Casual Friday Presents

Hemorrhoids and FM Perspectives

A BIOGENETIX CLINICAL PRESENTATION

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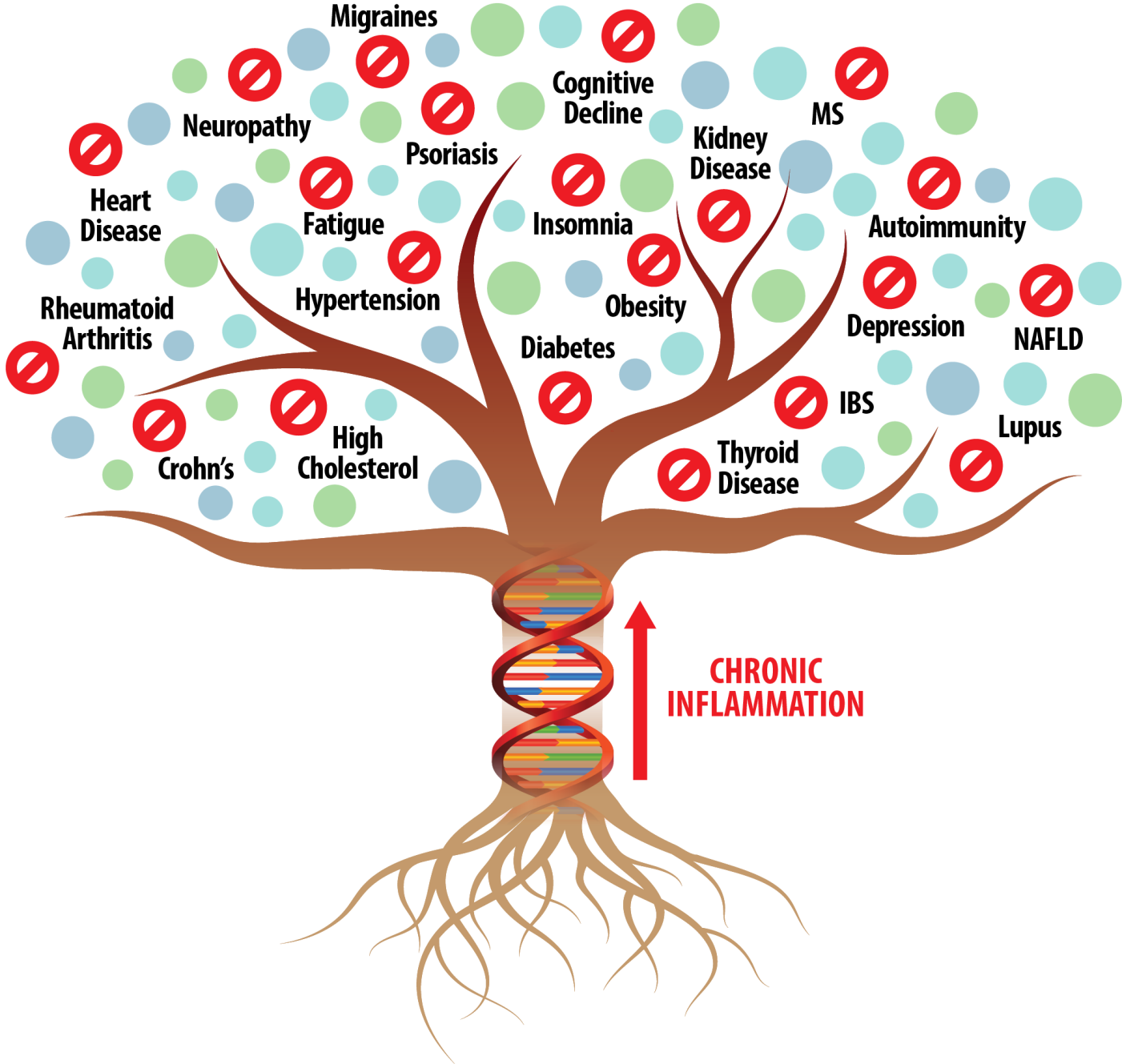
Migraines
Neuropathy
Psoriasis
Cognitive Decline
MS
Kidney Disease
Heart Disease
Fatigue
Insomnia
Autoimmunity
Hypertension
Obesity
Depression
NAFLD
Rheumatoid Arthritis
Diabetes
IBS
Lupus
Crohn's
High Cholesterol
Thyroid Disease
CHRONIC INFLAMMATION

