Herbs for Upper Digestive Overgrowth of Flora

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Abstract

Herbal medicine has a major therapeutic role in patients with upper digestive overgrowth of flora (UDOF), often incompletely called small intestinal bacterial overgrowth. Here, the use of herbs with various types of effects on gut motility are reviewed, along with their known antimicrobial effects relevant to UDOF. Bitter and pungent herbs that stimulate gut motility ("prokinetics") are crucial in UDOF patients with slow transit time. Berberine-containing herbs such as *Berberis aquifolium* (Oregon grape) and *Hydrastis canadensis* (goldenseal), as well as various members of the genus *Artemisia*, including but not limited to *Artemisia annua* (sweet Annie) and *Artemisia absinthium* (wormwood), are particularly reviewed, as antimicrobial bitters useful for many UDOF patients. The steam-distilled volatile oils of carminative herbs, which are antimicrobial and actually inhibit overactive gut motility and thus are most useful in patients with diarrhea and cramping, are also discussed at length. *Mentha* x *piperita* (peppermint), *Melissa officinalis* (lemonbalm), *Coriandrum sativum* (coriander), and *Pimpinella anisum* (anise) are all reviewed in particular. A combination formula containing bitters and carminatives is reviewed. The safety of these herbs with various antibiotics used to treat UDOF is discussed, along with the use of partially hydrolyzed guar gum as an adjunct to rifaximin therapy.

Keywords: small intestinal bacterial overgrowth, peppermint, herbal medicine, prokinetic, bitter, carminative

Introduction

A condition generally called small intestinal bacterial overgrowth (SIBO) has come to be recognized in the past few years, though documented well before this to occur commonly. The term "SIBO" reflects the early discoveries in people with the condition, in which nonpathogenic bacteria from the colon were demonstrated to be present in the small intestines in abnormally high amounts. However, more recent research shows that often there is overgrowth in the stomach or even esophagus, and fungi, archaea, and bacteria can be overgrown.[1–3] Therefore, the more accurate term "upper digestive overgrowth of flora" (UDOF) will be used here.

UDOF patients can experience a range of symptoms depending on where exactly the flora have overgrown, what exact microbes have overgrown, and what exact gases they are producing (hydrogen sulfide, molecular hydrogen, methane, or others). For instance, overgrowth of methanogens tends to cause decreased motility, which can lead to gastroparesis (for flora in the stomach) or constipation (for small intestinal overgrowth). Overgrowth of hydrogen-forming saccharolytic anaerobic bacteria more often leads to increased motility, causing esophageal reflux with or without inflammation if present in the esophagus or stomach and diarrhea when present in the small intestines. Many patients report a sensation of bloating, usually that cannot be confirmed by objective observation of the abdomen, with either type of overgrowth as the gases trigger stretch receptors in the stomach inappropriately. Overgrowth of *Candida* spp. in the small intestines and stomach can cause what is known as the autobrewery syndrome. Dietary carbohydrates are fermented, releasing sufficient alcohol to cause clinical inebriation in some patients, accompanied by fruity breath due to excess ketone body formation.

Different types of UDOF can be somewhat distinguished by lactulose- or glucose-stimulated breath testing. High hydrogen levels indicate overgrowth of saccharolytic, mostly anaerobic bacteria, and are more likely to result in increased gut motility and diarrhea. High methane levels clearly indicate overgrowth of the archaeon *Methanobrevibacter smithii*, which is more likely to cause slow transit times and constipation. Overgrowth of *Methanobrevibacter* can hide hydrogen overproduction, as this organism uses hydrogen to make methane. No validated clinical testing currently available can determine if patients have fungal overgrowth. Breath testing is far from perfect, and it should be noted that both false-positives and, less so, false-negatives do occur with this test.

