Correspondence

The effects of indomethacin on serum gastrin concentrations

Sir,

It is known that some patients with rheumatoid arthritis have a raised serum gastrin concentration. However, it has been difficult to separate the effects of the disease itself from those due to drug administration in the causation of raised gastrin levels. We have recently studied the effects of indomethacin on serum gastrin concentrations in both volunteers and patients.

Six healthy male volunteers were fasted overnight and 2 blood samples for basal serum gastrin levels were taken. Following a standard protein meal, further blood samples were taken every 10 minutes for the first hour and every 15 minutes for the second hour. Indomethacin (50 mg) or matching placebo were given by mouth in random order, half an hour before the first blood sample was taken, in 2 separate studies. In 4 of the 6 volunteers after 8 days on 25 mg q.d.s. indomethacin, basal and post protein diet gastrin were also determined, and indomethacin plasma concentrations were measured 2 to 3 hours after the last morning dose. In 25 patients with rheumatoid arthritis, treated in the long term with 25 mg t.d.s. indomethacin, a single blood sample for gastrin and indomethacin was taken 2 to 3 hours after breakfast.

Gastrin was measured in serum by the method of Dockray and Taylor and plasma indomethacin by gas liquid chromatography.

The results are shown in Table 1 and indicate that indomethacin caused no significant differences in the gastrin response to a standard test meal. In 24 of the patients serum gastrin concentrations were in the normal range of 15–45 pmol/l. One patient had serum gastrin concentrations in excess of 600 pmol/l, and she was later shown to have achlorhydria.

Plasma indomethacin concentrations were 415 ± 156 ng/ml in the volunteers at the end of the gastrin study and 326 ± 81 ng/ml in the patients. These results are in the usual therapeutic range and indicate compliance with the therapeutic regimen.

Table 1 Mean basal gastrin values (pmol/l) and peak increments in serum gastrin after standard protein meal

<table>
<thead>
<tr>
<th>Placebo</th>
<th>Indomethacin</th>
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<tbody>
<tr>
<td>Basin gastrin</td>
<td>Peak increment</td>
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<tr>
<td>Single dose volunteer</td>
<td>13 ± 1.9</td>
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<tr>
<td>Multiple dose volunteer</td>
<td>13.2 ± 3.0</td>
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Our results show that indomethacin has no effect on serum gastrin concentrations, and it is unlikely, therefore, that the indigestion seen with indomethacin is mediated via a rise in serum gastrin. Our studies are in agreement with those of Rooney et al., performed without the use of a standard protein meal, and the uncontrolled observations of Cutarelli et al., using phenylbutazone.


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References


Pyogenic arthritis presenting as a ruptured popliteal cyst

Sir,

I read with interest the paper by Drs Stewart, Swinson, and Hardinge but should like to draw your attention to the erroneous statement that rupture of a popliteal cyst is a frequent complication of rheumatoid arthritis. This statement is frequently repeated in literature more from habit than belief, I suspect. Enlargement of the cysts in the popliteal fossa is extremely common in rheumatoid arthritis, but they very rarely rupture. It is the knee joint itself which is the source of the leak. This, if repeated, leads to the development of a calf cyst, which is probably what Baker was referring to, and is of quite different origin to the popliteal cyst, which is...
merely the enlargement of an already formed synovial bursa behind the knee.\textsuperscript{2}

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References
\textsuperscript{1} Stewart I M, Swinson D R, Hardinge K. Pyogenic arthritis, presenting as a ruptured popliteal cyst. \textit{Ann Rheum Dis} 1979; 38, 181–182.

Sir,
Thank you for allowing us to reply to the letter from Dr Myles. We would agree that to say a ruptured popliteal cyst is frequent in rheumatoid arthritis is something of an exaggeration, but in fact nowhere in our paper do we state that this is a frequent occurrence. With reference to Dr Myles's point that popliteal cysts very rarely rupture, we would like to draw your attention to Dr Myles's own paper, in which he states that '... in some cases the popliteal cyst was found to be leaking.' We would certainly agree that a popliteal cyst is a direct extension of the knee joint and a calf cyst is the result of extravasation of fluid downwards. However, there is no doubt that calf cysts can arise directly from a leaking or ruptured popliteal cyst (see Figs. 1 and 2).

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Fig. 1 Popliteal cyst.

Fig. 2 Popliteal cyst leaking into large calf cyst (same knee, same patient, sequential arthrography).