measures, he was being maintained on 7 mg of prednisolone/day (with appropriate bone protection) at the time of writing this letter, as his bowel symptoms flared each time the dose was reduced below this level. Methotrexate was being considered as a steroid-sparing agent to control his bowel disease.

Extractcutaneous and visceral manifestations are increasingly recognized in patients with EF. Colitis has, however, been reported in such patients only twice, and EC only once. The only other EF patient who developed EC also had multisystem involvement with pericarditis, thyroiditis and monoclonal gammopathy [6]. In the other patient, the features of colitis were considered to be consistent with Crohn’s disease [7]. In our patient, EC occurred as an isolated visceral manifestation several months after the diagnosis of EF was made. It is difficult to prove that this association was not coincidental, but it may be possible to speculate that the mechanisms underlying the association are immune mediated.

Although the exact immunological mechanisms have not been identified, there is some evidence for the role of T lymphocytes in the causation of EC. In a murine model of oral antigen-induced diarrhea associated with colonic inflammation, colonic T cells have been shown to transfer the disease to naive mice through a STAT6-dependent mechanism [8]. The occurrence of hypergammaglobulinaemia and inflammatory cell infiltrate in the fascia and the association with autoimmune haematological disorders, Sjögren’s syndrome and thyroiditis support the role for immune-mediated mechanisms in the causation of EF. It should also be noted that EC in adults could be secondary to drug reactions and parasitic infections, and toxins such as l-tryptophan [9] and infection by Borrelia burgdorferi [10] have been reported as causative agents in EF.

In view of the rarity of this syndrome, evidence-based management drawn from controlled trials cannot be offered for patients with EF. Removal of the inciting agent (if one is identified) is the most important measure. Prednisolone is often used as most patients show partial or complete response. We, however, did not feel that prednisolone helped the EF component in our patient. Hydroxychloroquine, methotrexate and cimetidine have also been reported to be beneficial, of which the latter was used in our patient. Cimetidine is thought to act through blockage of T cells have been shown to transfer the disease to naive mice through a STAT6-dependent mechanism [8]. The occurrence of hypergammaglobulinaemia and inflammatory cell infiltrate in the fascia and the association with autoimmune haematological disorders, Sjögren’s syndrome and thyroiditis support the role for immune-mediated mechanisms in the causation of EF. It should also be noted that EC in adults could be secondary to drug reactions and parasitic infections, and toxins such as l-tryptophan [9] and infection by Borrelia burgdorferi [10] have been reported as causative agents in EF.

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In conclusion, it is important to appreciate the widening spectrum of EF and carefully look for internal organ involvement. The pathogenesis of the association between EF and EC is unknown, but is possibly immune mediated. Treatment for these patients is largely based on only anecdotal evidence. It is difficult to prove that this association was not coincidental, but it may be possible to speculate that the mechanisms underlying the association are immune mediated. Although the exact immunological mechanisms have not been identified, there is some evidence for the role of T lymphocytes in the causation of EC. In a murine model of oral antigen-induced diarrhea associated with colonic inflammation, colonic T cells have been shown to transfer the disease to naive mice through a STAT6-dependent mechanism [8]. The occurrence of hypergammaglobulinaemia and inflammatory cell infiltrate in the fascia and the association with autoimmune haematological disorders, Sjögren’s syndrome and thyroiditis support the role for immune-mediated mechanisms in the causation of EF. It should also be noted that EC in adults could be secondary to drug reactions and parasitic infections, and toxins such as l-tryptophan [9] and infection by Borrelia burgdorferi [10] have been reported as causative agents in EF.

In conclusion, it is important to appreciate the widening spectrum of EF and carefully look for internal organ involvement. The pathogenesis of the association between EF and EC is unknown, but is possibly immune mediated. Treatment for these conditions is largely based on only anecdotal evidence.

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The authors have declared no conflicts of interest.

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About the difficulty in interpreting ultrasonographic images of temporomandibular joint

Sir, We have read with interest the paper ‘A comparison of ultrasonography and magnetic resonance imaging in the evaluation of temporomandibular joint involvement in rheumatoid arthritis and psoriatic arthritis’ by Melchiorre et al. [1].