UDOF is a state that arises from some underlying cause, and thus this cause, or more likely multiple causative factors, really should be treated to resolve the problem. The most obvious triggers are chronic use of acid-suppressing drugs.[2,3] Chronic

hyperglycemia in diabetes mellitus causing glycation and dysfunction of innervation of the bowel and subsequent dysmotility is another common scenario whereby UDOF can arise.[4]

There is also mounting evidence that bacterial food poisoning and other gastrointestinal bacterial infections involving organisms that produce cytolethal distending toxin B (CDT-B) can induce a cross-reactive autoimmune reaction against vinculin, an important cell–cell junction protein involved in gut motility.[5] The impaired gut motility can be sufficiently severe that colon flora can migrate into the upper gut, no longer effectively kept in the colon by peristalsis. Serum anti-vinculin and anti-CDT-B antibodies are significantly increased in patients with irritable bowel syndrome diarrhea-predominant (IBS-D) compared to patients with inflammatory bowel diseases, celiac disease, and healthy controls.[6] This suggests even relatively mild cases of bacterial food poisoning and other gastrointestinal bacterial infections can have severe lasting consequences and should potentially be treated more aggressively.

Anyhow, the fact that UDOF is not just a simple "infection" of the upper digestive tract is a crucial issue. Generally, treatment so far has focused on killing the microbes present in the wrong area. However, by not addressing underlying causes, this is almost doomed to fail in the long run (and, indeed, clinically has been very disappointing in the author's experience). Thus, while antimicrobials will be discussed as a component of therapy, agents to correct gastric acidity and to promote overall motility will also be addressed as equally or more important components of successful resolution of disease. This is borne out in studies of conventional drugs and appears true using natural product-based therapy.[7]

UDOF may underlie a number of very common conditions, including but not limited to non-erosive esophageal reflux disease (NERD), erosive gastroesophageal reflux disease, IBS (as noted above), chronic noninfectious diarrhea, and functional dyspepsia. Other conditions and surgeries in which UDOF is commonly reported to co-occur (and it is unclear in each case which comes first: UDOF or the disease in question) include Crohn's disease, celiac disease, acute diverticulitis, bariatric surgery, short bowel syndrome, metabolic liver disease (so-called nonalcoholic fatty liver disease), diabetes mellitus with gastroparesis, liver cirrhosis, scleroderma, radiation enteritis, and many others.[8] Such a dizzying array of associations makes UDOF worth knowing about and worth treating when present, at least if other therapies haven't worked or patients are significantly bothered by gas, burping, bloating, heartburn, and other symptoms of UDOF.

Prokinetic Herbs

There are two groups of herbs that stimulate gut motility: bitter and pungent herbs (see Table 1). Most traditional are the bitter-tasting herbs, which includes a large number of herbs. These herbs are most appropriate in patients with definitively proven slow motility, including patients with slow-transit constipation and IBS constipation-predominant (IBS-C). It is also worth noting, as summarized in Table 1, that many of these herbs have secondary actions, particularly antimicrobial effects, which are almost always helpful in UDOF patients.

Several of these herbs (notably the berberine-containing herbs such as *Berberis*/*Mahonia* and *Hydrastis*) are more well known as antimicrobials. While this may indeed be a major way they work, this has not been definitively determined in any clinical trial. It is just as likely they help patients with UDOF because of their prokinetic effects, or a combination of their prokinetic and antimicrobial effects.

One clinical trial has been conducted on prokinetic (and antimicrobial) herbal therapy for UDOF patients.[9] In this open trial, 165 patients with symptoms and a positive lactulose-challenged breath test suggestive of UDOF were randomized to take either one of two herbal protocols (products 1a and 1b or 2a and 2b, two capsules b.i.d. of both products; see Table 2 for details) or rifaximin 400 mg t.i.d. for four weeks. While these herbal mixtures were fairly clearly formulated to be mostly antimicrobial products, it can be seen that each contains some of the prokinetic herbs mentioned above. At the end of the four weeks, 104 patients had follow-up breath tests that showed the herbal and rifaximin groups were equally likely to have achieved a negative breath test. Unfortunately, there was no report of whether symptoms improved, and there was no follow-up beyond the immediate end of the study (thus patients could have relapsed as early as one day after treatment ended, and this study would not have captured this information). The lack of blinding of course also weakened the validity of these results. The mechanism of action of these products was not determined.

