thrombocytopenia in severe leptospirosis, as shown by the high titres of surface bound immunoglobulin and C3d, the response to both pulse methyl prednisolone and hydrocortisone, and the prompt fall in platelet count after discontinuing the methyl prednisolone. Although both penicillin and heparin have been reported to cause immune mediated thrombocytopenia, the patient was thrombocytopenic before taking either drug, and both dose of heparin used and the time course of the thrombocytopenia argue against a drug mediated thrombocytopenia.3

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References

Non-Hodgkin’s lymphoma and Hodgkin’s lymphoma in the same patient

A 66 year old white man was referred with a subcutaneous swelling behind the right mid-thigh that had been present for one year. The indurated and oedematous swelling was resected. Histological assessment showed that it was non-Hodgkin’s lymphoma (NHL)—small lymphocyte type (fig 1a). There was no evidence of lymphoma elsewhere. Peripheral blood cell marker tests showed no conclusive evidence of monoclonal B cells. No other treatment was given; he was followed up three months. After 14 months of good health he reported with a four week history of severe diarrhoea, anorexia, and weight loss. He was dehydrated and had an upper abdominal mass separate from an enlarged spleen and a mass of enlarged lymph nodes in the right iliac fossa. The right leg was oedematous. An ultrasound scan showed infiltration in the liver and para-aortic, mesenteric, and pelvic adenopathy. The only laboratory abnormalities were a normocytic anaemia of 8·6 g/dl with an erythrocyte sedimentation rate (ESR) of 92 mm in one hour, hypoalbuminaemia, and a grossly raised alkaline phosphatase activity. There was variable expression of T cell receptors on peripheral blood mononuclear cells with a CD4:CD8 ratio of 1 and increased expression of CD25. There was no evidence of B cell monoclonality.

Surprisingly, a node biopsy specimen showed a typical picture of lymphocyte depleted Hodgkin’s disease (HD) (fig 1b). After chemotherapy there was no detectable bulk disease at three months. Repeated endoscopic biopsy specimens have shown chronic inflammatory changes only and his bowel symptoms slowly resolved. He remains on follow up one year later.

The term discordant lymphoma has been reserved for those cases with different histological types occurring in different anatomical sites. Kim et al used the term composite lymphoma to include those cases with both Hodgkin’s disease and non-Hodgkin’s lymphoma within a single anatomical site.5 Patients with discordant lymphoma with HD and NHL are well recognised, most often with NHL following treatment for Hodgkin’s disease. Carrato et al found five instances of Hodgkin’s disease developing after treatment for NHL from 2019 documented lymphoma cases with a latent period of between five to 23 years. Lymphocyte depleted Hodgkin’s disease was not found in this group. The pathogenesis of Hodgkin’s disease is unclear and both T and B cells have been implicated by various workers. A proliferation of B cells which might provoke a T cell reaction eventually predisposing to Hodgkin’s disease has been reported in cases of nodular Hodgkin’s paragranulomas, but our case was firmly diagnosed as NHL. Viral infection causing a change in T cell surface antigen expression may produce a chronic immune reaction leading to the appearance of neoplastic reticulum cells.4 We found variability of T cell antigen expression, evidence of interleukin 2 receptor expression, and disturbance of the CD4:CD8 ratio during the second illness which may have been due to an immunological response to the disease rather than a residual feature of a T cell reaction before the development of Hodgkin’s disease. It is difficult to ascribe the development of Hodgkin’s disease after treated NHL to the treatment itself because many more cases would be expected. It is even less common to find this sequence of events without intervening treatment. In this unusual case it seems that either the appearance of the two disorders was purely coincidental or that the lesion with clear features of NHL represented a stage in the development of the eventual Hodgkin’s disease. It was interesting to find that the only peripheral nodal disease at the presentation of Hodgkin’s disease was in the
immediate drainage area of the initial lesion.

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References


Assessment of rapid method for identifying *Escherichia coli*

Most pathogenic enterobacteria identified in medical microbiology laboratories are *Escherichia coli*. If a simple, inexpensive, and rapid yet reliable method for identifying these isolates was available it would greatly improve efficiency. A kit recently introduced by API-bioMérieux (UK) Ltd, Rapidec coli, seemed to fulfill these requirements and we assessed its reliability based on results for 1000 *E coli* and 250 other enterobacteria isolated from urinary tract infections.

The Rapidec coli kits were supplied by the manufacturers (API-bioMérieux (UK) Ltd Basingstoke). Each organism is tested using four cupules designated C, S, 1 and 2; C is an opacity standard for the bacterial suspension which is prepared in S while 1 and 2 contain media for the detection of β glucuronidase and β galactosidase, respectively. The addition of James reagent (API-bioMérieux (UK) Ltd) to cupule 2 also detects indole production. Five groups of four cupules are combined on a plastic strip and any sets of four cupules not required can be cut off, refrigerated, and used the following day. Sterile distilled water is added to cupules C and S and the growth from two to three colonies of the organism to be tested is homogenised in cupules S to the same opacity as the standard; 50 μL of the suspension are then transferred to each of the cupules 1 and 2. After incubation at 37° for two hours a yellow colour in cupules 1 and 2 indicates a positive test for β glucuronidase and β galactosidase, respectively; in negative tests the suspension remains colourless. After reading these results one drop of James reagent is added to cupule 2 in which the development of a red colour is positive for indole production. If all three tests are positive the organism is regarded as *E coli*, with only two tests positive the organism may be *E coli* but further tests are required (indeterminate) and with only one positive or all negative the organism is not *E coli* (non-*E coli*).

The 1250 isolates were designated as *E coli* or non-*E coli* on the basis of standard biochemical tests: the tests used included indole, MR/VP, citrate, malonate/PPA, urea, glucose, lactose and motility. If there was any doubt about the designation of any isolate, or this differed from that by Rapidec coli, then it was identified by API 20E as were all isolates designated as indeterminate by Rapidec coli; in these cases the definitive designation was determined by the API 20E result.

One thousand isolates were designated *E coli* by biochemistry or API 20E and 250 as non-*E coli* (Citrobacter, Enterobacter, Hafnia, Klebsiella, Morganella, Proteus, Providencia and Serratia); these designations, in relation to those derived from the Rapidec coli tests, are shown in the table. A designation of *E coli* by Rapidec coli was always correct. Three isolates were incorrectly designated non-*E coli* and in each case this was because the indole test was negative with Rapidec coli whereas it was positive by the standard method and by API 20E; two isolates were designated as indeterminate for the same reason. Of the 38 non-*E coli* designated indeterminate by Rapidec coli, 25 were *Klebsiella oxytoca*.

Rapidec coli was extremely easy and quick, both to set up and to read (average two minutes for an isolate). Used as a means of eliminating about 90% of *E coli* from more costly and time consuming identification procedures, it was absolutely reliable with these urinary enterobacteria, and by giving a result in two hours it caused no delay in obtaining a result for those isolates requiring further identification. The savings that could be made in any particular laboratory, by incorporating Rapidec coli in an identification procedure, would depend on its cost in terms of time and material relative to that of the usual identification method and on the proportion of enterobacteria routinely identified as *E coli*. For example, if the cost of Rapidec coli is 20% of the usual method then savings would be made once the proportion of *E coli* exceeded 22%; at 50% and 90% *E coli* the savings would be 25% and over 60% respectively.

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