We would like to dwell upon the method of ultrasonographic (US) examination, unfortunately not described in the paper, for a few observations that, in our opinion, could help to better understand the images obtained with this technique.

The echographic study of the temporomandibular joint (TMJ) consists of different scans in coronal, axial and oblique plans [2, 3]. The exploration of the bone profile of the mandibular condyle can give very precise information about the condition of this structure, which is visualized through a window of 120° in the coronal scans and 40° in the axial scans. Further information can be obtained with the dynamic scans: they allow us to observe the condyle posterosuperior surface, to measure the anterior translation of the condyle, having as a landmark the tragus cartilage, and finally to study the articular and peri-articular soft tissues, included part of the morphology and the movement of the disc (Figs 1 and 2).

As for the study of the articular space in the axial scans, our experience suggests that the image of the capsulosynovial thickening should be considered as the slightly echoic structure between
the convex bone profile of the condyle and the convex hyperechoic line of the capsular external surface.

In the axial scans we can consider as an effusion in the joint only the anechoic space that can be seen anteriorly or posteriorly just below the condylar convexity near to the mandibular condylar neck, particularly when it shows the unambiguous hallmarks of the effusion, mainly the distal acoustic enhancement (see Fig. 3).

In the coronal scan, an effusion can be detected superficially and caudally to the condylar convexity. Close to the condylar convexity, the compression can often hide the fluid: at this level only a major effusion can be detected [4].

The measure of the capsulosynovial thickening must be obtained perpendicularly to the tangent line to the convexity, to avoid over- or underestimating the real values. It is also quite difficult to measure this thickening if a low-frequency probe (under 10 MHz) is used. The instrument we use to assess the TMJ, a Toshiba Nemio with a multifrequency linear PLM-1204AT probe (8–14 MHz), provides an axial resolution of 0.14 mm and a lateral resolution of 0.35 mm.

Studying the condylar profile by US, it is possible to assess the following features: erosions of the cortical profile on the lateral side, superior irregularities of the condylar profile, its flattening and the sharpening of the convexity at the point of passage from the lateral to the superior condylar surface in the coronal scan (see Fig. 4). The last alterations are typical of the condylar resorption, similar to the radiographic images [5].

According to our observations, in the reported US image in the work of Melchiorre et al. [1], which presumably is an oblique scan showing the condylar convexity, the neck and a small part of the mandibular branch, the capsular synovial space is clearly thickened and hypoechic and shows a convexity along the plane of scanning (probably due to a thin effusion). The maximum thickness is detectable at the neck, i.e. distally to the point taken as a reference in the paper. The zygomatic arch generates the typical acoustic shadowing, which in the picture is appreciable proximally to the condyle. The subsequent hypoechic image, defined as pseudocystic in the paper, is produced by the muscular fibres of the temporal muscle. These fibres appear hypoechic because of the anisotropic effect as they pass below the zygomatic arch and are oblique to the surface of the probe (see Fig. 5).

All these remarks come from our study of the TMJ in patients with juvenile chronic arthritis, which we started in 1999 at the Gaetano Pini Institute in Milan. Some preliminary data have been previously presented [6].
FIG. 3. Posterior effusion and synovial thickening, axial scan.

FIG. 4. Erosion of the condylar head, coronal scan. Legend as in Fig. 1.
The authors have declared no conflicts of interest.

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About the difficulty in interpreting ultrasonographic images of temporomandibular joint: reply

We have read the letter about the difficulty in interpreting ultrasonographic (US) images of the temporomandibular joint (TMJ), which argues about the method of US examination presented in our previous paper. We wish to point out that the first description of our method of US examination was published first in 1997 [1] and again in 2003 [2]. In both papers the methodology is described extensively, and I believe that the authors can easily access these journals of the Italian and international literature.

On the basis of our experience in adults and children, we are able to assert that the distance between capsule and bone should be

FIG. 5. Our interpretation of Fig. 2 of the paper by Melchiorre et al. [1], shown specularly as previous images. Legend as in Fig. 1. The distance between the two marks indicates the space between the condylar profile and the capsular wall and does not represent the width of the joint capsule. A doubtful small effusion is present distally to the condylar head (small arrow).