There were two serious adverse effects in the rifaximin group (anaphylaxis and *Clostridium difficile* diarrhea) and none in the herbal group. There were four milder adverse reactions (diarrhea and hives) in the drug group compared to one case of diarrhea in the herbal group. Though these differences didn't reach statistical significance, they do tend to suggest the herbal treatment may be safer than rifaximin.

Unfortunately, prokinetic herbs have not been specifically studied to prove they are working by increasing motility in patients with UDOF and slow motility, particularly methane-predominant disease. This seems likely, but scientific proof is really needed to confirm this. Nevertheless, as Table 1 shows, they have many other properties. So, even if the prokinetic effects are not sufficient, they can be helpful.

A great example of a prokinetic herb is the *Artemisia* genus, which includes Eurasian native species such as *A. absinthium* (wormwood), *A. vulgaris* (ài yè, mugwort), *A. capillaris* (yīn chén hāo, capillary wormwood), and *A. annua* (qīng hāo, sweet Annie), American native species such as *A. tridentata* (big sagebrush), *A. ludoviciana* (Mexican white sagebrush), and *A. douglasiana* (California mugwort), and joint Eurasian/American native species such as *A. dracunculus* (tarragon, estragon). The prokinetic effects of wormwood have been demonstrated in humans, at least for the gallbladder.[10] The steam-distilled volatile oil of *A. judaica* (Judean wormwood) from the Middle East was demonstrated to suppress methanogenesis in

Table 1. Prokinetic Herbs

Category	Examples	Other actions	Typical tincture dose[a]
Bitter herbs	*Berberis aquifolium* (Oregon grape) and related species	Antimicrobial	1–2 mL t.i.d.
	Hydrastis canadensis (goldenseal)	Antimicrobial, astringent	1–2 mL t.i.d.
	Anemopsis californica (yerba mansa)	Antimicrobial, astringent	1–2 mL t.i.d.
	Artemisia absinthium (wormwood) and related species	Antimicrobial, inflammation modulating	0.5–1 mL t.i.d.
	Menyanthes trilobata (bogbean)	Astringent	1–3 mL t.i.d.
	Achillea millefolium (yarrow)	Astringent, inflammation modulating, antimicrobial	2–3 mL t.i.d.
	Cynara scolymus (artichoke)	Antiadhesive	2–3 mL t.i.d.
	Gentiana lutea (gentian)	None	0.25–1 mL t.i.d.
Pungent herbs	*Zingiber officinale* (ginger)	Inflammation modulating, antimicrobial, antiemetic, corrigent	1–2 mL t.i.d.
	Alpinia galanga (galangal)	Inflammation modulating, antimicrobial, antiemetic, corrigent	1–2 mL t.i.d.
	Cinnamomum verum (cinnamon) and related species	Antimicrobial	1–2 mL t.i.d.
	Origanum basilicum (basil) and *O. vulgare* (oregano)	Antimicrobial	1–2 mL t.i.d.
	Syzygium aromaticum (cloves)	Antimicrobial	3–5 drops t.i.d.

[a]Optimally all of these should be dosed 10–15 minutes before meals in a little water. Note: 1 mL roughly equals one dropperful, which roughly equals 25–30 drops.

ruminants, which might correlate to suppression or killing of *Methanobrevibacter*, though research is needed in humans to confirm this.[11] The steam-distilled volatile oil of sweet Annie has been shown to have a broad range of antibacterial and anti-*Candida* activity in vitro.[12] One study found that a sesquiterpene lactone called dehydroleucodine from California mugwort could reduce excessive motility in mice with diarrhea as mediated by alpha-2 adrenergic receptors.[13] This suggest *Artemisia* spp. plants may be able to increase or decrease motility depending on the state of the gut.