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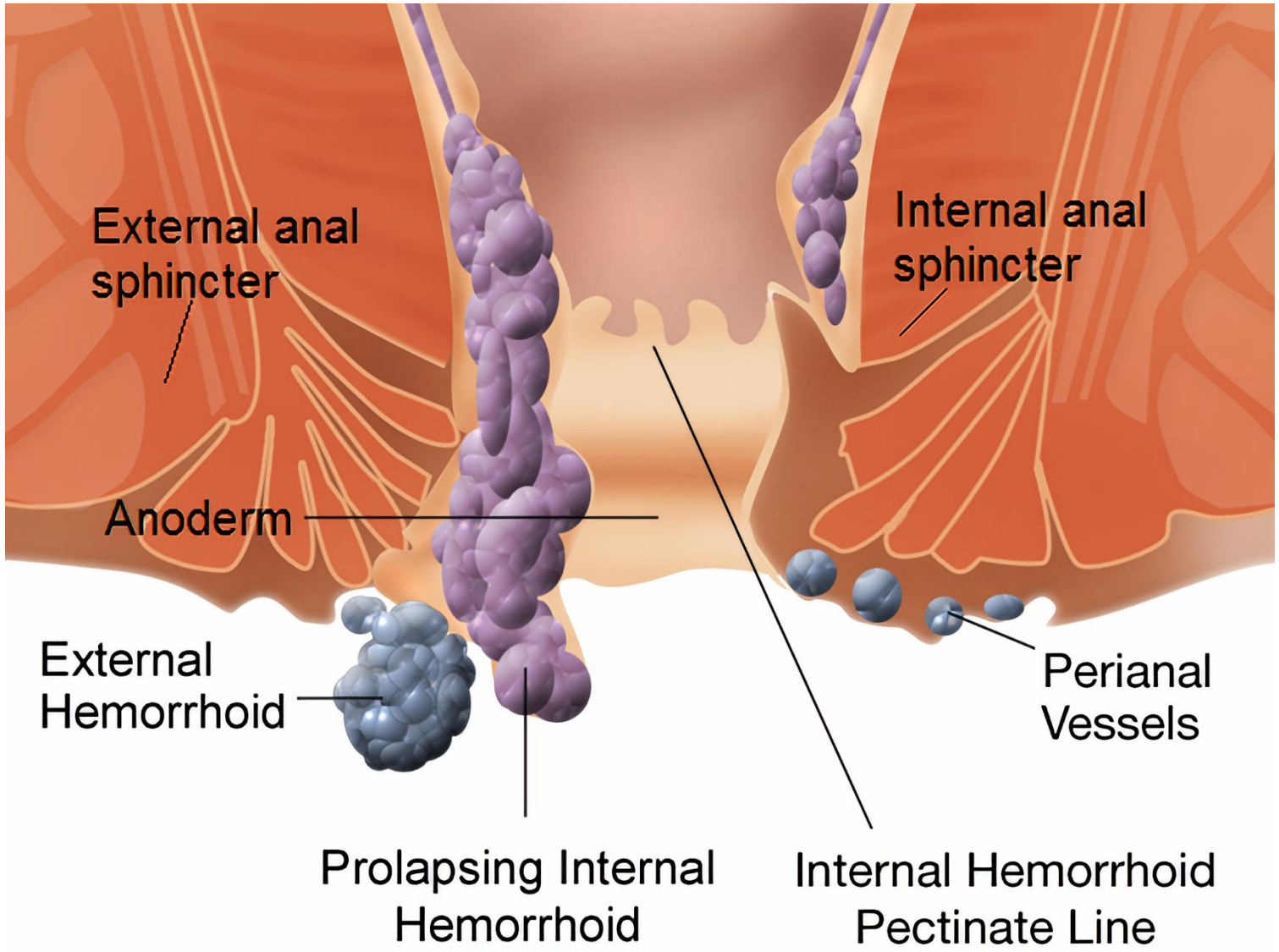
Hemorrhoids



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World J Gastroenterol. 2012 May 7;18(17):2009–2017. doi: 10.3748/wjg.v18.i17.2009

Hemorrhoids: From basic pathophysiology to clinical management

Varut Lohsiriwat [1]

