Editorial

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Acute appendicitis still is the most common cause of an intra-abdominal infection. Data suggest that 6-20% of normal people develop acute appendicitis during their lifetime. Appendicitis is a classical example of an acute illness in which the stages of the clinical syndrome are closely correlated with the pathological evolution of the disease. The characteristic pathological finding consists of mucosal disruption with an acute inflammatory infiltrate. The respective pathogenetic role of increased intraluminal pressure, of obstruction, or of bacterial overgrowth, however, is ill defined.

At presentation, abdominal pain, initially often localized in the midabdomen and eventually migrating to the right lower quadrant, is present in 95% of patients. 60-80% become nauseated and vomit. Diarrhea occurs in less than 10% of patients. Simple, uncomplicated appendicitis is not commonly associated with temperature elevation to above 38°C. Localizing abdominal tenderness to both direct and remote pressures represents the typical clinical sign.

However, about one fourth of all patients present with atypical signs and symptoms. Extremes of age, variable appendiceal position within the abdomen, and conditions such as immunosuppression, pregnancy, or Crohn's disease are factors associated with an atypical clinical picture. This can be a taxing diagnostic problem, yet a delay in diagnosis often results in a marked increase in both morbidity and mortality.

Perforation occurs in about 15-20% of patients with acute appendicitis. This is clearly related to delays in presentation and diagnosis and may be due to a more rapid course of disease. Negative appendectomy, on the other hand, may carry a significant morbidity from intestinal obstruction or wound infection or infertility from fimbrial damage. The approximate incidence of such complications after a negative exploration is 15%. At present, the incidence of a normal appendix at exploration for presumed appendicitis is reported to range from < 10% to > 40%. A 'safe' negative appendectomy rate of about 10-20% is now commonly accepted.

Several attempts to improve diagnostic accuracy have been concentrated on refined laboratory studies. Basic laboratory data generally are of no major help and remain adjuncts to the clinical diagnosis: leukocyte counts are normal in < 5% of children with acute appendicitis, but are less sensitive in adults. Other inflammatory signs as well may depend on the duration and the severity of the illness: the acute phase protein (C-reactive protein) may be of some diagnostic value, if an inflammatory process persists for more than 12 h. Measurements of proinflammatory cytokines (e.g., interleukin 1, tumor necrosis factor alpha)

or other products of leukocyte activation (e.g., leukocytic elastase), however, have not yet resulted in an improved diagnosis.

In this issue, Landau et al. [1] report on the diagnostic value of a leukocyte adhesiveness/aggregation test, a simple parameter of leukocyte activation, in a retrospective comparison of patients with and without acute appendicitis. From this study it may be concluded that consideration of this test would be helpful to decrease the rate of unnecessary appendectomies (15.8% in this study).

It seems not surprising that some kind of leukocyte activation can be detected in cases of acute inflammation. White blood cell counts depend on both the activation state with margination and the release from bone marrow, and acute-phase proteins represent a relatively late response. Fast-reacting inflammatory parameters thus may well improve our diagnostic accuracy with respect to the differentiation between unspecific abdominal pain and an acute intra-abdominal inflammation. The indication, however, to operate on an acute inflammation in the right lower abdomen should still be based on classical clinical findings.

Reference: Landau O, Watemberg S, Arber N, Subchi A-A,

Lev D, Hutba S, Klausner J, Aronson M, Berliner S: Retrospective analysis of the benefit of various acute-phase reactants for the diagnosis of acute appendicitis. Dig Surg 1996;13:457-459.

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