

ment of Buruli ulcer as pyoderma gangrenosum with high-dose corticosteroids that impair cellular immune defense may cause harm.

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THE AUTHORS REPLY: We agree with Dr. Roupheal and colleagues that colonoscopy is not mandatory in the workup of pyoderma gangrenosum. However, it should be considered, as described in Table 3 of our article. The prevalence of inflammatory bowel disease in patients with bona fide pyoderma gangrenosum ranges from 27 to 36 percent, but the activity of the bowel disease may not correlate with a flare of or recovery from pyoderma gangrenosum.^{1,2}

We recognize that the identification or ruling out of inflammatory bowel disease does not confirm or refute a diagnosis of pyoderma gangrenosum. As with all laboratory tests, the decision to perform a colonoscopy should be considered on a patient-by-patient basis, and it should be performed only if clinically indicated.

We thank Dr. van der Werf and colleagues for identifying another entity that may produce pyoderma gangrenosum-like ulceration — the Buruli ulcer. As they mention, our study was a retrospective review of cases from one institution and a review of the English-language literature. Had we known of their unreported case, we would have included it in our review. We are certain that there are additional conditions, yet to be described, that can result in severe cutaneous ulceration that mimics pyoderma gangrenosum.

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Genetic Testing

TO THE EDITOR: In Burke's review of genetic testing (Dec. 5 issue),¹ Tables 1 and 2 offer examples of genetic tests. The tables are intended to be illustrative, not exhaustive, and most entries are based on information from GeneTests–GeneClinics at <http://www.geneclinics.org>. According to the author, this Web site contains a comprehensive and continually updated listing of available genetic tests. To our surprise, a search on this Web site for hereditary pancreatitis (Online Mendelian Inheritance in Man [OMIM] number 167800) failed.

Hereditary pancreatitis follows an autosomal dominant pattern of inheritance with 80 percent penetrance and is associated with mutations in the cationic trypsinogen gene (PRSS1, OMIM number 276000).² The disorder is characterized by multiple episodes of acute pancreatitis and the development of chronic pancreatitis, and the risk of pancreatic

cancer is increased by a factor of more than 50.³ Genetic testing is recommended in cases with recurrent (unexplained) acute pancreatitis, unexplained chronic pancreatitis, or a family history of pancreatitis in a first-degree or second-degree relative. In our opinion, genetic tests for hereditary pancreatitis should therefore be listed on the Web site.

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DR. BURKE AND A COLLEAGUE REPLY: The current pace of genetic research will produce many new genetic tests. Given this changing environment, clinicians need sources of information that provide comprehensive and unbiased information about genetic testing. It is a challenge to develop and maintain such data bases, because they require contributions from experts with diverse expertise, sophisticated search mechanisms, and a commitment to regular updating. We believe the publicly funded GeneTests–GeneClinics Web site is the best current source of information about genetic testing and one that will be of increasing value to clinicians as more genetic tests become available. For this reason, it was cited as a source in the review article.

Drs. Spanier and Bruno express concern about the completeness of this Web resource, because they did not locate information about genetic tests for hereditary pancreatitis when they searched for it. However, several tests for hereditary pancreatitis are listed in the “Laboratory Directory” section of the Web site. Drs. Spanier and Bruno may have initiated their search in the GeneReviews section of the

Web site; if so, they received a “failed search” message, reflecting the lack of an expert review on hereditary pancreatitis. Although the Web site has the long-term goal of providing detailed expert reviews (GeneReviews) for all genetic conditions of clinical interest, this goal has not yet been reached. Nevertheless, the screen indicating that the search had failed also offered the opportunity to search the Laboratory Directory, where tests for hereditary pancreatitis are listed. In addition, the Laboratory Directory can be reached from the home page of the Web site.

The comments by Drs. Spanier and Bruno are in keeping with other feedback indicating that the search mechanisms for GeneTests–GeneClinics could be improved. Changes have been instituted, and visitors to the site should find genetic testing information easier to locate as a result.

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Editor’s note: Dr. Pagon is editor-in-chief of the GeneTests–GeneClinics Web site.

Delayed-Onset Heparin-Induced Thrombocytopenia and Cerebral Thrombosis after a Single Administration of Unfractionated Heparin

TO THE EDITOR: Delayed-onset heparin-induced thrombocytopenia, a newly described syndrome of immune-mediated thrombocytopenia that begins several days after heparin therapy has been discontinued,^{1,2} is caused by IgG antibodies that are reactive against platelet factor 4–heparin complexes that activate platelets even in the absence of pharmacologic heparin.¹ The minimal amount of heparin required to initiate this syndrome is unknown.

We describe a 44-year-old woman who received a single injection of unfractionated heparin (5000 units) before undergoing Roux-en-Y gastric bypass for obesity. Previous exposure to heparin was unlikely, since she did not have a history of thrombosis or hospitalizations (other than two hospitalizations for childbirth, which were without complications). The preoperative platelet count was 163,000 per cubic millimeter (Fig. 1). Because of hematemesis and postoperative dilutional thrombocytopenia, no further heparin was given. No blood transfusions or

heparin flushes were administered. The patient was discharged on the fourth postoperative day.

Seven days after surgery, scintillations developed in the left lower visual field, with a persistent occipital headache, followed two days later by mild left-sided hemiparesis and hemisensory loss; a computed tomographic (CT) scan of the head was normal. The platelet count was 23,000 per cubic millimeter eight days after surgery. Left hemiplegia developed, and the left hemisensory deficit worsened. Repeated CT scans showed infarction in the right superior-parietal region (on day 11), with subsequent hemorrhagic conversion (on day 22). Cerebral venous thrombosis was suspected, on the basis of the prominent headache, the subacute progression of the disorder, and the ultimate hemorrhagic infarction (not confirmed by angiography).

Severe thrombocytopenia (nadir platelet count, 7000 per cubic millimeter) and disseminated intravascular coagulation were present, with no apparent