Lymphoid reaction in chlamydial endometritis

As Paavonen *et al*¹ report, the in vivo lymphoid response to chlamydial infection is largely unknown. I report the results of lymphocyte marker studies in a case of chlamydial endometritis.

The patient, a 21 year old para 1 woman, underwent uterine curettage because of nine weeks' of continuous vaginal bleeding; mucopus had been noted on the cervix and mild urinary symptoms were present. A high vaginal swab cultured routinely grew no obvious pathogen.

Histopathological examination of the curettings obtained showed an infiltrate of abnormal mononuclear cells occasionally resembling Reed-Sternberg cells with abnormal mitoses. Plasma cells and a few polymorphonuclear leucocytes were also present. The infiltrate had only a very vague suggestion of follicularity. The appearances raised serious concern about the possibility of lymphoma and a further curettage was recommended.

At the second curettage five and a half weeks later the curettings were submitted unfixed to the laboratory and divided into specimens for lymphocyte marker studies, routine microbiological and mycobacterial culture, and histological examination. Swabs for viral and chlamydial culture and blood for antibody studies were taken.

Histologically, these curettings showed evidence of a chronic endometritis, although a few large lymphocytes were still present. Microbiological culture yielded negative results. Marker studies were reported as showing many leucocytes consisting of both B and T cells. A normal helper:suppressor ratio was present. Immunoglobulins showed a possible monoclonal proliferation but were equivocal, and definite distinction between lymphoma and endometritis could not be made.

Chlamydia trachomatis was isolated from cervical and urethral swabs and antibody to *C. trachomatis* was detected at a titre of 1/1280; all other studies were normal. A ten day course of tetracycline was given. At follow up appointment 11 weeks later (she failed to attend an earlier appointment) there were no gynaecological symptoms and *Chlamydia* were not isolated from a cervical swab.

Review of all the histological material with Giemsa staining and of a cervical smear taken at the first outpatient visit reported as inflammatory but not neoplastic, showed no inclusion bodies.

The case is of interest because, as far as I

am aware, it is the first time that lymphocyte markers have been described in female genital tract chlamydial infection, and it highlights the considerable lymphoid reaction to this organism which, in this case, simulated a lymphoma, both morphologically and immunologically.

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Reference

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111 Indium labelling of red cells: its clinical use in haemolytic anaemias

Heyns *et al*¹ recently reported the use of ¹¹¹In for labelling erythrocytes. Present methodologies use ⁵⁹Fe for estimating the rate of autologous red cell destruction and ⁵¹Cr for labelling homologous red cells to distinguish between intrinsic or extrinsic causes of haemolysis. Four years ago we studied erythrokinetics using ¹¹¹In oxinate, but as our results showed the unsuitability of ¹¹¹In for this purpose we did not publish them.

Our results showed:

- 1 In nine adult controls the mean half life of ¹¹¹In was 10.6 (SD) 0.6 days, with an elution rate of 5.5 (1%) 1 day, (allowing for a red cell life span of 110 days).
- 2 In 15 extracorporeal haemolytic disorders (Coombs positive) the life span of both ¹¹¹In labelled homologous and ⁵¹Cr labelled autologous red cells was decreased; the correlation between the methods, however, was weak ($r = 0.55$).
- 3 In five intrinsic red cell cases of haemolysis (hereditary spherocytosis) the mean half lives as measured with ¹¹¹In were remarkably similar to those of the control series (10.5 (1) days), whereas the ⁵¹Cr labelled autologous red cell destruction showed an increase as expected (t50:8 to 13 days—that is, 4% to 7%/day).

With ⁵¹Cr labelling the t50 is 30 (3) days, so that sampling at intervals of one or two days is acceptable; with ¹¹¹In, however, errors may be introduced as the natural t50 is only 10.6 (0.6) days. An additional criticism of the ¹¹¹In technique is that a clinical assessment of the site of red cell destruction is not clear from the results. After red cell destruction ⁵¹Cr is slowly released from the sites of sequestration into the plasma and then urine. In contrast, ¹¹¹In may re-enter the circulation bound to transferrin, which can be further bound to receptors in the bone marrow and liver.

In conclusion, the use of ¹¹¹In for labelling red cells is restricted. Both autologous labelling with ¹¹¹In and homologous red cell labelling with ⁵¹Cr are necessary to show intrinsic or extracorporeal mechanisms of haemolysis when the aetiology is unknown.

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Serum isoamylases and liver diseases

The activity of α -amylase in human sera is variable in patients with liver diseases. In some such patients, especially those with chronic active hepatitis, the activity of serum amylase is persistently raised, whereas other patients have serum amylase activities well below the normal limits.² The existence of hepatic α -amylase is still in dispute. Although the livers of several mammals contain amylase, and rat liver has been shown to synthesise amylase,³ most attempts to find an amylase in human liver have failed.^{4,5} Increases in serum amylase activity may occur in liver diseases,⁶ including hepatitis⁷ and biliary obstruction,⁸ but whether this increment to circulating amylase activity originates in the liver has been unclear.

