Impact of Anti-TNF on The Prevalence of Hepatobiliary and Pancreatic Disorders Associated with Inflammatory Bowel Disease: A Population-based Study

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Introduction and Aim

Hepatobiliary and pancreatic disorders are among the common extra-intestinal manifestations of inflammatory bowel diseases (IBD), both in Crohn's Disease and Ulcerative Colitis (UC). Tumor necrosis factor inhibitor agents (anti-TNF) have been widely used in the management of IBD, and are considered relatively safe. The aim of this study is to investigate the impact of anti-TNF agents on the prevalence of hepatobiliary and pancreatic disorders in IBD patients using a large nationwide US-based database.

Methods

We used a commercial database (Explorys Inc, Cleveland, OH) which includes electronic health record data from 26 major integrated US healthcare systems. We identified adults (age >18 years) who were diagnosed with either CD or UC in the period between 1999 and 2022 who were treated with any type of anti-TNF agents for IBD. We collected demographic data including age, gender and race. We investigated the prevalence of known hepatobiliary and pancreatic disorders in IBD including Autoimmune Hepatitis (AIH), Cholelithiasis, Primary Sclerosing Cholangitis (PSC), portal vein thrombosis (PVT), Cholangiocarcinoma (CCA), and idiopathic pancreatitis (IP) in patients with and without anti-TNF therapy. Moreover, We also identified patients with CD and UC on anti-TNF who developed lupus (excluding Sulfasalazine). IBM SPSS® Statistics version 28.0.1 is used for statistical analysis.

Results

Out of the 69,260,780 adult patients in the database, we identified 249,480 patients with CD (0.36%) of whom 39280 received anti-TNF (15.7%). In addition, 209,020 with UC (0.30%) of whom 19860 received anti-TNF (9.50%). Figure 1 illustrates demographic characteristics of IBD patients on anti-TNF. CD patients who received anti-TNF were less likely to develop cholelithiasis (OR 0.88 [0.84-0.93]), PVT (OR 0.61 [0.50-0.74]), PSC (OR 0.59 [0.37-0.94)] or CCA (OR 0.50 [0.34-0.73]). Similarly, UC patients who received anti-TNF were less likely to develop cholelithiasis (OR 0.88 [0.79-0.97]), PVT (OR 0.65 [0.52-0.81]), PSC (OR 0.64 [0.44-0.93]), or CCA (OR 0.52 [0.36-0.75]). Both CD and UC groups on anti-TNF have a higher risk of AIH (OR 1.38 [1.13-1.69]) vs (OR 1.27 [1.02-1.58]), respectively. Likewise, both groups are at higher risk of IP (OR 1.95 [1.59-2.38] vs (OR 1.79 [1.38-2.31]). Furthermore, patients on CD and UC groups who received anti-TNF are more likely to develop lupus (OR 2.14 [1.59-2.90]) vs (OR 1.92 (1.29-2.84].

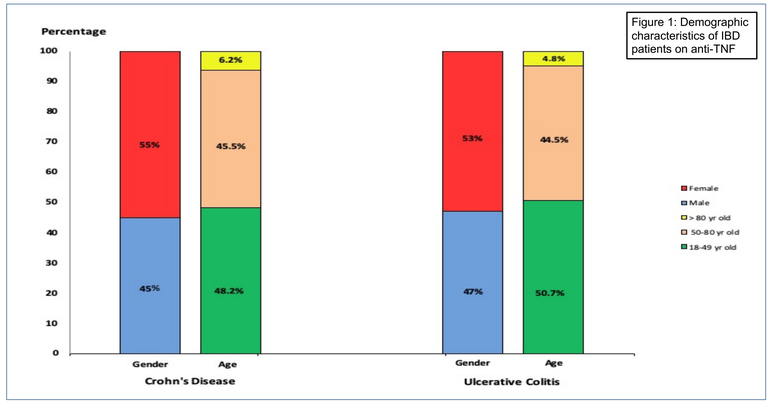

Figure 1: Demographic characteristics of IBD patients on anti-TNF

Table 1: Hepatobiliary and pancreatic disorders in CD and UC with anti-TNF therapy

	Crohn's Disease			Ulcerative Colitis		
	Total (Percentage)	On Anti-TNF (Percentage)	OR (95% CI)	Total (Percentage)	On Anti-TNF (Percentage)	OR (95% CI)
	249,480	39280		209,020	19,860	
AIH	590 (0.23%)	120 (0.030%)	1.38 (1.13-1.69)	790 (0.37%)	90 (0.45%)	1.27 (1.02-1.58)
CCA	350 (0.14%)	30 (0.07%)	0.50 (0.34-0.73)	590 (0.28%)	30 (0.15%)	0.52 (0.36-0.75)
Cholelithiasis	16420 (6.58%)	1560 (3.97%)	0.88 (0.84-0.93)	13030 (6.23%)	430 (2.16%)	0.88 (0.79-0.97)
IP	490 (0.19%)	130 (0.33%)	1.95 (1.59-2.38)	450 (0.21%)	70 (0.35%)	1.79 (1.38-2.31)
PVT	1090 (0.43%)	110 (0.28%)	0.61 (0.50-0.74)	1320 (0.63%)	80 (0.40%)	0.65 (0.52-0.81)
PSC	200 (0.08%)	20 (0.05%)	0.59 (0.37-0.94)	480 (0.22%)	30 (0.15%)	0.64 (0.44-0.93)
Lupus*	210 (0.084%)	60 (0.15%)	2.14 (1.59-2.90)	180 (0.086%)	30 (0.15%)	1.92 (1.29-2.84)

OR: Odds Ratio; CI; Confidence Interval; AIH: Auto-immune hepatitis; CCA: Cholangiocarcinoma; IP: Idiopathic Pancreatitis; PVT: Portal vein thrombosis; PSC: Primary sclerosing cholangitis.
*Lupus is investigated for correlation with anti-TNF

Conclusion

In this large retrospective study, we found that IBD patients who were treated with anti-TNF were significantly less likely to develop cholelithiasis, PVT, PSC or CCA. However, a higher risk of AIH and IP is observed in the anti-TNF group. This relationship may reflect an autoimmune side effect of anti-TNF, e.g. lupus-like reactions. Further study is needed to explore potential causality.

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