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Effect of daily corticosteroid treatment on CRP response to hip or knee replacement in patients with RA

Serum C reactive protein (CRP) is an acute phase reactant which may be continuously increased in patients with persistently active rheumatoid arthritis (RA),1 or raised only temporarily to a high concentration for a few days as a normal response to uncomplicated hip or knee replacement in patients with osteoarthritis or RA.² CRP usually decreases in patients with RA when inflammatory activity is treated with daily low dose corticosteroid. This prompts the question whether the CRP response to hip or knee replacement is decreased in patients with RA taking a daily low dose of oral corticosteroid compared with those not taking corticosteroid. This is an important issue because CRP is used as an index to indicate postoperative complications. In this letter we compare the CRP response to hip or knee replacement in two groups of patients with RA: those taking and those not taking oral low dose corticosteroid.

Sixty patients (47 women, 13 men) fulfilling the American Rheumatism Association 1987 criteria for RA,4 treated at the Rheumatism Foundation Hospital, Heinola, in 1999, underwent hip or knee replacement. Fifty two patients were seropositive. The group receiving prednisolone comprised 44 patients, mean age 62 (SD 8.5) years. The prednisolone doses were as follows: four patients received <5 mg daily, 37 had 5-10 mg daily, and three had 12.5-30 mg daily. The patient group not receiving prednisolone comprised 16 patients, mean age 59

The CRP concentration was measured by the Randox, United Kingdom, immunoturbidimetric assay. The magnitude of the CRP response was measured by assessing the difference between measurements taken preoperatively and during the first one to two days postoperatively (the time of the peak CRP level³) in both patient groups. The CRP responses in the respective groups were compared and statistically evaluated with the Mann-Whitney U test. In the group not receiving prednisolone the preoperative median CRP level was 12 (interquartile range (IQR) 5-26) mg/l, and at day 1 or 2 postoperatively the median CRP had risen to 80 (IQR 53-112) mg/l. In the group in which patients were taking prednisolone the preoperative median was 14 (IQR 6-38) mg/l, the

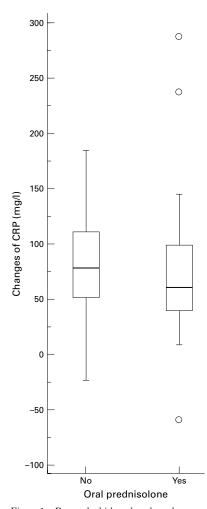


Figure 1 Box and whisker plots show the postoperative C reactive protein (CRP) response to hip or knee replacement in 16 patients with RA not receiving prednisolone and 44 patients with RA receiving continuous prednisolone.

postoperative median 62 (IQR 41-100) mg/l. The difference in CRP response to the operation between the groups was not significant, p=0.15 (fig 1). None of the patients had bacterial infection or substantial haematoma after the operation.

The rise in CRP concentration in response to hip or knee replacement was slightly, but not significantly, smaller in patients with RA receiving than in those not receiving prednisolone. Increased CRP concentration was a normal phenomenon in the first few days after hip or knee replacement in these patients with RA and was not altered by low dose prednisolone treatment. This study affords no information as to the CRP response in the presence of postoperative complications, because no such case was encountered. However, we recommend further measures if the CRP concentration remains raised for several days postoperatively and does not decrease steadily.

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Lumbar spondylodiscitis secondary to Enterobacter cloacae septicaemia after extracorporeal shock wave lithotripsy

Infections of the lumbar spine may affect either the intervertebral disc or the vertebral body. Most infections of the intervertebral disc occur as an extension of vertebral osteomyelitis or direct inoculations during diagnostic or surgical procedures, or include urinary tract infections and septicaemia. This paper reports a case of L5-S1 spondylodiscitis secondary to Enterobacter cloacae septicaemia after extracorporeal shock wave lithotripsy (ESWL).

A 52 year old man presented with side pain, pollakiuria, haematuria, and nocturia. He had been treated with ciprofloxacin for acute pyelonephritis and nephrolithiasis as an outpatient. One week later, the patient was admitted to hospital by the urology department with symptoms of left side pain, fever, chills, shaking, and dysuria. Right renal and right ureter distal lithiasis and right hydronephrosis due to the lithiasis were diagnosed. One week after ESWL the patient was sent to the physical medicine and rehabilitation clinic with chills, shaking, high fever, and low back pain complaints. Lumbar movements were found to be restricted. There was an increase in severe pain at rest. The patient could not stand or walk. No neurological deficit was present. Body temperature was 39°C, pulse 110 beats/min, blood pressure 130/70 mm Hg, breathing 20 breaths/min.

Laboratory findings were as follows: haemoglobin 133 g/l, packed cell volume 0.31, white blood cells 18×10⁹/l, platelets 316×10⁹/l, erythrocyte sedimentation rate 110 mm/1st h, antistreptolysin O 25 IU, C reactive protein 12.3 mg/l, rheumatoid factor negative. Urea was 14 mmol/l of urea, creatinine 170 µmol/l. Glucose and electrolytes were normal and serum aspartate aminotransferase was 80 U/l, serum alanine aminotransferase 44 U/l, lactate dehydrogenase 321 U/l, total bilirubin 22 μmol/l, direct bilirubin 10 µmol/l. A considerable number of leucocytes and erythrocytes were noticed in urine microscopy. Enterobacter cloacae was isolated from blood and urine. The isolated pathogen was sensitive to ceftriaxone and amikacin.