

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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Reference

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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 enzyme activity.⁹ In man there is no convincing evidence to support the hypothesis that the liver contributes to the total serum α -amylase, although Warsaw *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.