Letters to the Editor

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Absence of Abdominal Pain Does Not Rule out Diagnosis of IBS

Original Article: Diagnosis and Management of IBS in Adults

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TO THE EDITOR: I appreciate this informative article on irritable bowel syndrome (IBS). However, I do not agree with the SORT recommendation, "The absence of abdominal pain can be used to rule out IBS." Given the data presented in *Table 1* of the article and the cited systematic review,[1] the clinical evidence does not support this assertion.

Based on data in *Table 1,* the positive likelihood ratio of abdominal pain in diagnosing IBS is about 1.3, and the negative likelihood ratio is about 0.31. A negative likelihood ratio in this range can assist in ruling out a disorder, but does not definitively rule it out.[2] For abdominal pain to rule out a disorder, the patient's pretest probability must be considered. For instance, the disorder could not be ruled out in a patient with a pretest probability of 50 percent, which would result in a posttest probability of 24 percent. However, it would be useful to rule out the disease in a patient with a pretest probability of 10 percent, which would result in a posttest probability of 3 percent. Successfully ruling out the diagnosis of IBS in many patients would likely take more than the presence or absence of any one symptom, such as abdominal pain.

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2. Furukawa TA, Strauss S, Bucher HC, Guyatt G. Diagnostic tests. In: Guyatt G, Rennie D, Meade MO, Cook DJ, eds. *Users' Guides to the Medical Literature.* 2nd ed. New York, NY: McGraw-Hill; 2008.

IN REPLY: In our article, we summarized information from a 2008 systematic review of the accuracy of individual symptoms to diagnose IBS.[1] For abdominal pain, the sensitivity is 90 percent and the specificity is 32 percent (positive predictive value = 9 percent; negative predictive value = 97 percent). The negative likelihood ratio is 0.016 to 0.034 (when weighted for a prevalence of 5 to 10 percent). Using the estimated prevalence of IBS in North America of 5 to 10 percent,[2] the posttest probability for IBS in a patient with abdominal pain is 6.5 to 12.8 percent, and the posttest probability for IBS in a patient without abdominal pain is 1.6 to 3.4 percent.

Because individual symptoms (e.g., abdominal pain, diarrhea, constipation) lack sufficient sensitivity and specificity to accurately diagnose IBS, clinical diagnostic criteria were developed. Of these, the Manning criteria are the most extensively studied (sensitivity, 63 to 90 percent; specificity, 70 to 93 percent).[3,4] The Rome I criteria in 1990 developed a consensus definition and criteria (sensitivity, 65 to 85 percent; specificity, 70 to 100 percent).[3,4] These were revised in 1999 with the Rome II criteria (sensitivity, 64 to 89 percent; specificity, 66 to 73 percent), and in 2006 with the Rome III criteria (sensitivity, 81 percent; specificity, 60 percent).[3,4] There have been eight validation trials for the Manning criteria, four for the Rome I criteria, three for the Rome II criteria, and none for the Rome III criteria.[4] All of these criteria include abdominal pain, which is required for the diagnosis of IBS using the Rome II and III criteria.

IBS is a complex disorder with nonspecific symptoms defined as abdominal discomfort or pain associated with altered bowel habits for at least three days per month in the previous three months, with the absence of organic disease. An accurate diagnosis of IBS is important to minimize risks and reduce unnecessary medical procedures and tests ▶

Table 1. Clinical Features of Type 2 Diabetes, Type 1 Diabetes, and Latent Autoimmune Diabetes of Adulthood

Features	*Type 2 diabetes*	*Type 1 diabetes*	*Latent autoimmune diabetes in adults*
Ketoacidosis	Usually absent	Will develop rapidly unless patient receives insulin replacement therapy	Absent at diagnosis, but may be present when patient becomes severely insulinopenic
Cardiovascular complications	Risk 2-4 times higher than individuals who are euglycemic	Increased risk of cardiovascular morbidity and mortality related to strokes, acute coronary events, and coronary revascularizations; high incidence rates compared with euglycemic individuals, especially in women	Same risk as patients with T2DM
Microvascular complications (retinopathy, nephropathy, neuropathy)	Increased	Increased	Increased
Pathophysiology	Peripheral insulin resistance; reduced pancreatic beta-cell mass and function; reduced insulin secretion	Autoimmune destruction of pancreatic beta-cells	Latent autoimmune destruction of pancreatic beta-cells
Autoantibodies	Negative	• GAD-65 autoantibodies • Islet-cell antigen-2 • Insulin autoantibodies • NOTE: Unlike LADA, T1DM patients typically are positive for all three autoantibodies	• GAD-65 autoantibody is typically the only one detected • Islet-cell antibodies
Insulin requirements for treatment	Usually late in the disease when the remaining beta-cell mass and function can no longer support acceptable glycemic control achieved by oral agents or incretin mimetics	Insulin is required from the time of diagnosis	Insulin should be initiated as soon as the patient develops autoantibodies

T2DM indicates type 2 diabetes mellitus; GAD-65 indicates glutamic acid decarboxylase; LADA indicates latent autoimmune diabetes in adults; T1DM indicates type 1 diabetes mellitus.

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while reducing cost. Without specific biomarkers or genetic tests, positive clinical diagnostic criteria in the absence of red flags, with positive history and physical examination findings, are the best way to diagnose IBS.

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3. Dang J, Ardila-Hani A, Amichai MM, Chua K, Pimentel M. Systematic review of diagnostic criteria for IBS demonstrates poor validity and utilization of Rome III. *Neurogastroenterol Motil.* 2012;24(9):853-e397.
4. Mearin F, Lacy BE. Diagnostic criteria in IBS: useful or not? *Neurogastroenterol Motil.* 2012;24(9):791-801.

Correction

Error in table column headings. In the editorial "Latent Autoimmune Diabetes in Adults" (April 1, 2010, p. 843), Table 1 (p. 844) contained errors in the column headings. The rightsholder of the original table, which contained these errors, granted *AFP* permission to reprint the table as published. The heading over the fourth column (Diabetes) should have been "Type 1 diabetes" and appeared over the third column. The heading over the third column (Latent autoimmune type 1 diabetes) should have been "Latent autoimmune diabetes in adults" and appeared over the fourth column. The table has been corrected online and is reprinted here. ■