Cynara scolymus (artichoke) is a bitter that has also been studied in a context suggesting it might help UDOF patients. A standardized aqueous extract of the leaves of artichoke was given to patients with functional dyspepsia in an open trial, and then those retrospectively determined to have mixed or alternating IBS (IBS-M) were also also studied.[14] Of the 208 patients with IBS-M, 26% improved sufficiently with artichoke therapy as to no longer be diagnosable with IBS-M by standard criteria. Total symptoms and quality of life improved significantly compared to baseline on average. The mechanism of

Table 2. Herbal Products in a Randomized Trial for UDOF Patients[a]

Product 1a	Product 1b	Product 2a	Product 2b
Tinospora cordifolia	*Anethum graveolens*	*Thymus vulgaris* volatile oil 0.2 mL	*Coptis chinensis* 30 mg
Equisetum arvense	*Stemona sessilifolia*		*Berberis aristata* 70 mg
Tabebuia spp.	*Artemisia absinthium*	*Origanum vulgare* volatile oil 0.1 mL	Berberine sulfate 400 mg
Thymus vulgaris	*Pulsatilla chinensis*		*Scutellaria baicalensis*
Artemisia dracunculus	*Brucea javanica*	*Salvia officinalis* extract 75 mg	*Phellodendron chinense*
Sida cordifolia	*Picrasma excelsa*		*Zingiber officinale*
Olea europaea	*Acacia catechu*	*Melissa officinalis* extract 50 mg	*Glycyrrhiza uralensis*
	Hedyotis diffusa		*Rheum officinale*
	Achillea millefolium		

[a]Only those amounts of ingredients disclosed on product labels are given here.
UDOF, upper digestive overgrowth of flora.

action was not investigated, and presence of UDOF not confirmed, but the association of IBS and UDOF makes it possible that the bitter nature of this herb was part of what worked here. Artichoke, as well as Mexican white sagebrush, were found to inhibit growth and adherence of *Campylobacter jejuni* and *C. coli*, while Mexican white sagebrush inhibited their cytotoxicity in vitro.[15] Thus, these herbs might also help prevent UDOF due to bacterial food poisoning or gastroenteritis. Other research suggests artichoke has antibacterial properties that could be helpful for UDOF.[16]

It has long been debated how bitter herbs increase gut motility, with theories ranging from direct stimulation of bitter receptors in the gut to effects on the brain. A recent human study found that with sufficient doses of gentian or wormwood tinctures (but not capsules), the cephalic phase of digestion was stimulated with subsequent increased gut motility.[17] This study supported that tasting these herbs is important to their function but that a sufficiently large dose must be given for them to be effective.

Clinically, prokinetic herbs have been crucial in preventing relapse and leading to long-term remission (possibly cure) of UDOF. For example, a 22-year-old man with chronic NERD that worsened when he ate fermented foods or foods high in fermentable oligosaccharides, disaccharides, monosaccharides, and polyols (FODMAPs) had a positive lactulose-stimulated breath test suggesting he might have UDOF. He was initially treated with a combination of natural antimicrobials for four weeks, specifically lactoferrin 250 mg t.i.d., volatile oil of *Origanum vulgare* (oregano) 0.5 mg t.i.d., and a mixture of berberine-rich herbs 1,000 mg t.i.d. His symptoms resolved completely but quickly relapsed once he stopped taking the herbs. He was on a low FODMAPs diet during this treatment. He completed a second round of treatment, but this time he followed up with a tincture of *Gentiana lutea* (yellow gentian) 15 drops sipped before meals as a prokinetic. Again his NERD symptoms vanished with the treatment, but this time they stayed away with only a very occasional, minor, transient episode of epigastric burning. With two years' follow-up, he still has not had any significant recurrence, even after discontinuing the yellow gentian at about the one year mark.

Berberine-Containing Herbs

Much of the focus on herbs and UDOF has been finding alternative antimicrobials. Many of the herbs in the formulas reviewed above (and see Table 2) are clearly antimicrobial. Often, it is berberine that is first considered as an herbal antimicrobial for UDOF patients. This isoquinoline alkaloid is notoriously poorly bioavailable and thus has a particular affinity for the gastrointestinal tract.[18] It is found in such herbs as *Berberis aquifolium* (Oregon grape) and all other *Berberis* and *Mahonia* species, *Hydrastis canadensis* (goldenseal), *Coptis chinensis* (goldthread, huáng lián), *Phellodendron amurense* (Amur cork tree, huáng bǎi), and *Xanthorrhiza simplicissima* (yellow root). Other than the study mentioned above, there has been no clinical research on isolated berberine or any of these herbs in UDOF patients.

