Correspondence

1 Guarino M, Reale D, Miccoli G, Tricomi P, Cristofori E. Xanthogranulomatous gastritis: association with xanthogranulomatous chol- 


5 Rogers S, Slater DN, Anderson JA, Parsons MA. Cutaneous xanthogranulomatous inflammation: a potential indicator of interstitial 


8 40:112–17.

Dr Guarino et al comment:

We find no contradiction between our interpretations and Dr Parsons’ statements. Some points, however, deserve specification. The conclusion drawn by Dr Parsons that the gastric lesion which we described is the result of extension of xanthogranulomatous cholecystitis is exclusively based on the finding of sinuses and fistulae formation in the cases he has studied, none of which affects the stomach. Although we agree that adhesions to surrounding tissues are not uncommon in xanthogranulomatous cholecystitis, in our case sinuses or fistulae were absent and, therefore, we find the comparison made by Dr Parsons inappropriate.

As we pointed out in our paper, we suppose that the gastric lesion may result by adhesion and deepening of the primary gall bladder lesion that Dr Paul theory postulates, but there is no definite proof of such an event. Although the cellular mechanisms underlying the collection of xanthomatosus histiocytos and other cellular elements is basically the same, the causes of xanthogranulomatous inflammation may be very different in various anatomic sites, suggesting caution in drawing general conclusions on aetiology.

Therefore, the conviction of Dr Parsons that our xanthogranulomatous lesion we studied certainly results from extension of xanthogranulomatous cholecystitis could be an oversimplification.

Finally, the objection made by Dr Parsons on the choice of term doesn’t carry any meaning, in our opinion. Indeed, even supposing that the gastric lesion originates from extension of primary xanthogranulomatous cholecystitis, the term xanthogranulomatous gastritis seems to be adequate and is as applicable as the extension to the colon of Crohn’s disease is named granulomatous colitis.


5 Guarino M, Reale D, Miccoli G, Tricomi P, Cristofori E. Xanthogranulomatous gastritis: association with xanthogranulomatous chol- 


8 Cozutto C, Carbone A. The xanthogranulomatous process. Xanthogranulomatous inflammation. Pathol Res Pract 1988;183: 

9 395–402.


11 745–6.

NEQAS parasitology scheme

The recent report of the NEQAS parasitology scheme by Hawthorne et al raised several 

12 points of concern.1 As a participant in both the main parasitology scheme and the parasitology included in the general haema-

13 tology NEQAS, it seems to me that double standards are operating. The parasitology NEQAS for blood parasites has developed a complex scoring system that seems to be removed from clinical reality in relation to malaria. The haematology NEQAS asks us only for species identification or estimation of the parasitaemia in Plasmodium falciparum infections (the standard practice of the African laboratories that I have worked in).

In table 2 of Hawthorne’s paper the “correct” parasitaemia is given by the reference laboratory without 95% confidence limits to compare individual laboratories’ performance. This is obviously an important area for two reasons: as an assessment of the severity of infection; and as to whether further therapeutic intervention is necessary, such as exchange transfusion.2

Confusing Plasmodium vivax for Plasmodium ovale is a common problem, but clinically less important because the therapeutic approach is usually the same. In routine practice we always look at both thin and thick films in this laboratory (the latter are not distributed as part of the NEQAS exercises). We usually also have the benefit of more clinical information and often multiple samples over a short period of time which can be of diagnostic use. Getting a sample to the laboratory is the most critical step for laboratory diagnosis; once malarial parasitaemia has been documented, there are relatively few problems. My plea is for one blood parasite scheme with a clinically reasonable scoring system.


Dr Chiodini et al comment:

We are delighted to note that Dr Paul is enrolled in the parasitology scheme as well as the general haematology NEQAS. The schemes, however, have slightly different objectives. The haematology scheme distributes blood films four times a year and our communications with that scheme have indicated that the haematologists wish to maintain and sharpen diagnostic skills within the whole breadth of haematology. The inclusion of more blood parasite films to this scheme would introduce a bias out of proportion to the prevalence of such abnormal films in a district general hospital. Thus the level of input and sophistication of the scoring will inevitably differ between the haematology scheme and the parasitology scheme where the whole undertaking is devoted to blood parasites. Nevertheless, the parasitology scheme supplies both the material and the teaching sheets for the haematologists in respect of blood parasites. As I understand it, the haematologists do not have an actual score for their blood film reports, but the mailing is analysed by participant comments. Expert malaria diagnosis, however, is not a consensus of peer opinion but should be assessed against the actual material sent by the designated expert centre against which performers must match their attainment.

The parasitology scheme asks for species identification or estimation of the parasitaemia in Plasmodium falciparum infection (as do the haematologists). In addition, however, the parasitology scheme does expect an indication of which parasite is involved for example, gametocytes only of Plasmodium falciparum would be managed differently from trophozoites alone.

As for Dr Paul’s comment on parasitaemia, we have the 95% confidence limits for estimates of parasitaemia and participants are scored according to the difference in their report in terms of standard errors from the actual parasitaemia. We entirely agree with Dr Paul that parasitaemia is important and we are therefore disappointed with the poor performance of participants in this area.

We agree that thick and thin films would be an ideal specimen to send, but at the present time we are sending only obvious thin films and have to be realistic in terms of our expectations, particularly as performance, even with these films, is far from satisfactory. Another problem is that thick films, even when stained are unfixed, and therefore additional concerns over biohazard would be raised.

In our routine practice we too have the benefit of more clinical information and multiple samples over a short period of time but this is clearly impractical when one is considering a NEQAS scheme and we have all to accept that it is impossible to reproduce the exact clinical situation in any external scheme, though we feel that we do come close.

We feel that our scoring system is clinically reasonable and would like to point out that a NEQAS scheme with parasitology subclass will be introduced in which the parasitology subclass reports gave the organiser permission to act on severity of clinical errors if it was felt that individual laboratories were scoring in such a way as to constitute a clinical danger. A scheme which takes account of significant errors in estimation of parasitaemia or missing potentially fatal infections such as Plasmodium falciparum is, we feel, clinically relevant. It is clear that Plasmodium falciparum and Plasmodium ovale have the same treatment and thus the severity of error here is less significant, but none the less we are aiming at precision and it is important for participants to know how well or otherwise they are doing.