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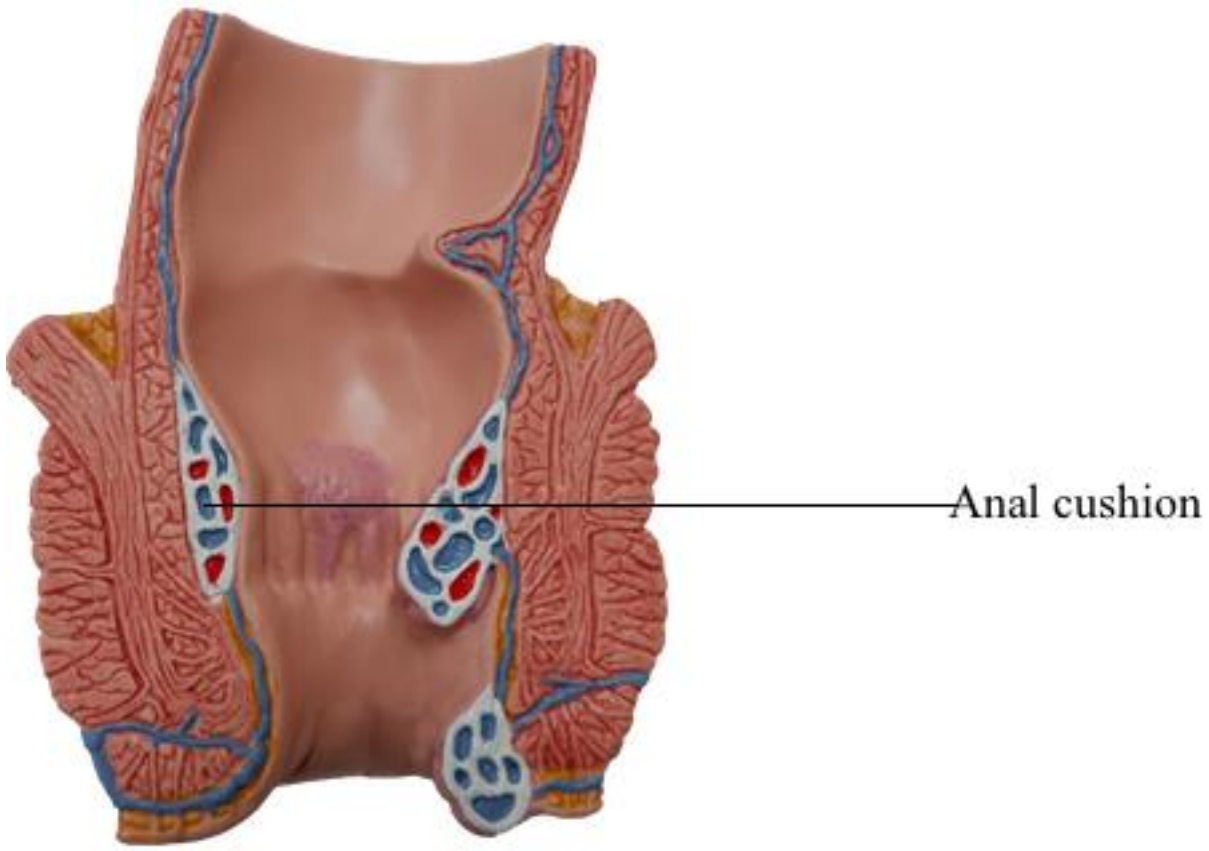
PMCID: PMC3342598 PMID: 22563187

Hemorrhoids are a very common anorectal condition defined as the symptomatic enlargement and distal displacement of the normal anal cushions. They affect millions of people around the world, and represent a major medical and socioeconomic problem. Multiple factors have been claimed to be the etiologies of hemorrhoidal development, including constipation and prolonged straining. The abnormal dilatation and distortion of the vascular channel, together with destructive changes in the supporting connective tissue within the anal cushion, is a paramount finding of hemorrhoidal disease[1]. An inflammatory reaction[2] and vascular hyperplasia[3,4] may be evident in hemorrhoids. This article firstly reviewed the pathophysiology and other clinical backgrounds of hemorrhoidal disease, followed by the current approaches to non-operative and operative management.

Hemorrhoids



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Today, the theory of sliding anal canal lining is widely accepted[6]. This proposes that hemorrhoids develop when the supporting tissues of the anal cushions disintegrate or deteriorate. Hemorrhoids are therefore the pathological term to describe the abnormal downward displacement of the anal cushions causing venous dilatation. There are typically three major anal cushions, located in the right anterior, right posterior and left lateral aspect of the anal canal, and various numbers of minor cushions lying between them[7] (Figure 1). The anal cushions of patients with hemorrhoids show significant pathological changes. These changes include abnormal venous dilatation, vascular thrombosis, degenerative process in the collagen fibers and fibroelastic tissues, distortion and rupture of the anal subepithelial muscle (Figure 2). In addition to the above findings, a severe inflammatory reaction involving the vascular wall and surrounding connective tissue has been demonstrated in hemorrhoidal specimens, with associated mucosal ulceration, ischemia and thrombosis[2].

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Several enzymes or mediators involving the degradation of supporting tissues in the anal cushions have been studied. Among these, matrix metalloproteinase (MMP), a zinc-dependent proteinase, is one of the most potent enzymes, being capable of degrading extracellular proteins such as elastin, fibronectin, and collagen. MMP-9 was found to be over-expressed in hemorrhoids, in association with the breakdown of elastic fibers[8]. Activation of MMP-2 and MMP-9 by thrombin, plasmin or other proteinases resulted in the disruption of the capillary bed and promotion of angioproliferative activity of transforming growth factor β (TGF-β)[9].