These increases in serum amylase have been taken as evidence that the liver contributes to the total serum α -amylase or that it may act as a regulator of circulating α -amylase activity.⁹ In man there is no convincing evidence to support the hypothesis that the liver contributes to the total serum α -amylase, although Warshaw *et al*¹⁰ found an increase in L₁ type α -amylase in a variety of patients with liver diseases. Only 5%–45% of human serum α -amylase is normally excreted in the urine, and the catabolic pathways of the remaining percentages are unknown.¹¹ If the liver provides one of the normal mechanisms for α -amylase catabolism then injury or disease of the liver may lead to accumulation of α -amylase in the serum, as is the case in renal diseases.

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Letters

Table Total amylase, P, and S type amylase activities in patients with liver disease

Group	Mean (SD)		
	Total amylase (IU/l)	P type isoamylase (IU/l)	S type isoamylase (IU/l)
A Normal volunteers (n = 30) reference range	199 (66) 77-317	110 (44) 25-184	89 (45) 1-184
B Patients without liver disease (n = 18)	194 (47)	96 (30)	98 (47)
C Patients with liver disease (n = 30)	234 (88)*	127 (69)	107 (79)
Primary biliary cirrhosis (n = 7)	210 (84)	92 (38)	118 (63)
Chronic active hepatitis (n = 9)	242 (74)	164 (71)**	78 (47)
Alcoholic cirrhosis (n = 9)	253 (121)	122 (87)	131 (116)

Patients v normal controls; *p < 0.1; **p < 0.05.

While evaluating the "Phadebas" isoamylase kit (Pharmacia) by comparing it with electrophoresis,¹² we applied the inhibition technique to study the serum isoamylases in patients with liver diseases. Sera were also obtained from normal volunteers and patients without liver diseases (Table). The diagnosis was based on clinical examination and laboratory findings. The distribution of men to women was similar in the three groups. We separated those who suffered from primary biliary cirrhosis, chronic active hepatitis, and alcoholic cirrhosis.

Our findings differed from those of Bhutta and Rahman² and agreed with those of others.^{1 6 13} Although MacGregor and Zakim¹ reported that hyperamylasaemia associated with chronic active hepatitis may be of salivary gland origin, as part of the spectrum of extrahepatic manifestations of the disease, we found that it was of pancreatic gland origin.

Finally, we found that three of our patients with alcoholic cirrhosis had S type hyperamylasaemia, whereas only one had P type hyperamylasaemia. The findings of this study suggest that in patients with liver disease and hyperamylasaemia the prominent type of isoenzyme is that of pancreatic origin, with the exception of patients with alcoholic cirrhosis where the prominent isoenzyme is that of S type. Further details of our study, including comparison of results between volunteers and patients and correlation with an electrophoretic technique for isoamylase separation, will be published elsewhere.

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Cryptosporidium sp a "new" human pathogen

We were delighted to see our paper in print

and thank you for doing such an excellent job of the presentation. Unfortunately, on reading the published version, two errors were apparent, which we feel are of sufficient importance to require an addendum. The first is an editorial correction in the final proof copy. This had to be dealt with in great haste, and the nature of your correction was not realised. The error is on page 1323, right hand column, in which the word "not" has been incorrectly inserted. The point may be a pedantic one, but it is widely agreed that although extracytoplasmic, the fact that the parasite is enveloped by the outer cell membranes makes it intracellular. The word "not" should, therefore, be deleted from the text.

Secondly, in Table 2, page 1327, right hand column. I have been asked to point out that Liverpool did give a figure for total positives (41), although the denominator figure had not been given.¹

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Book reviews

Cellular and Molecular Aspects of Aging: The Red Cell as a Model. Progress in Clinical and Biological Research. Vol 195. Ed JW Eaton, Diane K Konzen, JG White. (Pp 464; £52.) Alan R Liss Inc. 1985.

Because of its readily measured biological processes and its finite life span, the red blood cell is a good model for investigating the cellular and molecular aspects of aging. This was the theme of a conference held in Minneapolis in September 1984 with some 80 participants. The 30 papers presented at that conference, together with a verbatim record of the discussions are here presented in the *Progress in Clinical and Biological Research* series. Is aging controlled by 2, 3-DPG and determined by the DPG gene, as suggested by Brewer, or is the process determined by a programmed mechanism of progressive loss of genetic information? The problem of determining which is cause and which is effect was well illustrated in a study on the role of raised red cell calcium concentration: innocent bystander or kiss of death? In the end the validity of using the relatively