In an obese mouse model, ethanol extracts of goldthread and pure berberine both significantly inhibited normal gut microbes in the phyla Firmicutes and Bacteroidetes, resulting in reduced body fat, improved glucose tolerance, and an improved lipid profile.[19] This included inhibition of organisms in the genus *Lactobacillus*, normally considered beneficial flora. Berberine targets bacteria in multiple ways, including inhibiting proteins, damaging nucleic acids, and suppressing metabolism.[20]

There are some compelling reasons to use whole plant extracts of berberine-containing species over isolated berberine. *Berberis lycium* (Indian barberry), native to the mountainous region of Kashmir and surroundings, has been shown to be active against *Escherichia coli* and *Candida albicans* in vitro.[21] In this study, *E. coli* became resistant to pure berberine after 72 hours of exposure, while resistance never developed to a methanol extract of Indian barberry roots. Such an extract was more effective than berberine alone at killing *E. coli* (though the reverse was true for *C. albicans*). Efflux pumps appear to play a major role in resistance to berberine, including P-glycoprotein/multidrug-resistance pumps and the more recently described TetA efflux pump in *E. coli*.[22] Methoxylated flavonolignans in the fruits of Oregon grape and *Berberis fendleri* (Fendler's barberry) have been shown to inhibit these efflux pumps and increase antimicrobial activity of berberine in vitro.[23,24]

Antimicrobial Carminatives

Carminative herbs are generally part of the Lamiaceae family and contain calcium channel-blocking, low molecular weight terpenoids. These herbs are helpful for situations in which there is intestinal hypermotility and intestinal cramping, such as that which commonly occurs in IBS-D. If anything, these would be contraindicated in states of low motility such as IBS-C. Note that low motility could cause UDOF resulting in either of these conditions, but once the misplaced flora start to produce gases in the wrong part of the gut, they can cause increased or decreased motility. These herbs, again usually due to their terpenoid content, are also antimicrobial, making them interesting for those patients with UDOF and problems with hypermotility. Some members of the Apiaceae family are also carminative as a result of their phenylpropanoid content.

For decades now, *Mentha* x *piperita* (peppermint; Fig. 1) steam-distilled volatile oil has been reported to help adults and children with IBS.[25–27] It has long been assumed and stated that peppermint oil works simply as a carminative/spasmolytic. However, it is also plausible in hindsight that it was active as an antimicrobial and that it helped a significant number of IBS patients because they had undetected UDOF.[28,29] Peppermint oil is also antifungal, an action that has been shown to reduce visceral sensitivity in a rat model.[30]

Many other volatile oils have also been studied in people with IBS, the effectiveness of which has been attributed to their carminative effects. However, the actual mechanisms weren't

