Letters to the Editor

References


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 4 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 1 in the T regions. Staining for a proliferation associated antigen with the antibody Ki 67 3 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.

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 DRC 4 and IDRC shows that the presence of the CD4 (T+) 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.


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) x 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 described 1 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 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.

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

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<tr>
<th></th>
<th>HBD</th>
<th>GOT</th>
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<tr>
<td>Non-cardiac deaths</td>
<td>1800</td>
<td>730</td>
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<tr>
<td></td>
<td>(484–3928*)</td>
<td>(339–1750)</td>
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<tr>
<td>Cardiac deaths</td>
<td>4415</td>
<td>1865</td>
</tr>
<tr>
<td></td>
<td>(1450–5490)</td>
<td>(810–3460)</td>
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Summary of pericardial fluid enzyme results (U/l) (figures in parentheses are No of observations)