Parkinson's

GI tract dysfunction and Parkinson's

1. Gastrointestinal dysfunction in Parkinson's disease.

Parkinsonism & Related Disorders. 2011;17:10–15.

"GI dysfunction is receiving increasing attention and recognition as an important clinical component of PD. Beyond this, however, evidence is now emerging that the ENS may even be the pathophysiologic "ground zero" of the disorder. GI dysfunction in PD involves virtually all levels of the GI tract and is the source of significant functional impairment for many individuals with PD. All of this makes it important for neurologists and other clinicians to be familiar with the GI features of PD and to inquire about them when seeing patients so that truly optimal care can be administered to individuals with this disorder."

2. Gastrointestinal dysfunction in Parkinson's disease.

The Lancet Neurology. 2003 Feb;2(2):107-16.

"There is growing recognition that gastrointestinal dysfunction is common in Parkinson's disease (PD). Virtually all parts of the gastrointestinal tract can be affected, in some cases early in the disease course. Bowel dysfunction can consist of both slowed colonic transit with consequent reduced bowel-movement frequency, and difficulty with the act of defecation itself with excessive straining and incomplete emptying."

3. Gastrointestinal dysfunction in Parkinson's disease.

Journal of Clinical Neuroscience. 1998;5(2):136-46.

" Gastrointestinal dysfunction is a frequent and occasionally dominating symptom of Parkinson's disease (PD). Features of gastrointestinal dysfunction include disordered control of saliva, dysphagia, gastroparesis, and constipation in the sense of decreased bowel movement frequency, and defecatory dysfunction necessitating increased straining and resulting in incomplete evacuation. Gastroparesis may produce a variety of symptoms, including nausea, and also may be responsible for some of the motor fluctuations seen with levodopa therapy. Bowel dysfunction in PD may be the result of both delayed colon transit and impaired anorectal muscle coordination."

4. Gastrointestinal dysfunction in Parkinson's disease.

Klaus Krogh. Department of Hepatology and Gastroenterology V, Aarhus University Hospital Denmark

"... infrequent defecation in otherwise healthy men is associated with increased risk of developing PD in later life."

"Parkinson's disease is a multi-system disorder including gastrointestinal dysfunction. Most gastrointestinal symptoms in PD can be attributed to dystonia of the striated muscle cells in the oesophagus and anal canal and to reduced stimulation of the smooth muscle forming the remaining part of the gastrointestinal canal. No guidelines to evaluation and treatment of gastrointestinal dysfunction in PD exist and evidence for treatment is generally poor."

5. <u>Treatment for Helicobacter pylori infection and risk of Parkinson's disease in</u> <u>Denmark.</u>

The European Journal of Neurology 2012;19:864–869.

"We identified 4484 patients with a first time PD diagnosis between 2001 and 2008 from the Danish National Patient Register (DNPR) and 22 416 population controls from the Danish Civil Registration System (CRS). Information on drug use was obtained from the National Prescription Registry (NPR). We used logistic regression to compute odds ratios (OR) for the association between treatment for HP and risk of PD."

"Prescriptions for HP-eradication drugs and proton pump inhibitors (PPI) 5 or more years prior to the diagnosis of PD were associated with a 45% and 23% increase in PD risk, respectively."

Fungus and Parkinson's disease

1. <u>Chronic polysystemic candidiasis as a possible contributor to onset of idiopathic</u> Parkinson's disease.

Bratislava Medical Journal. 2006;107:227-30

In the presence of acetaldehyde dopamine is converted into salsolinol, a neurotoxin involved in apoptosis of dopaminergic neurons. Increased production of acetaldehyde is associated with chronic polysystemic candidiasis (CPC). Chronically elevated levels of acetaldehyde in patients with CPC might participate in the formation of salsolinol and its metabolites in the brain

contributing to the destruction of dopaminergic cells in substantia nigra. Clinical mental symptoms of PD often correspond with the mental manifestations of CPC. This hypothesis may constitute basis for further scientific and clinical research of PD etiopathogenesis

A correlation between fungal infection and PD is also supported by findings that <u>seborrheic</u> <u>dermatitis</u> is particularly common in patients with PD.

2. <u>A laboratory-based study on patients with Parkinson's disease and seborrheic</u> <u>dermatitis: the presence and density of Malassezia yeasts, their different species</u> <u>and enzymes production.</u>

BMC Dermatology 2014, 14:5

The concept of skin as a mirror of Parkinsonism dates back to the beginning of the last century.

From this laboratory-based study a positive correlation between Seborrheic dermatitis, Parkinson's disease, Malassezia yeasts incidence was established.

3. Anemia or low hemoglobin levels preceding Parkinson disease.

Neurology. Oct 27, 2009; 73(17): 1381–1387.

Our results support an association between anemia experienced early in life and the later development of Parkinson disease.

SIBO and Parkinson's

1. <u>Prevalence of small intestinal bacterial overgrowth in Parkinson's disease.</u>

Movement Disorders 2011;26:889–892.

Small intestinal bacterial overgrowth is highly prevalent in PD.

2. The role of small intestinal bacterial overgrowth in Parkinson's disease.

Movement Disorders. 2013 Aug;28(9):1241-9.

The prevalence of small intestinal bacterial overgrowth was significantly higher in Parkinson patients. The eradication of small intestinal bacterial overgrowth resulted in improvement in motor fluctuations.

54.5% of PD subjects were found to have SIBO by virtue of abnormality on one or both of the breath tests, thus virtually duplicating their earlier results in this regard. The investigators went on to show that the presence of SIBO in individuals with PD may have important consequences, not only in the form of GI symptoms but also with regard to motor function in that individuals with SIBO experienced more severe motor fluctuations, particularly of the "delayed-on" and dose failure type. They also demonstrated improvement in these motor fluctuations following antibiotic treatment of the SIBO.

GI dysfunction, now including both gastroparesis and SIBO, may be responsible for at least some portion of motor fluctuations also suggests that movement disorders neurologists might do well to cultivate a working relationship with a gastroenterologist who is interested in motility disorders. In fact, successful treatment of these GI aspects of PD may obviate, or at least delay, the need for more invasive medical and neurosurgical treatment approaches for motor fluctuations in some individuals with PD.

Ketogenic diet and Parkinson's disease

1. <u>Neuroprotective and disease-modifying effects of the ketogenic diet</u>

2. <u>Treatment of Parkinson disease with diet-induced hyperketonemia: a feasibility</u> <u>study.</u>

- 3. Dr Perlmutter [video]
- 4. Blog post from Perlmutter