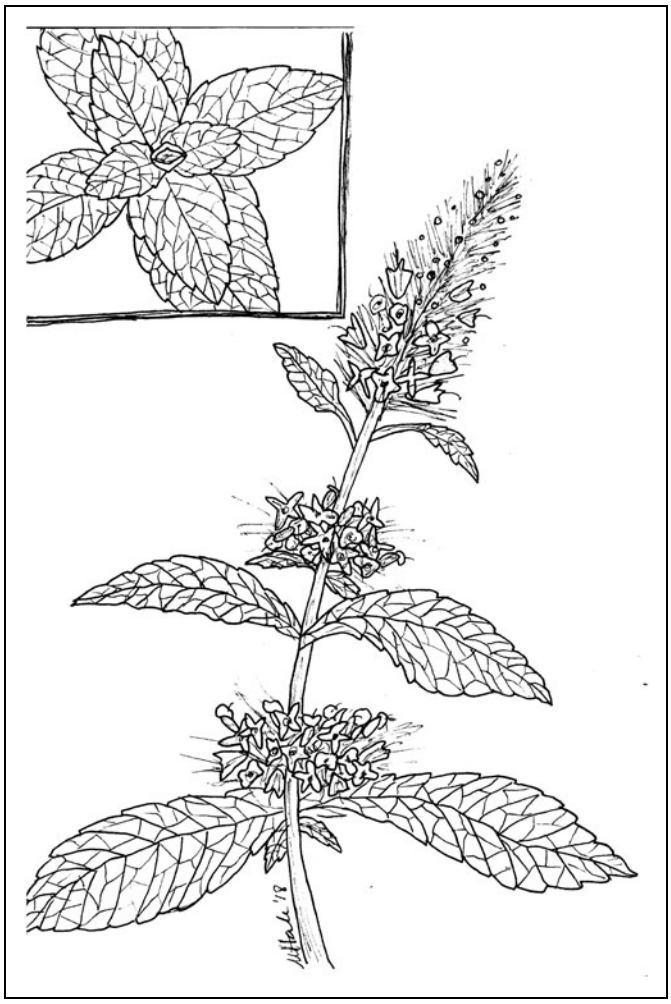

Figure 1. ***Mentha* x *piperita* (peppermint). Drawing by Meredith Hale and reprinted with permission.**

determined, so activity against misplaced colon flora was possibly involved. One double-blind trial compared a combination of *Mentha spicata* (spearmint), *Melissa officinalis* (lemonbalm), and *Coriandrum sativum* (coriander) steam-distilled volatile oils 30 gtt t.i.d. to placebo in 32 adults with IBS of any type for eight weeks.[31] They were further treated with loperamide if they had IBS-D and psyllium if they had IBS-C. By the end of the trial, the volatile oil group had significantly less abdominal pain and bloating than the placebo group. Benefits were seen equally in IBS-D and IBS-C patients, suggesting the mechanism couldn't be just from motility-inhibiting effects (as this would have worsened IBS-C). One study found that peppermint, lemonbalm, and coriander were the most potent steam-distilled volatile oils against *E. coli* in vitro, and that peppermint and coriander oils were more potent than rifaximin in this model.[32]

Another double-blind trial randomized 120 adults with any subtype of IBS to either *Pimpinella anisum* (anise) or peppermint steam-distilled volatile oils or placebo for four weeks.[33] Both volatile oils were enteric coated. Patients taking anise oil were significantly more likely to be symptom free by the end of the trial compared to peppermint oil or placebo. Fully 75% of the anise oil group were symptom free at the end the trial (vs. 53% in the peppermint oil group and 35% in the placebo group). Again, benefits were seen in all subtypes of IBS, raising the possibility that UDOF-related treatment effects could have been responsible for the benefits.

Mixing Bitters and Carminatives

A proprietary formula known as Iberogast® or STW has been studied in IBS patients and, given the strong association of IBS and UDOF, suggests the formula could be useful for UDOF (and very frequently is used this way). The formula usually contains the ingredients listed in Table 3. As can be seen, it includes both bitters and carminatives. It is widely regarded as a prokinetic formula, though its ingredients are far more complex than this simple description. In fact, one clinical trial compared STW formula, *Iberis amara* (bitter candytuft) alone, a simplified STW formula (with bitter candytuft, chamomile, peppermint, caraway, licorice, and lemon balm), and placebo in 208 adults with IBS of any subtype.[34] The complete formula as well as the simplified formula both significantly reduced IBS symptoms compared to placebo or bitter candytuft alone.

Table 3. Ingredients in Iberogast Formula

Herb	Actions
Iberis amara (bitter candytuft) herb	Bitter
Carum carvi (caraway) fruit	Carminative, antimicrobial
Glycyrrhiza glabra (licorice) root	Inflammation modulating, hepatoprotective
Mentha x *piperita* (peppermint) leaf	Carminative, antimicrobial
Matricaria recutita (chamomile) flower	Carminative,[a] inflammation modulating
Chelidonium majus (celandine) herb	Bitter, cholagogue
Silybum marianum (milk thistle) seed	Hepatoprotective, insulin sensitizing
Angelica archangelica (garden angelica) root	Carminative

[a]This herb becomes bitter when heated; it is not clear how it is prepared in this formula.