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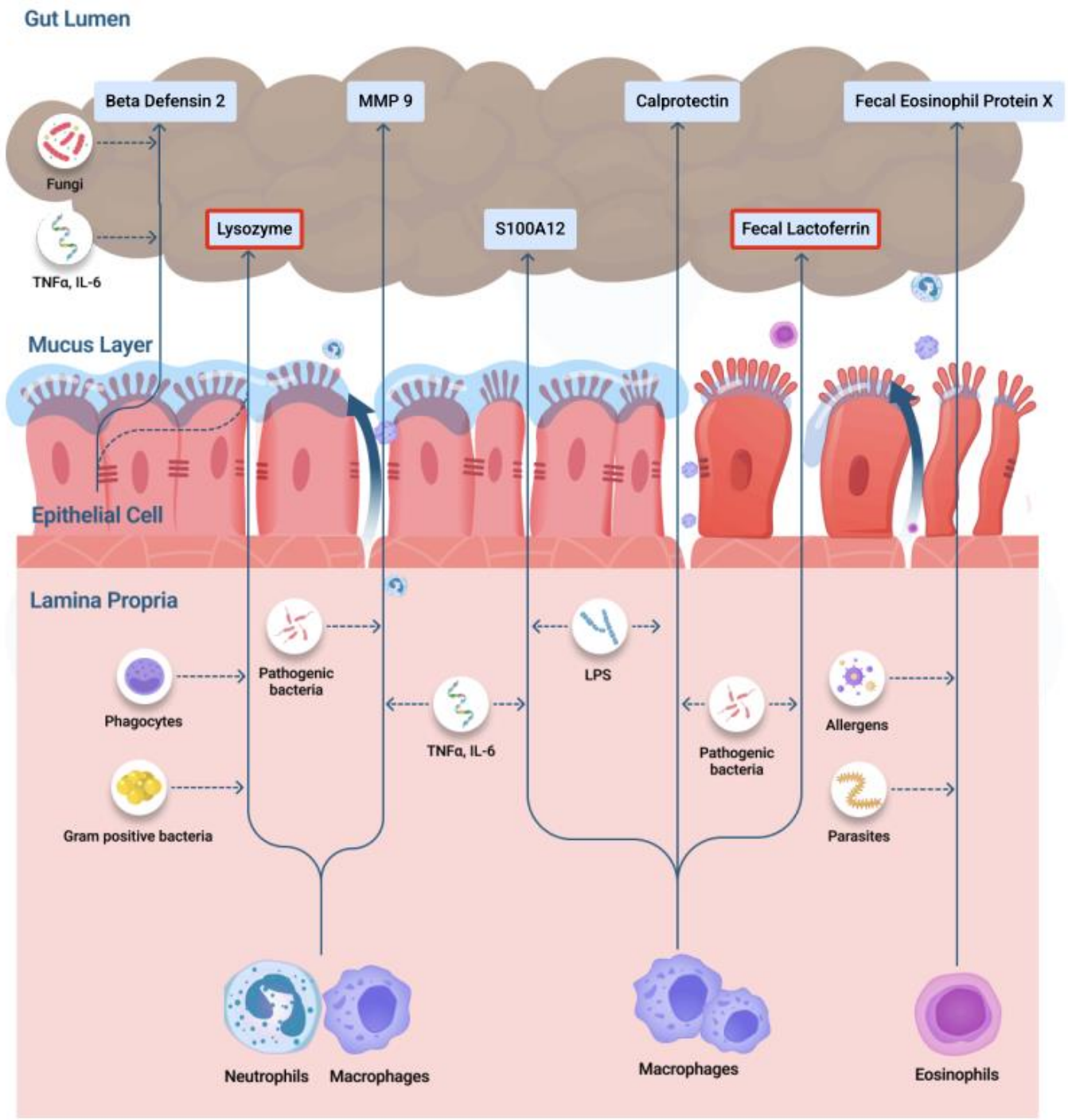
Gut Lumen
Beta Defensin 2
MMP 9
Calprotectin
Fecal Eosinophil Protein X
Fungi
TNFα, IL-6
Lysozyme
S100A12
Fecal Lactoferrin
Mucus Layer
Epithelial Cell
Lamina Propria
Pathogenic bacteria
LPS
Phagocytes
TNFα, IL-6
Pathogenic bacteria
Allergens
Gram positive bacteria
Parasites
Neutrophils
Macrophages
Macrophages
Eosinophils

