EDITORIAL

C-REACTIVE PROTEIN AS A DIAGNOSTIC TOOL

K T Woo

SING MED J. 1989; No 30: 10 – 11

C-Reactive Protein (CRP) is an acute phase protein that reflects acute inflammatory reactions within the body (1, 2). It tends to correlate with activity rather than course of the disease and has been reported to be elevated in myocardial infarction, pulmonary embolism, severe thrombosis, sepsis, exacerbation of rheumatic fever, rheumatoid arthritis, acute transplant rejection and any form of surgical trauma (3, 4).

In the April 1988 issue of the journal, Chan et al reports on the use of serum CRP in the diagnosis of renal allograft rejection. Because of its nature as an acute phase reactant, its serum concentration rises in response to tissue injury. Immediately after transplantation, CRP rises and usually falls by the 4th day in the absence of rejection. Thereafter, any rise of CRP in the absence of infection could be indicative of acute rejection (3, 5, 6, 7).

The traditional way of monitoring the renal graft for acute transplant rejection is the detection of raised serum creatinine in the absence of obstruction, infection, drug nephrotoxicity or other causes of graft dysfunction. However, the serum creatinine is a relatively late indicator of rejection, but it has for years been the standard means of monitoring development of transplant rejection. It is readily available in all laboratories, is economical and results can be obtained within the hour.

There have been other attempts (8, 9) to replace the role of serum creatinine including the use of plasma beta-thromboglobulin serum and urine beta-2- microglobulin which have also been shown to diagnose acute rejection earlier than serum creatinine (9, 10).

In the case of the use of serum CRP in Chan et al's paper, the rise in serum CRP preceded a rise of serum creatinine by a mean of 0.95 days. The test has a sensitivity and a specificity approaching 95%.

In the assessment of any diagnostic test one should consider, apart from sensitivity, specificity and coefficient of variation also the cost, and more importantly the practicality and the availability of the test. Some tests come in kits and it would be very expensive to use a whole kit to perform a single test, which very often means that one has to wait for days to collect 20 test samples before making it worthwhile to run a kit.

Department of Renal Medicine
Singapore General Hospital
Outram Road
Singapore 0316

K T Woo, MBBS (S'pore), M Med (Int Med), FRACP,
Renal Physician

Nowadays, with new technology, some of these problems may be of the past. Using the nephelometer it is possible to test one sample of serum by itself together with a control. The nephelometer is a versatile instrument that can measure specific serum protein, perform drug assay as well as other routine biochemical tests like urea and creatinine. All factors considered it would still appear that the days of serum creatinine as an indicator of transplant rejection are far from over yet, bearing in mind that the first report on the role of CRP in diagnosing rejection was published in 1978, ten years ago and some recent reports (3, 5, 6, 7) are still contradictory.

CRP (4) has also been shown to be involved in the response of platelets to various stimuli during inflammation. It is also an activator of the complement system. In addition, it shows certain functional similarities with the immunoglobulins and binds to T lymphocytes and inhibits certain of their reactivities.

IgA nephritis is an immune-complex glomerulonephritis associated with suppressor T cell abnormality and increased platelet aggregation (11, 12). Its pathogenecity seems to comprise an assortment of as yet unlinked markers, mainly immunological. These markers are still not clinically useful in monitoring disease activity. It was therefore thought that CRP may be a marker which may link activity of an immune complex nephritis associated with abnormal T cell activity, increased platelet aggregation, elevated serum IgA and beta-2-microglobulin.

We therefore studied 20 healthy subjects and 98 patients with IgA nephritis for the presence of CRP in their sera by means of the CRP Wellcotest available from Burroughs and Welcome. The results were disappointing. Among the 20 healthy control subjects, there were 2 with positive CRP in their sera, an incidence of 10%. In the 98 patients with IgA nephritis, there were 8 with CRP present (8/98 or 8%). There was no correlation between CRP and any of the clinical and laboratory indices in the patients. CRP does not appear to be a significant marker in patients with IgA nephritis.

Boey et al (13) evaluated the role of serum CRP as a diagnostic tool in patients with systemic lupus erythematosus. They found no correlation between serum CRP and lupus activity. However CRP was found to be useful in differentiating between infection and lupus activity as CRP levels are increased in infection.

CRP therefore appears to be a nonspecific marker. It is perhaps a sophisticated, albeit more expensive way of performing the ESR.

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