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Accessory cells as primary target of human immunodeficiency virus HIV infection

We recently reported a high increase in the number of dendritic reticulum cells (DRC) that were positive for the monoclonal antibody KiM 4¹ in lymph nodes from patients with persistent generalised lymphadenopathy (PGL).² Further studies on 12 PGL lymph nodes showed an increase of interdigitating reticulum cells (IDRC) positive for S100 protein and KiM 1¹ in the

T regions. Staining for a proliferation associated antigen with the antibody Ki 67³ showed that most cells within the germinal centres in PGL express this antigen. Double staining with Ki 67 and Kim 4 showed that most of these cells are DRC. In the T region numerous cells were also positive for Ki 67; their distribution and morphological features indicated that they were IDRC.

Immunohistochemically Epstein Barr virus (EBV) was identified in cells of the B region, while cytomegalovirus (CMV) was present in the T and B region. In situ hybridisation detects the EBV genome in most cells of the B region, whereas most of the cells in the T region contain CMV DNA. Staining with an antibody against the gag protein p24 of HIV showed retroviral infection of some lymphocytes and several macrophages, DRC, and IDRC.

Interdigitating cells showed a positive reaction in their cytoplasm, on the nuclear membrane, and within the nucleus. Characteristically, these infected cells were surrounded by a corona of lymphocytes whose cell membranes also stained for p24 (figure).

Our results indicate that HIV or concomitant viral infections, such as EBV or CMV, or a combination, can cause a proliferation of IDRC as well as DRC that have hitherto been regarded as "end cells." The detection of HIV in DRC⁴ and IDRC shows that the presence of the CD⁴ (T⁴) antigen is not a prerequisite for an infection by the retrovirus. The characteristic arrangement of lymphocytes staining for p24—with the reaction still restricted to the cytoplasm and sometimes found only in

areas in close contact with IDRC—around infected interdigitating cells indicates that accessory cells such as IDRC, DRC, and macrophages are the first target of HIV infection and may thus serve as a reservoir for the virus.

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Figure
T region of PGL lymph node.
Interdigitating cell positive for
p24 on cell membrane within
cytoplasm and on nuclear
membrane (centre)
surrounded by T4 lymphocytes
(confirmed by double
staining). Their positive
reaction for p24 is restricted to
cell membranes indicating
HIV absorption. (Cryostat
section, direct
immunoperoxidase.) $\times 1000$.

Diagnosis of acute myocardial infarction at necropsy

We were interested to read a report of a method for diagnosing acute myocardial damage at post mortem examination by enzyme analysis of pericardial fluid.¹

When death occurs within a few hours of a myocardial infarct there are often no macroscopic nor histological features to confirm the diagnosis, other than perhaps an impaired coronary arterial supply. Though techniques to show early changes have been described,^{2,3} none has proved universally acceptable, either because it is not readily available or because reproducibility is poor. A method for diagnosing acute myocardial infarction by enzyme analysis of pericardial fluid, as described¹ is therefore very

attractive. The sample is easily obtainable at necropsy and the assays can be performed in a routine clinical chemistry laboratory.

In the study concentrations of creatine kinase (MB fraction) and lactate dehydrogenase were measured. Higher concentrations were shown in patients who had acute cardiac disease than in those who had died of a non-cardiac cause. Very high concentrations were found in necropsies with cardiac massage or severe trauma. The results suggested that these measurements could be helpful in diagnosing death related to acute myocardial damage.

We decided to pursue this finding and collected a series of samples over 18 months. Our criteria for inclusion in the study were not as strict as those of the original study. Stewart *et al* only included samples taken within 24 hours of death where a detailed history was available. Our samples were taken at routine post mortem examination between two and six days after death; the clinical notes were available in every case. The samples were spun and stored frozen. Hydroxybutyrate dehydrogenase (HBD) and glutamate oxaloacetate transaminase (GOT) concentrations were measured.

Cases were divided into two groups, patients with a non-cardiac cause of death and those with clinical evidence suggesting an acute cardiac cause of death, or who had received cardiac massage. The results were disappointing and are summarised in the table. As was found in the original study the range of results was large, and the values

Summary of pericardial fluid enzyme results (U/l) (figures in parentheses are No of observations)

	HBD		GOT	
Non-cardiac deaths	1800 844-3928* (47)	p = 0.078	730 339-1750 (45)	p = 0.051
Cardiac deaths	4415 1450-5490 (12)		1865 810-3460 (12)	

*Interquartile ranges; **Mann-Whitney U test.

were not normally distributed. For this reason we expressed the results in terms of median values and interquartile ranges. There was considerable overlap of values between the two groups, and although the median values in our cases of cardiac death were higher than in the cases of non-cardiac death, the difference between the two groups was not quite significant and this was despite having included in the cardiac death category all patients who had received cardiac massage and who tended to have the highest enzyme activities. We found no correlation between the time the sample was taken after death and the enzyme values, nor did there seem to be a correlation between the time the sample was stored and the enzyme values.

In conclusion, our results suggest that the activity of enzymes in pericardial fluid is unlikely to provide the definitive diagnosis

of acute myocardial infarction at routine necropsy.

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Book reviews

Cell, Tissue and Disease. The Basis of Pathology. 2nd ed. (Pp 502; £8.50.) WB Saunders. 1986. ISBN 0 7020 1125 8.

The second edition of Professor Woolf's book on general pathology will, I am sure, be welcomed by a wide range of both undergraduates and postgraduates. Certain other shorter textbooks of general pathology have been criticised in the past for being too superficial. Professor Woolf has skillfully avoided this pitfall by sticking to his policy of selectivity: he does not attempt to deal comprehensively with disorders of heredity and development but he still manages to cover many hereditary aspects of current interest and importance such as heredity and neoplasia. He gives a succinct and clear account of oncogenes and DNA hybridisation technology. The book contains a large number of line drawings and diagrams which are both clear and helpful, though I

am sure that Professor Woolf will be slightly disappointed by the poor reproduction quality of some of the histological illustrations. I suspect, however, that this is a concession to financial pressures, and this book is excellent value for money.

DA LEVISON

Interpretive Medical Microbiology. HP Dalton, HC Nottebart. (Pp 1062; £95.) Churchill Livingstone. 1986.

This is a tour de force, comprising over 1000 pages and, at 6lb 4oz, is no pocket book. As might be expected, it is a multiauthor book and aims at a fully comprehensive coverage of all the micro-organisms (bacterial, viral, fungal and parasitological) which have been reported to cause damage to man. As such, it covers a vast range of micro-organisms, some of which this reviewer has not encountered, even as names before, and it will prove valuable as a aide de téléphone.

The comprehensiveness makes it difficult for a single reviewer to assess the book fully

and fairly, and I have turned, therefore, to my own subject, for which both good and bad was to be found. There were some surprising suggestions for techniques (yes, this book covers methods as well) such as clarifying and filtering specimens for the isolation of viruses and using multiwell plates for cell cultures. The former is impractical (and liable to reduce isolation rates substantially) and the latter a recipe for disaster even the experienced in a busy laboratory. Adenovirus serotypes stop at type 33 and are not found in faeces—no mention at all!

The strength of this book lies in its review of the literature; the other aspects may be less authoritative and reflect American practice. There is less discussion of interpretation than the title suggests, and those who are prepared to pay the considerable cost should browse through their own topics before deciding whether to buy. Because each body system is treated separately, there is a certain amount of repetition, unavoidable in such a format.

CR MADELEY