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Constipation and prolonged straining are widely believed to cause hemorrhoids because hard stool and increased intraabdominal pressure could cause obstruction of venous return, resulting in engorgement of the hemorrhoidal plexus[1]. Defecation of hard fecal material increases shearing force on the anal cushions. However, recent evidence questions the importance of constipation in the development of this common disorder[14,16,17]. Many investigators have failed to demonstrate any significant association between hemorrhoids and constipation, whereas some reports suggested that diarrhea is a risk factor for the development of hemorrhoids[16]. Increase in straining for defecation may precipitate the development of symptoms such as bleeding and prolapse in patients with a history of hemorrhoidal disease. Pregnancy can predispose to congestion of the anal cushion and symptomatic hemorrhoids, which will resolve spontaneously soon after birth. Many dietary factors including low fiber diet, spicy foods and alcohol intake have been implicated, but reported data are inconsistent[1].

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CLASSIFICATION AND GRADING OF HEMORRHOIDS

A hemorrhoid classification system is useful not only to help in choosing between treatments, but also to allow the comparison of therapeutic outcomes among them. Hemorrhoids are generally classified on the basis of their location and degree of prolapse. Internal hemorrhoids originate from the inferior hemorrhoidal venous plexus above the dentate line and are covered by mucosa, while external hemorrhoids are dilated venules of this plexus located below the dentate line and are covered with squamous epithelium. Mixed (interno-external) hemorrhoids arise both above and below the dentate line. For practical purposes, internal hemorrhoids are further graded based on their appearance and degree of prolapse, known as Goligher's classification: (1) First-degree hemorrhoids (gradeI): The anal cushions bleed but do not prolapse; (2) Second-degree hemorrhoids (grade II): The anal cushions prolapse through the anus on straining but reduce spontaneously; (3) Third-degree hemorrhoids (grade III): The anal cushions prolapse through the anus on straining or exertion and require manual replacement into the anal canal; and (4) Fourth-degree hemorrhoids (grade IV): The prolapse stays out at all times and is irreducible. Acutely thrombosed, incarcerated internal hemorrhoids and incarcerated, thrombosed hemorrhoids involving circumferential rectal mucosal prolapse are also fourth-degree hemorrhoids[18].

Table 1.

Current management of internal hemorrhoids by grade

Treatments	**GradeI**	**Grade II**	**Grade III**	**Grade IV**	**Acute thrombosis or strangulation**
Dietary and lifestyle modification	×	×	×	×	×
Medical treatment	×	×	×-selected		
Non-operative treatment					
Sclerotherapy	×	×			
Infrared coagulation	×	×			
Radiofrequency ablation	×	×			
Rubber band ligation	×	×	×-selected		
Operative treatment					
Plication		×	×		
DGHAL		×	×		
Hemorrhoidectomy		×-selected	×	×	×-emergency
Stapled hemorrhoidopexy			×	×	

