Small Bowel Infarction in Association with Giant Cell Arteritis

Sir—We read with interest the article by Phelan et al. [1] on small bowel involvement in giant cell arteritis and would like to report a case of polymyalgia rheumatica and giant cell arteritis in which small bowel infarction occurred due to mesenteric vasculitis one month after discontinuation of steroid therapy.

A 60-yr-old man was diagnosed as having polymyalgia rheumatica and was commenced on oral steroids. His symptoms and ESR remained well controlled on low dose steroids for 22 months. He then started complaining of intermittent bitemporal headache and blurring of vision. The ESR at this stage was 27 mm/h. He was seen at the Ophthalmology Department but no abnormality was detected on fundal examination. His headache and blurring of vision settled within a month. Because of lack of symptoms and a normal ESR, steroid therapy was stopped after 2 yr of treatment.

Two weeks after discontinuing steroids, he started feeling tired, his headache returned and he also had a transient episode of left-sided weakness for which he did not go to see his general practitioner. One month after discontinuing steroid therapy, he was admitted as an emergency with severe left iliac fossa pain. He was apyrexial, but had generalized abdominal tenderness with absent bowel sounds. An erect abdominal radiograph showed multiple fluid levels. At laparotomy, 16 cm of gangrenous small bowel was resected and an end to end anastomosis made. Histology of the bowel showed haemorrhagic infarction of the small bowel with multiple ulceration of the mucosa. Some of the mesenteric arteries supplying the area were thrombosed and there was destruction of muscular media and internal elastic lamina with diffuse infiltration with inflammatory cells, but no giant cells were seen. The initial post-operative recovery was unremarkable, although his headache and feeling of ill health persisted. His ESR at this stage was noted to be elevated at 68 mm/h. In view of his symptoms, elevated ESR and biopsy findings, a diagnosis of giant cell arteritis was made and he was commenced on oral steroids. His symptoms and ESR became normal within 2 weeks of commencing therapy and he has remained asymptomatic on low dose steroids for 4 yr.

Polymyalgia rheumatica and giant cell arteritis frequently occur in the same patient [2], and polymyalgia rheumatica usually precedes the onset of giant cell arteritis [3]. Our patient developed symptoms consistent with giant cell arteritis 22 months following polymyalgia rheumatica. This underlines the importance of considering the co-existence of giant cell arteritis in patients with polymyalgia rheumatica while on steroids and it would be prudent to continue steroids for longer than 2 yr in such patients despite a normal ESR. The treatment of uncomplicated giant cell arteritis is usually discontinued within 2 yr of commencing therapy as recurrence of giant cell arteritis is considered to be rare [3]. However, there is no data regarding the duration of steroid therapy required in patients with giant cell arteritis developing life threatening complications such as small bowel infarction. Although, our patient has remained asymptomatic and his ESR has remained normal, we have kept him on low dose steroids for the last 4 yr.

A. O. ADEBAJO*, P. J. CHARLES†, B. L. HAZLEMAN*, R. N. MAIN†
*Rheumatology Research Unit, Addenbrooke’s Hospital, Cambridge; †The Kennedy Institute of Rheumatology, London
Accepted 14 April 1993

Murphy et al., in which the authors make reference to our recent study on antineutrophil cytoplasmic antibodies (ANCA) and infection [2]. We wish to clarify their comments on our study by pointing out that whilst we found IgM antibodies to ANCA in three patients with tuberculo-sis (TB) and one with malaria, only one case (a patient with TB) possessed IgG antibodies to ANCA. This suggests that the isotype specificities of ANCA are important.

There are at least three other reports of a positive ANCA in association with TB [3-5] and it is possible that a positive ANCA may on occasion precede the development of clinical features of vasculitis in TB patients. Interestingly at least one centre has reported cases of a positive ANCA in association with TB but without overt vasculitis [5].

Our current view is that IgG antibodies to ANCA are highly specific for the classical vasculitides. The possibility of TB being misdiagnosed as Wegener’s granulomatosis [4] particularly with IgM antibodies to ANCA must, however, be borne in mind.

