

## LETTERS TO THE EDITOR

### Effect of long term intramuscular gold therapy on the seroprevalence of *Helicobacter pylori* in patients with early rheumatoid arthritis

*Helicobacter pylori* is an important causative factor in chronic gastritis and peptic ulcer disease.<sup>1</sup> Heavy metals such as bismuth salts are used to eradicate *H pylori* infection;<sup>2</sup> gold treatment is also considered to possess antibacterial activity and sodium aurothiomalate has been shown to possess marked bactericidal activity against *H pylori* in vitro,<sup>3</sup> but sulphasalazine, another common anti-rheumatic drug used in rheumatoid arthritis (RA), does not affect the prevalence of *H pylori* infection.<sup>4</sup> The effect of gold treatment on *H pylori* in RA patients remains controversial, however.<sup>5-7</sup> We examined the long term effect of intramuscular gold on *H pylori* by evaluating serum IgA and IgG antibodies to the organism in a prospective study of patients with early RA<sup>8</sup> and without any previous antirheumatic treatment.

Initially, 87 patients with early RA (mean age 46.3 years, range 19-65; mean duration of disease 7.6 months, range 2-12) were attending a prospective three year follow up. Selection of the first disease modifying anti-rheumatic drug (DMARD) was adjusted to individual requirements. From the initial patient group of 87, we enrolled in the study five men and 15 women who were able to continue intramuscular gold throughout the three year follow up, and four men and 16 women who were treated with sulphasalazine during the corresponding period (table 1).

Samples for measuring circulating IgA and IgG antibodies to *H pylori* were taken at months 0, 9, and 36; concurrent use of non-steroidal anti-inflammatory drugs (NSAIDs), antiulcer drugs and antibiotics was recorded. IgA and IgG class antibodies to *H pylori* were measured by an enzyme immunoassay method<sup>9</sup> (Pyloriset<sup>®</sup>EIA-A, Pyloriset<sup>®</sup>EIA-G, Orion Diagnostica, Espoo, Finland). The lower limits for increased titres (expressed as reciprocals) were 600 for both IgA and IgG

Table 1 Demographic data for patients with early rheumatoid arthritis treated with intramuscular gold or sulphasalazine

	Treatment group	
	Intramuscular gold (n = 20)	Sulphasalazine (n = 20)
Gender (M/F)	5/15	4/16
Age (yr)	42.9 (22-59)	40.9 (19-61)
Disease duration (months)	8.3 (4-12)	7.6 (2-12)
RF positive (%)	70	60

Values are number or mean (range). RF = Rheumatoid factor; Waaler-Rose  $\geq 1:64$ . No significant differences between treatment groups.

Table 2 Seroprevalence of *Helicobacter pylori* during a three year follow up of patients with early rheumatoid arthritis

	Month 0		Month 9		Month 36	
	Gold (n = 20)	SASP (n = 20)	Gold (n = 20)	SASP (n = 20)	Gold (n = 20)	SASP (n = 20)
Positivity to IgA <i>H pylori</i> antibodies (%)	45	20	45	20	40	20
Positivity to IgG <i>H pylori</i> antibodies (%)	45	15	40	15	35	15

SASP = Sulphasalazine.

antibodies.<sup>9</sup> Statistical analysis was performed using the  $\chi^2$  test with Yates' correction or Wilcoxon's signed rank test and Student's *t* test.

At entry to the study, no significant differences between the clinical data of the two groups of RA patients was observed (data not shown). None of the patients had symptoms of peptic ulcer and none used antiulcer drugs during the three year follow up. Short term antibiotic treatment, mostly for upper respiratory infections, was used by 10% of patients in both groups during the follow up.

At month 0, before gold or sulphasalazine treatment started, 32% (13/40) of patients showed serological evidence of *H pylori* infection (IgG positive). Initially, more patients who subsequently received gold treatment had serological evidence of *H pylori* infection than was observed among those treated with sulphasalazine (table 2). In the subgroup of patients seropositive for *H pylori*, one patient with gold therapy showed a significant decline (more than 50%) of both IgA and IgG anti-*H pylori* titres at 36 months, indicating eradication of the *H pylori* bacteria,<sup>10</sup> while none of the patients in the sulphasalazine group exhibited such a decline.

In an earlier report, RA patients who underwent at least six months of intramuscular gold treatment showed lower IgA and IgG antibody titres against *H pylori* compared with RA patients receiving antimarial drugs.<sup>5</sup> In contrast, in a study of unselected RA patients, no reduction in *H pylori* seroprevalence was found in patients treated with gold compounds.<sup>6</sup> Recently, intramuscular gold therapy for 12 months was not found to influence the serological markers for *H pylori* infection;<sup>7</sup> our data from this three year follow up confirm this finding. No clinical evidence was observed in favour of a relationship between *H pylori* seropositivity and NSAID induced gastric damage.

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### Vertebral fracture induced by chronic contained rupture of aortic aneurysm

We report an exceptional case of a vertebral fracture. A 64 year old man had attended our rheumatology clinic since 1982 for idiopathic osteoporosis. He had multiple vertebral fractures (D7 and D8 in 1982, L1 in 1983 and L2 in 1991). In January 1992 he presented with acute lumbar pain, radiating to the right leg. A non-pulsatile abdominal mass was palpable and peripheral pulses were absent. Neurological examination was normal and laboratory results unremarkable. However, a new fracture of L4 and an abdominal soft tissue mass were noted on radiography (figure). Computed tomography (CT) scan revealed a chronic contained aortic aneurysm 17 cm in diameter extending from the level of L1 to L4. Compression of the inferior vena cava, displacement of the right kidney, atrophy of the right psoas and extensive erosion of the anterior wall of L4 with crush fracture were seen. L4 radiculopathy was documented on EMG.