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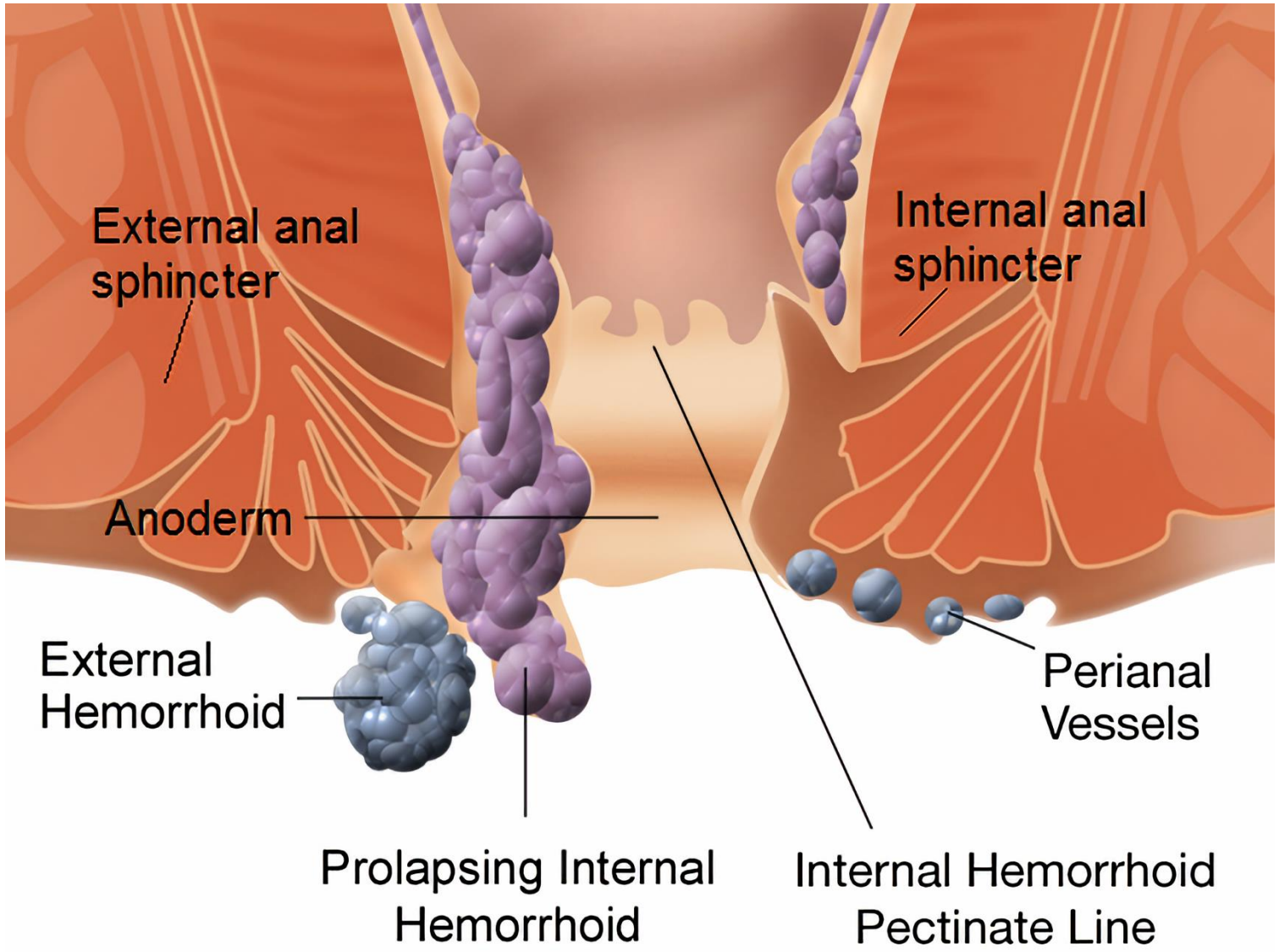
Therapeutic treatment of hemorrhoids ranges from dietary and lifestyle modification to radical surgery, depending on degree and severity of symptoms. Although surgery is an effective treatment of hemorrhoids, it is reserved for advanced disease and it can be associated with appreciable complications. Meanwhile, non-operative treatments are not fully effective, in particular those of topical or pharmacological approach. Hence, improvements in our understanding of the pathophysiology of hemorrhoids are needed to prompt the development of novel and innovative methods for the treatment of hemorrhoids.

Hemorrhoids



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Summary Drivers

•Straining during bowel movements, which raises intra-abdominal pressure and stresses the veins.

•Chronic constipation or diarrhea, which prolongs straining or irritates the area.

•Prolonged sitting, especially on the toilet, reducing blood flow and weakening vein walls.

•Low-fiber diets that result in hard stools, exacerbating straining.

•Other factors like obesity, pregnancy (due to added weight and hormonal changes), aging (as tissues lose elasticity), heavy lifting, all of which can contribute to pelvic floor dysfunction and sustained pressure on the veins.

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Summary Connections? Test.

•Sedentary lifestyle → Increases risk of constipation and obesity, both linked to hemorrhoids and cardiovascular problems.

•Obesity and poor diet → Excess weight raises intra-abdominal pressure (promoting hemorrhoids) and is a major risk factor for hypertension, diabetes, and coronary heart disease.

•Chronic straining → Can temporarily spike blood pressure, but this isn't a primary driver of long-term cardiovascular damage.

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The Workup

1. Biogenetix general screen

2. Stool testing with zonulin family peptide.

Functional Imbalance Scores

Key (< 2) : Low Need for Support (2-3) : Optional Need for Support (4-6) : Moderate Need for Support (7-10) : High Need for Support