A. O. ADEBAJO*, P. J. CHARLES†, B. L. HAZLEMAN*, R. N. MAIN†
*Rheumatology Research Unit, Addenbrooke’s Hospital, Cambridge; †The Kennedy Institute of Rheumatology, London
Accepted 14 April 1993


This letter was shown to Dr Murphy and colleagues who reply:

Sir—We note the comments made by Adebajo et al. with regard to their study. We agree that isotype specificity may be important in determining the clinical relevance of ANCA and infection [2]. We wish to clarify their comments on our study by pointing out that whilst we found IgM antibodies to ANCA in three patients with tuberculo-sis (TB) and one with malaria, only one case (a patient with TB) possessed IgG antibodies to ANCA. This suggests that the isotype specificities of ANCA are important.

There are at least three other reports of a positive ANCA in association with TB [3-5] and it is possible that a positive ANCA may on occasion precede the development of clinical features of vasculitis in TB patients. Interestingly at least one centre has reported cases of a positive ANCA in association with TB but without overt vasculitis [5].

Our current view is that IgG antibodies to ANCA are highly specific for the classical vasculitides. The possibility of TB being misdiagnosed as Wegener’s granulomatosis [4] particularly with IgM antibodies to ANCA must, however, be borne in mind.

A. O. ADEBAJO*, P. J. CHARLES†, B. L. HAZLEMAN*, R. N. MAIN†
*Rheumatology Research Unit, Addenbrooke’s Hospital, Cambridge; †The Kennedy Institute of Rheumatology, London
Accepted 14 April 1993

Small Bowel Infarction in Association with Giant Cell Arteritis

Sir—We read with interest the article by Phelan et al. [1] on small bowel involvement in giant cell arteritis and would like to report a case of polymyalgia rheumatica and giant cell arteritis in which small bowel infarction occurred due to mesenteric vasculitis one month after discontinuation of steroid therapy.

A 60-yr-old man was diagnosed as having polymyalgia rheumatica and was commenced on oral steroids. His symptoms and ESR remained well controlled on low dose steroids for 22 months. He then started complaining of intermittent bitemporal headache and blurring of vision. The ESR at this stage was 27 mm/h. He was seen at the Ophthalmology Department but no abnormality was detected on fundal examination. His headache and blurring of vision settled within a month. Because of lack of symptoms and a normal ESR, steroid therapy was stopped after 2 yr of treatment.

Two weeks after discontinuing steroids, he started feeling tired, his headache returned and he also had a transient episode of left-sided weakness for which he did not go to see his general practitioner. One month after discontinuing steroid therapy, he was admitted as an emergency with severe left iliac fossa pain. He was apyrexial, but had generalized abdominal tenderness with absent bowel sounds. An erect abdominal radiograph showed multiple fluid levels. At laparotomy, 16 cm of gangrenous small bowel was resected and an end to end anastomosis made. Histology of the bowel showed haemorrhagic infarction of the small bowel with multiple ulceration of the mucosa. Some of the mesenteric arteries supplying the area were thrombosed and there was destruction of muscular media and internal elastic lamina with diffuse infiltration with inflammatory cells, but no giant cells were seen. The initial post-operative recovery was unremarkable, although his headache and feeling of ill health persisted. His ESR at this stage was noted to be elevated at 68 mm/h. In view of his symptoms, elevated ESR and biopsy findings, a diagnosis of giant cell arteritis was made and he was commenced on oral steroids. His symptoms and ESR became normal within 2 weeks of commencing therapy and he has remained asymptomatic on low dose steroids for 4 yr.

Polymyalgia rheumatica and giant cell arteritis frequently occur in the same patient [2], and polymyalgia rheumatica usually precedes the onset of giant cell arteritis [3]. Our patient developed symptoms consistent with giant cell arteritis 22 months following polymyalgia rheumatica. This underlines the importance of considering the co-existence of giant cell arteritis in patients with polymyalgia rheumatica while on steroids and it would be prudent to continue steroids for longer than 2 yr in such patients despite a normal ESR. The treatment of uncomplicated giant cell arteritis is usually discontinued within 2 yr of commencing therapy as recurrence of giant cell arteritis is considered to be rare [3]. However, there is no data regarding the duration of steroid therapy required in patients with giant cell arteritis developing life threatening complications such as small bowel infarction. Although, our patient has remained asymptomatic and his ESR has remained normal, we have kept him on low dose steroids for the last 4 yr.

W. U. HASSAN, T. J. DAIMOND

The Royal Infirmary, Sunderland SR4 7RJ
Accepted 15 July 1993

Correspondence to W. Hassan, Department of Rheumatology, The Leicester Royal Infirmary, Leicester LE1 5WW.