Severity and extent of ulcerative colitis: role of C-reactive protein

Traditionally, severity of idiopathic ulcerative colitis (IUC) is assessed by the clinical criteria of Truelove and Witts,1 but these lack precision especially in the definition of severe cases. C-reactive protein (CRP) has been known to be of value in monitoring response to treatment and also correlates with disease activity and helps in management of patients with ulcerative colitis.2,3

All consecutive patients with IUC seen during the period July 2003 to May 2004 were included in the study. The diagnosis was made after clinical, sigmoidoscopic and histologic evaluation. Extent of the disease was determined by colonoscopy and/or double-contrast barium enema (12 patients) and was classified as proctosigmoiditis, left-sided colitis (involvement up to splenic flexure) and pancolitis. Severity of the disease was assessed by the original criteria of Truelove and Witts.1 CRP estimation was done by standard semiquantitative latex agglutination method (Biosystems SA, Barcelona, Spain) technique at admission. It was also estimated in 30 age- and sex-matched healthy volunteers.

Of the 35 patients seen (median age 33.8 years, range 19.4 to 61.8), 17, 6 and 12 patients had se-
vere, moderate and mild disease, respectively. Pancolitis was seen in 11 patients, 15 had left-sided colitis and 9 patients had proctosigmoiditis.

Of 11 patients with pancolitis, 8 had CRP >12 mg/L while only 2 of 9 patients with proctosigmoiditis had CRP >12 mg/L. Of 17 patients with severe IUC, 14 had CRP >12 mg/L, while 10 of 12 patients with mild IUC had CRP <6 mg/L. All normal volunteers had CRP <6 mg/L.

In a study by Fagan et al, mean CRP level was 0 (range 0-15) mg/L in mild disease, 3 (range 0-29) mg/L in moderate IUC, and 12 (range 2-23) mg/L in severe IUC. They concluded that CRP levels correspond closely with clinical and pathological index of relapse, remission and response to therapy. Patients with severe IUC with pancolitis had higher CRP level (median 25 mg/L [range 12-33]) than those with distal disease (median 3 mg/L [range 0-11]). In our study 14 of 17 patients with severe disease had CRP >12 mg/L.

CRP level also is proved to have close relation with the extent of disease in patients with IUC: disease limited to the rectum or even the rectosigmoid rarely causes rise in CRP level unless the disease is particularly severe. In our study only two patients of 9 with proctosigmoiditis had CRP level >12 mg/L, and both had severe disease. Raised CRP >45 mg/L after three days of intensive medical treatment indicated requirement for colectomy in one study.

In conclusion measurement of CRP level is a simple method of assessing disease activity and extent in IUC. CRP level >12 mg/L is indicative of severe and extensive disease.

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References

Follow up of patient with totally resected gastric stromal tumor

We do not understand why Kumar et al offered treatment with imatinib mesylate to their patient with completely resected gastric stromal tumor. Imatinib mesylate is effectively used for non-resectable or metastatic GIST but complete surgical resection is associated with 48%-65% five-year survival.

Malignancy is defined functionally by the presence of invasion of adjoining structures or metastasis, irrespective of size, site and number of mitoses. Based on prognostic categorization the resected gastric GIST in the case reported had low risk of malignancy.

Positron-emission tomography (PET) with 18F-fluoro-2-deoxy-D-glucose is a very useful tool for the follow up of patients receiving imatinib after surgical operation, especially those with metastatic lesions. If PET is not available, we suggest follow up with CT scan instead of ultrasonography as was done in the reported case.

The synchronous occurrence of gastric stromal tumor and colonic adenocarcinoma raises the question of whether this is an incidental association or whether the two are connected by a causal relationship. When the stomach is affected by GIST and another tumor, it is believed that the same stimulus resulted in simultaneous proliferation of different cell lines (lymphocytes, stromal and epithelial cells). We do not know if this mechanism could be responsible for the development of GIST and adenocarcinoma in different parts of the gastrointestinal tract, as in this interesting case.

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References