	Need for Digestive Support	Need for Inflammation Modulation	Need for Microbiome Support	Need for Prebiotic Support	Need for Antimicrobial Support
	MALDIGESTION	**INFLAMMATION**	**DYSBIOSIS**	**METABOLIC IMBALANCE**	**INFECTION**
	3	0	2	10	8
Biomarkers	Products of Protein Breakdown △ Fecal Fats △ Pancreatic Elastase ●	Secretory IgA △ Calprotectin ● Eosinophil Protein X ● Occult Blood ●	PP Bacteria/Yeast △ IAD/Methane Score ● Reference Variance ● Total Abundance ●	Beta-glucuronidase ▲ Total SCFA's ▽ n-Butyrate Conc. ● SCFA (%) ●	Pathogenic Bacteria ▲ PP Bacteria/Yeast △ Parasitic Infection ● Total Abundance ●
Therapeutic Support Options	• Digestive Enzymes • Betaine HCl • Bile Salts • Apple Cider Vinegar • Mindful Eating Habits • Digestive Bitters	• Elimination Diet/ Food Sensitivity Testing • Mucosa Support: Slippery Elm, Althea, Aloe, DGL, etc. • Zinc Carnosine • L-Glutamine • Quercetin • Turmeric • Omega-3's • GI Referral (If Calpro is Elevated)	• Pre-/Probiotics • Increase Dietary Fiber Intake • Consider SIBO Testing • Increase Resistant Starches • Increase Fermented Foods • Meal Timing	• Pre-/Probiotics • Increased Dietary Fiber Intake • Increase Resistant Starches • Increase Fermented Foods • Calcium D-Glucarate (for high beta-glucuronidase)	• Antibiotics (if warranted) • Antimicrobial Herbal Therapy • Antiparasitic Herbal Therapy (if warranted) • *Saccharomyces boulardii*

