

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Can CD74 Predict Treatment Response to Anti-TNF Agents in Inflammatory Bowel Disease?
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INTRODUCTION: Anti-TNF treatment, a game changer for inflammatory bowel disease (IBD) therapy, promotes mucosal healing in patients with IBD (1). By poorly understood mechanisms, a considerable amount of IBD patients either do not primarily respond or lose response to anti-TNF agents (2). A recent study identified a strong association between a polymorphism in the CD74 gene and anti-TNF failure in IBD patients (3). This led us to hypothesize that anti-TNF failure is more likely to occur in the setting of defective CD74 signaling.

METHODS: To test this hypothesis, we used the common DSS-induced colitis mouse model that mimics key immunological and histopathological features of IBD in humans. We studied both normal wild-type (WT) and CD74 deficient mice. Mice were given alternating days of DSS and normal drinking water to allow for partial repair. Anti-TNF antibody at 10 mg/kg was injected one hour prior to oral DSS administration.

RESULTS: We found that anti-TNF therapy did not protect CD74 deficient mice from DSS-induced body weight loss, colon shortening, and tissue damage measured by the histological score and FITC- fluorescence staining for the Claudin-3 tight junction marker. In keeping with the severe intestinal damage observed, extensive loss of Claudin-3 was found in CD74 deficient mice after treatment with DSS. Also, myeloperoxidase (MPO) levels, a widely used marker of intestinal inflammation, was significantly higher in the colonic tissue of CD74 deficient mice from DSS-induced colitis compared to WT mice. Anti-TNF treatment, a game changer for inflammatory Bowel Disease?

CONCLUSION: Thus, our findings support the hypothesis that an inferior response to anti-TNF drugs occurs with defective CD74 signaling, and provides a novel insight into the mechanism of treatment failure. Whether CD74 gene variants can be used to predict success in a personalized medicine approach to the management of IBD should be a focus of future research.

REFERENCES

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Serum Biomarkers Are Associated With Endoscopic and Clinical Outcomes in Crohn’s Disease Patients Receiving Vedolizumab Therapy
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INTRODUCTION: Vedolizumab is a selective monoclonal antibody directed against α4β7 integrin on lymphocytes and is safe and effective for the induction and maintenance of remission in Crohn’s disease. Serum biomarkers are needed to help guide therapy and predict patient outcomes. This study evaluated biomarker concentrations and patient outcomes during vedolizumab treatment.

METHODS: Serum was collected from patients with Crohn’s disease receiving vedolizumab infusions at weeks 0, 2, 6, 14 and ≥26. Biomarkers including soluble (s)-ICAM-1, s-vascular cell adhesion molecule (VCAM)-1, s-intracellular adhesion molecule (ICAM)-1, and s-mucosal addressin cell adhesion molecule (mAdCAM)-1 were measured and evaluated as surrogate markers associated with clinical and endoscopic outcomes. Statistical analysis was performed using the Mann-Whitney U test for continuous data.

RESULTS: Twenty-two patients with Crohn’s disease were included (Table 1). In all patients, s-ICAM-1 significantly decreased over time as compared to baseline, and s-a4b7 increased compared to baseline (Figure 1). Overall, s-ICAM-1 and s-a4b7 did not change significantly over time in the entire cohort, however, differentially changed in patients with remission. At week 2, median concentrations of s-ICAM-1 were higher in patients with compared to those without clinical remission: 675.1 ng/mL vs. 270.1 ng/mL (P = 0.046). At week 6, median s-ICAM-1 concentrations were higher in patients with compared to without endoscopic remission: 545.7 ng/mL vs. 286.2 ng/mL (P = 0.046) and clinical remission: 669.1 ng/mL vs. 291 ng/mL, respectively (P = 0.046). At week 26, median s-ICAM-1 concentrations were higher in patients with compared to without endoscopic remission: 515.7 ng/mL vs. 286.2 ng/mL (P = 0.046) and numerically higher in patients with clinical remission: 533.3 ng/mL vs. 291 ng/mL, respectively (P = 0.1).

CONCLUSION: These findings suggest that serum biomarkers may help guide clinical decision-making in patients receiving vedolizumab treatment.