Table 4. Summary of Herbs for Different UDOF-Related Conditions

Condition	Treatment suggestions[a]
IBS-D	Carminatives
IBS-C	Bitters and/or pungents
IBS-M	Carminatives, bitters, and pungents
Functional dyspepsia	Carminatives, bitters, or pungents
NERD	Bitters; avoid LES-relaxing carminatives
GERD	Bitters; avoid LES-relaxing carminatives and pungents (which are more likely to irritate already inflamed tissues)
Other conditions with rapid transit times or spasmodic pain	Carminatives (do not use bitters or pungents alone, but may be ok to combine with carminatives)
Other conditions with slow transit time	Bitters or pungents (do not use carminatives alone, but may be ok to combine with bitters and/or pungents)

[a]In all cases, antimicrobials without effects on gut motility may be useful.
IBS-D, irritable bowel syndrome diarrhea-predominant; IBS-C, irritable bowel syndrome constipation-predominant; IBS-M, mixed or alternating irritable bowel syndrome; NERD, non-erosive esophageal reflux disease; GERD, gastroesophageal reflux disease; LES, lower esophageal sphincter.

Again, UDOF was not specifically proven to be present in these patients, but it is likely many of them were affected, and so this mixed formula could have helped by treating dysbiosis (presence of abnormal flora in the colon, either because harmful microbes are present, beneficial microbes are absent or deficient, or the ratio of otherwise normal flora is imbalanced) as much as any other mechanism. It may have also, as needed, increased or decreased gut motility. A separate human clinical trial in fact found that the simplified STW formula mentioned above does not increase gut motility in patients with functional dyspepsia or gastroparesis, but it did not evaluate if these patients had UDOF and if the herbs were helpful for other reasons.[35] No human studies, but at least one rat study, confirm the simplified formula is also helpful for reflux esophagitis, another condition strongly associated with UDOF in humans.[36]

Drug Interactions

There is no evidence of negative interactions between any of the products mentioned herein and any of the common drugs used to treat UDOF patients (particularly rifaximin, metronidazole, and neomycin), though this has not been systematically evaluated in most cases. One old clinical trial did find berberine safe to combine with neomycin in children with infectious diarrhea.[37] None of these three antibiotics is a significant substrate for cytochrome P450 enzymes, so there is unlikely to be a pharmacokinetic interaction between the herbs mentioned in this article and these drugs. In particular berberine, which by itself in sufficient concentrations is a clinically significant inhibitor of CYP3A4, is safe with these drugs.

One randomized trial compared rifaximin 1.2 g q.d. to rifaximin at the same dose plus partially hydrolyzed guar gum 5 g q.d. in 77 adults with UDOF for 10 days.[38] Significantly more patients in the guar gum group (85% vs. 62%) than in the rifaximin-only group achieved a negative breath test by the end of the trial. Symptomatic improvement was equally likely to occur in both groups. The researchers speculated that guar gum helped with intestinal motility (regardless of whether patients had diarrhea, constipation, or alternating bowel patterns) and thus augmented the efficacy of rifaximin, but this mechanism was not proven. Given the safety of this treatment, it is often used clinically, though double-blind trials are warranted to confirm these preliminary observations.

Conclusion

Herbs can play a major role in helping patients with UDOF. As noted in Table 4, there is some individualization necessary to achieve optimal outcomes. In all cases, an antimicrobial strategy may be warranted, but regulation of gut motility is also very helpful both for alleviating symptoms and fixing the underlying problem that may have caused the UDOF. Once a patient's symptoms are eliminated, it is recommended that antimicrobial herbs should usually be stopped while the appropriate motility-related herbs (be they carminative, bitter, or pungent) be continued. Often, it is possible to choose herbs with both antimicrobial and motility-regulating effects, which may be the optimal approach, along with dietary adjustments (various types of low-FODMAPs diets usually) that are then slowly weaned over time. ■

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