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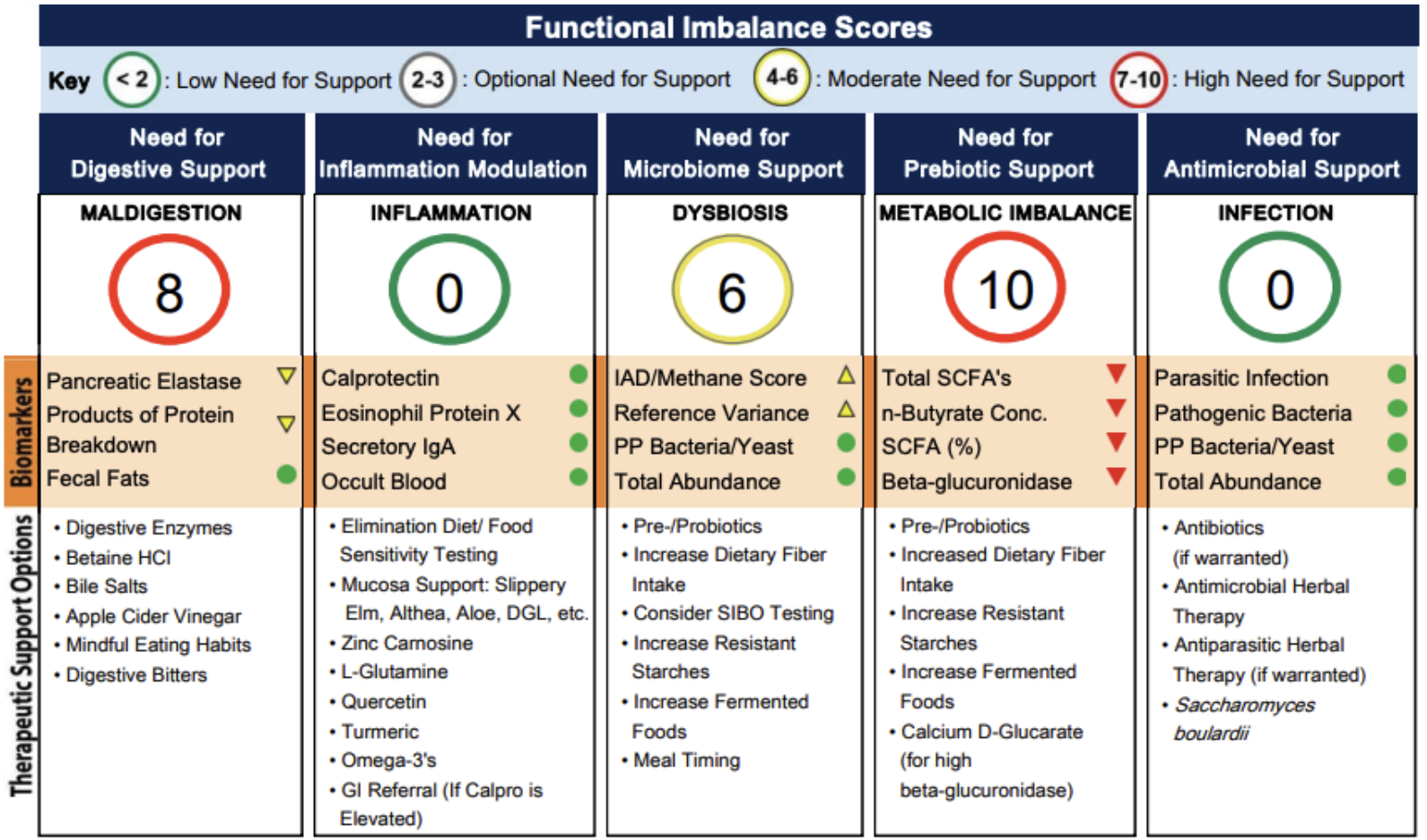
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MALDIGESTION
8
INFLAMMATION
0
DYSBIOSIS
6
METABOLIC IMBALANCE
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INFECTION
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Biomarkers
Pancreatic Elastase
Products of Protein Breakdown
Fecal Fats
Calprotectin
Eosinophil Protein X
Secretory IgA
Occult Blood
IAD/Methane Score
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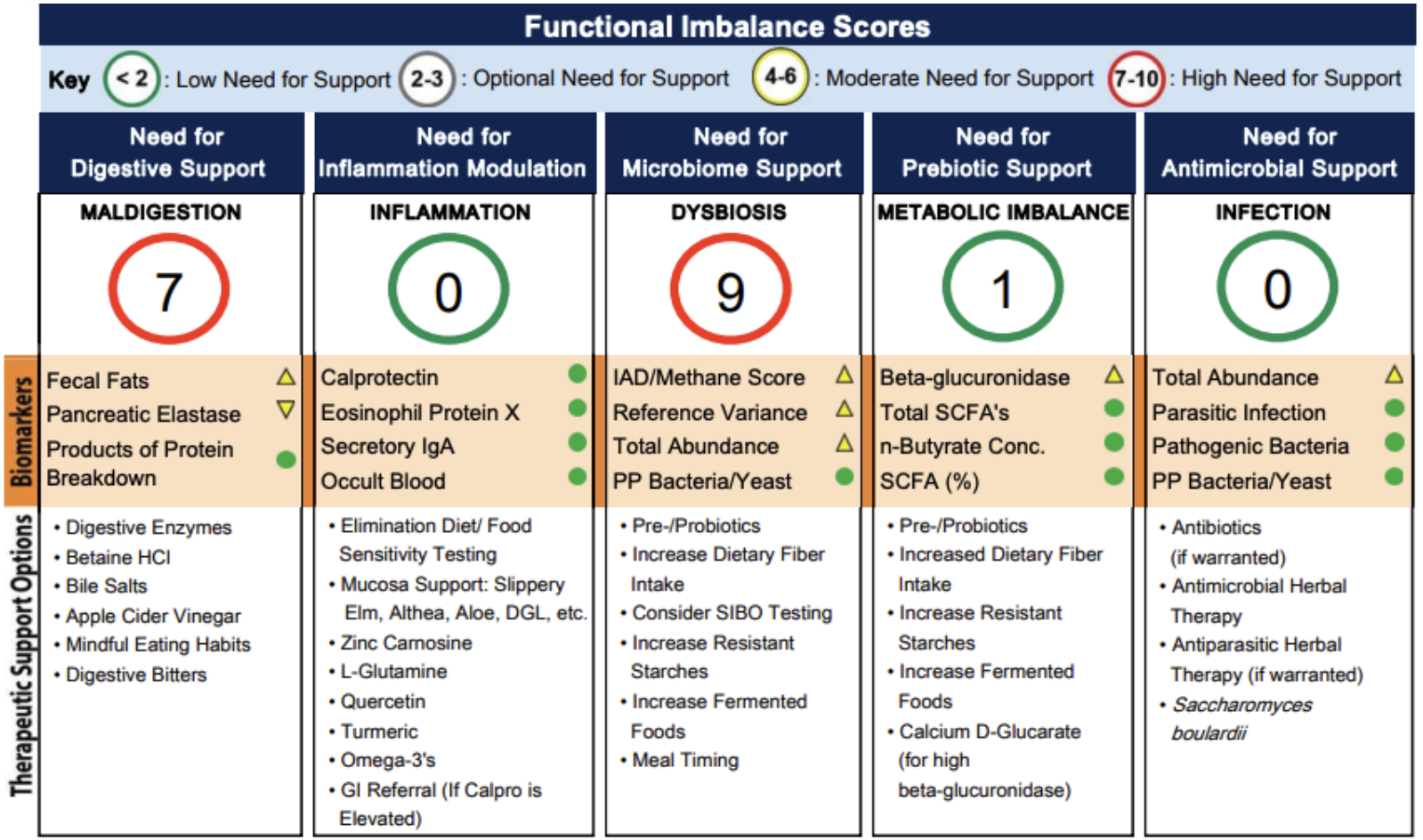
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DYSBIOSIS
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In general…

1. Is there a cardiovascular mechanism (age, body weight, lipids, inflammation).
2. Varicose veins anywhere else?
3. How is the gut health? Stool consistency and frequency.
4. One time issue? → perhaps an event onset.
5. Chronic? → get to work on the FM.

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