Rise in serum C reactive protein after hip and knee arthroplasties in patients with rheumatoid arthritis

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Abstract

Objective—Serum C reactive protein (CRP) concentration was evaluated in patients with rheumatoid arthritis (RA) undergoing total hip arthroplasty (THA) or total knee arthroplasty (TKA) to ascertain the postoperative CRP response.

Methods—Thirty seven consecutive patients with RA who had undergone THA or TKA were included in the study. The CRP concentration was measured in every patient once preoperatively and every other day for one week postoperatively.

Results—The peak median CRP concentration (94 mg/l) was achieved on the first and second day postoperatively and was seven times higher than the median preoperative concentration (13 mg/l). CRP declined to the preoperative concentration in about one week. The rise of the CRP concentration was significant (p< 0.001). No infection was encountered in this series.

Conclusion—A rapid rise in the postoperative CRP concentration is normal in patients with RA treated by THA or TKA. The CRP concentration decreases to the preoperative value in about one week. Serial CRP measurements, including at least one preoperative measurement, are needed when the clinical significance of the postoperative CRP values is evaluated. When the postoperative CRP concentration remains raised for several days compared with the preoperative value, or even rises, it may indicate the presence of a complication in these patients. (*Ann Rheum Dis* 2001:60:275–277)

Serum C reactive protein (CRP) is an acute phase reactant most commonly used as an indicator of infection. In surgery it is of importance in detecting postoperative infections. In several studies a rise in CRP concentration during the first few postoperative days has been documented and is regarded as usual, as is the rapid decrease in CRP to normal. The fall in CRP concentration may be delayed when postoperative complications are present.¹⁻³ However, in patients with rheumatoid arthritis (RA) not only operative treatment and possible infection but also the RA itself may have an influence on the rise in CRP. It is thus important to determine whether RA has an effect on the postoperative CRP behaviour.⁴

The present study aimed at evaluating the CRP response in patients with RA after total hip arthroplasty (THA) or total knee arthroplasty (TKA).

Patients and methods

This study was carried out at the Rheumatism Foundation Hospital, Heinola, Finland between 1 January and 30 October 1999. The series comprised 37 consecutive patients (28 women, nine men) with RA fulfilling the American Rheumatism Association 1987 criteria.⁵ They all received a hip or knee replacement. Their mean age was 60 (34–79) years and the mean duration of the disease was 25 (3–43) years.

THA was performed on 16 patients and TKA on 21. The procedure was a primary replacement in 28 patients and a revision in nine. All received prophylactic anticoagulation with 20 mg or 40 mg of enoxaparin once daily, the first dose being given on the evening before surgery and lasting for the period of stay in hospital. Antibiotic prophylaxis consisted either of cloxacillin 500 mg or cefuroxime 750 mg given three times a day for two days. Regional (epidural or spinal) or general anaesthesia was used in both THA and TKA. The bloodless field technique was used in TKA operations with a pneumatic torniquet pressure of 100 mm Hg above the preoperative systolic arm blood pressure. Preoperative planning was used for component sizing. The surgical approaches were medial parapatellar retinacular incision in TKA and, the lateral or posterior approach in THA. Patients with TKA were treated postoperatively with compressive dressing and a continuous passive motion device. Early weight bearing was allowed in association with cemented arthroplasty if no bone graft was applied.

Routine preoperative laboratory, chest radiographic, and electrocardiographic evaluations were made in all cases. Blood samples for CRP analysis were obtained once preoperatively and postoperatively every other day for one week.

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Figure 1 Box and whisker plot showing rapid rise and decrease in CRP concentrations. The box part covers the interquartile range and the median; the whiskers extend to include all but outliers, these being indicated separately.

The CRP concentration was measured by the Randox, United Kingdom, immunoturbidimetric assay, where the principle is that the sample is allowed to react with a specific antiserum to form a precipitate which is measured turbidimetrically at 340 nm. By constructing a standard curve from the absorbances of standards, the CRP concentration in the sample can be determined.⁶ Preoperative leucocyte and platelet concentrations were also assessed. CRP values ≤ 10 mg/l were considered normal and those >10 mg/l abnormal.

The statistical significance of the rise and decline in CRP concentration was calculated with Wilcoxon's signed ranks test and Page's test for trend. Correlation between preoperative leucocyte or platelet values and the postoperative CRP rise was assessed with Spearman's coefficient.

Results

The form of the postoperative CRP response curve was similar in all patients, though the amplitude of the curve varied between them. The peak CRP value was achieved on the first or second postoperative day and the values decreased to the preoperative concentration in about a week (fig 1). The median preoperative CRP was 13 mg/l (0-127) and the peak median value 94 mg/l (10-294). At the seventh and eighth postoperative day the median CRP concentration was 18 mg/l (5-110). The rise of CRP from the preoperative concentration to the first and second postoperative day value was significant (p<0.001). No statistical significance was found between the preoperative CRP concentration and the postoperative CRP response concentration ($r_{=}=-0.11$ (95%) CI -0.42 to 0.23)). No correlation was found between preoperative leucocyte or platelet values and the CRP rise during the first two postoperative days (r_s <0.01). None of the patients had bacterial infection after the operation.

Discussion

Total hip and knee replacements are fairly common surgical procedures, often with excellent outcome. Postoperative infection is one of their most serious complications, and must be detected and treated without delay. CRP is used as a routine test for bacterial infections even after surgery. Unfortunately, it is not optimal during the immediate postoperative period because the concentration of serum CRP is usually significantly increased after major operations—for example, arthroplasties in patients with osteoarthritis.^{2 3}

Because patients with active RA often have raised CRP concentrations without any infections, it is essential to know precisely how the CRP concentration behaves in these patients in the postoperative period in order to avoid unnecessary treatments but to provide treatment when necessary. To our knowledge, the only previous article concerning postoperative CRP response after THA or TKA in patients with RA is that published by Maury et al.7 In that study CRP concentrations were measured in 40 patients with RA after THA, TKA, or knee synovectomy, a rapid rise and decline in concentrations being recorded postoperatively; however, the writers do not discuss the clinical significance of their results.

In our study the rise in CRP peaked on the first or second postoperative day in patients with RA after hip or knee replacement. The median peak concentration was seven times higher than the preoperative median. Readings reverted to the preoperative concentration in about one week. The form of response pattern of CRP was similar in every patient, though the concentrations varied. This finding is in accordance with previous results obtained in patients with osteoarthritis and RA.^{1-3 7} The preoperative CRP concentration had no influence on the postoperative CRP response value.

It would seem that the presence of RA has no major influence on the postoperative CRP response. Larsson *et al* state that a brief postoperative CRP rise would result solely from tissue damage during surgery, when no bacterial infection has occurred.³ This would seem also to be an issue in the CRP response of patients with RA. As no postoperative infections occurred in the present series, this study yields no data on postoperative CRP response in infected arthroplasties.

A significant postoperative rise in CRP concentration is a normal response to surgical trauma and does not indicate complications. Immediately after the operation no antibiotic treatment is needed despite high CRP concentrations, provided that the clinical situation is otherwise satisfactory. On the other hand, anti-inflammatory treatment with glucocorticoids and non-steroidal anti-inflammatory drugs will inhibit the rise of fever and other signs of infection, so that clinical evaluation presupposes some experience. CRP measurements, including at least one preoperative measurement, are needed when the clinical significance of the postoperative CRP values is evaluated. If postoperative CRP values continue to be raised for several days compared with the preoperative value, or even show a tendency to rise, the reason for this (for example, an infection, a haematoma) should be carefully evaluated.

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