Update on Cutaneous Manifestations of IBD

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Pearls:

On Microbiome...
- Aberrations in gut microbiome (dysbiosis) may have implications for health at both intestinal and extraintestinal sites (e.g. lungs, joints, skin) (1,2).
- Probiotics may help to correct dysbiosis, but optimal agents/doses/etc. for individual diseases is unknown. Species and bacterial counts vary by preparation. VSL#3 may be beneficial in ulcerative colitis (3).
- Probiotic effects may extend beyond gut—e.g. there may be a role for oral probiotics in pregnancy or early life for prevention of atopic dermatitis (4,5).

On IBD and skin...
- IBD is a spectrum of systemic inflammatory diseases (ulcerative colitis, Crohn’s disease) which is linked to a complex interplay of genetic factors (immunoregulatory defects, gut permeability), environmental triggers, and the microbiome (6).
- Inflammation in the gut can drive inflammation at other extraintestinal sites including skin.
- Cutaneous markers of IBD include neutrophilic dermatoses, psoriasiform eruptions, nutritional dermatoses, and granulomatous skin lesions (i.e. cutaneous Crohn’s).
- Women with psoriasis have significantly increased risk of Crohn’s (RR, 3.86, 95% CI 2.23 to 6.67), especially with psoriasis + psoriatic arthritis (RR, 6.43, 95% CI 2.04 to 20.32) (7).
- Consider nutritional (e.g. zinc) deficiency in IBD patients with recalcitrant perianal/genital dermatitis (8).
- Management perianal/genital eruptions in IBD: check alk phos/zinc level, swab culture for staph/strep/candida; add zinc supplement, barrier creams, mild steroid ointments; consider contacts/discontinue hygiene wipes; skin biopsy if persists.

On working up GI disease...
- Who should be worked up for IBD? Abdo pain or diarrhea with red flag symptoms; dermatoses highly associated with IBD (PG, severe aphthae, granulomatous cheilitis, cutaneous Crohn’s, severe perianal HS like lesions); dermatoses sometimes associated with IBD AND GI sx.
- Fecal calprotectin is a marker for neutrophil activity; stable at room temp/can be collected at home; 93 percent sensitive and 96 percent specific for IBD in adults; specificity is less (76 percent) in children and teenagers; screening with fecal calprotectin could result in a 67 percent reduction in the use of colonoscopies for IBD diagnosis in adults; also helpful in monitoring IBD activity (9).

On prescribing to patients with IBD...
- Consider disease pathophysiology when determining treatment strategy: IBD involving the skin (treat the IBD), adverse event due to drug used to treat IBD (modify treatment or treat through), reactive skin condition triggered by IBD (treat IBD +/- addl specific treatment).
- IBD / celiac dz pts may have lower bone mineral density even in absence of prior steroid exposure (10). Provide appropriate bone prophylaxis EARLY when using systemic steroids for skin disease (rapid decline in BMD in first 3 months of steroid use, peaks at 6 months, then slower steady loss).
- Optimize Ca intake (800-1000 mg/d) and vitamin D intake (600-800 units/d). Check 25-OH D; goal > 20 ng/mL (per Institute of Medicine). Resources for bone health: American College of Rheumatology 2017 Recommendations for the Prevention and Treatment of Glucocorticoid Induced Osteoporosis; FRAX Tool to determine risk category: WHO Fracture Risk Assessment Tool – URL: [http://www.shef.ac.uk/FRAX](http://www.shef.ac.uk/FRAX).

- Oral antibiotics. Gut flora recovers incompletely after repeated antibiotic courses in healthy individuals (11). The longterm implications of this for healthy patients and for IBD patients are unknown (11). Per a recent study, the hazard ratio (HR) for developing IBD for any exposure to a tetracycline antibiotic was 1.39 (1.02, 1.90); for Crohn’s disease associations was particularly pronounced with doxy (HR) 2.25 (1.27 4.00) (12).

- Isotretinoin. Two recent studies show no significant association between isotretinoin use and IBD (13,14); a 2016 metaanalysis similarly showed no increased risk of IBD in patients exposed to isotretinoin (15).

**On skin diseases associated with TNF-alpha inhibitors…**

- In a recent cohort, 1.6% of TNF inhibitor treated IBD patients got psoriasis; most commonly plaque (57%), scalp (14%), palmoplantar (14%); >75% controlled with topical therapies (16).

- Genetic polymorphisms may play a role in who gets psoriasiform eruption. Pediatric patients with Crohn’s who developed infliximab associated psoriasis were more likely to be homozygous for specific polymorphisms in the IL-23R gene (17).

- Ustekinumab is approved for treatment of Crohn’s and beneficial for TNFi induced psoriasiform eruption, in particular psoriatic alopecia (18).

- Secukinumab, brodalumab, and ixekizumab for psoriasis have the potential to exacerbate IBD (19).

**References:**

2- Inflamm Bowel Dis 2012; 18:968–980.
4- Epidemiology 2012; 23(3):402-14.
5- Int J Dermatol. 2018 Feb 8. PMID: 29417549
6- Inflamm Bowel Dis 2006; 12 Suppl 1:S3-9.

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