The number of intraepithelial T cells decreases from ascending colon to rectum

The αβ-integrin (CD103) is expressed almost uniquely by T cells of the mucosal immune system, where it is upregulated on activated cells by the action of transforming growth factor β. The only known ligand for this integrin is E-cadherin, which is expressed by all epithelial cells, where it constitutes a homotypic adhesion system necessary for tight junction formation. A role for interaction between the αβ-integrin and E-cadherin in the localisation of intraepithelial T cells is supported by the reduction in numbers of mucosal T cells seen in CD103 deficient mice.7

The role of CD103+ T cells remains unclear. However, the potential of these cells to bind specifically to the epithelium is consistent with a capacity to mediate damage localised to this tissue. Indeed, CD8+CD103+ T cells have been shown to kill epithelial targets in vitro, and have been implicated in disease processes such as tubular destruction during renal allograft rejection.7 The potential for modulation of experimental colitis by the administration of antibodies directed at CD103 provides evidence that these cells might also act as effectors during this disease.7 Given the increasing severity of ulcerative colitis from the proximal to distal colon, it is perhaps reasonable to propose the existence of a similar gradient in the number of potential T cell effectors within the epithelium of the normal colon.

In this study, we performed a survey of the linear distribution of cells expressing the CD3, CD8, and CD103 phenotypic markers within the normal human colon. Pinch biopsies were collected from the ascending, transverse, descending, and sigmoid colon and the rectum of patients attending clinic for routine diagnosis. Frozen sections were analysed from eight patients who were considered normal after routine histological evaluation. Endogenous peroxidase was blocked and the sections were stained with appropriate monoclonal antibodies (CD3, clone T3-4B5; CD8, clone CD8/144B; and CD103, clone BerAct8). After counterstaining with Mayer’s haematoxylin, the number of CD3, CD8, and CD103 positive cells was counted in each crypt cross section, and the mean number of each cell type in each crypt was calculated. Image analysis was used to demonstrate that the crypt cross sectional area did not vary between different sites within the colon.

Figure 1 shows the typical distribution of CD103+ T cells within the normal colon; it is apparent that many, but not all, of these cells are present within the epithelium. Figure 2 presents a summary of the numerical data derived from each of the eight normal patients. In the case of each phenotypic marker, the data are reproducible between individuals and show a significant decrease in the number of cells in each crypt from ascending colon to the rectum (CD3, p < 0.005; CD8, p < 0.02; CD103, p < 0.03).

Our data show clearly and for the first time a linear decrease from the normal ascending colon to the rectum in the number of cells expressing the CD3, CD8, and CD103 phenotypic markers. Although it appears paradoxical that this gradient runs contrary to that which may be expected if CD103+ T cells are, indeed, the effectors responsible for tissue damage in ulcerative colitis, it is tempting to speculate that this gradient of T cell distribution has some impact on the potential for immune reactivity within the gut.

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References


Food for thought

We read the poem by Dr Tirumalae with interest.1 Our particular interest stems from the work we did some years ago on the understanding, use, and potential modernisation of food terms in pathology. Having moved south from Lancashire and Cheshire, respectively, we found our missionary zeal to maintain the position of “sago spleen” as core knowledge in the medical curriculum was thwarted. The students of the South did not find reference to a Lancashire puddling manufactured from imported Indonesian rice back to be of value in their education at all. In an attempt to rectify this we tried to update archaic food terms to ones more fitting to the 21st century. However, we stayed in the South and continued to modernise a small part of pathology, some years ahead of the “Pathology Modernisation

Figure 1 Immunochemical localisation of CD103+ cells (stained black) within the normal human colon.

Figure 2 Summary cell count data showing the number of cells positive for CD3 (closed diamonds), CD8 (closed circles), and CD103 (closed squares) in each crypt within sections from the ascending (asc), transverse (trans), descending (desc), and sigmoid (sig) colon and the rectum (rect) from eight normal patients. Data points show mean value; the error bars represent the SEM.
How do we define Hodgkin’s disease? The authors’ reply

We have read with interest the eletter by Dr Naresh dated 26 March in response to our earlier letter, which dealt with the problem of the borders between classic Hodgkin’s lymphoma (CHL) and anaplastic large cell lymphoma (ALCL) and suggested that the proponents of the World Health Organisation (WHO) classification had a more flexible attitude towards CHL than ALCL. We believe that the WHO classification simply reflects the philosophy of the revised European-American (REAL) classification and morphologically evaluates a list of entities that can be readily recognised with the techniques available at the moment, which are defined by the amalgamation of molecular data, and clinical findings. This list can be easily updated, whenever new validated information becomes available in the literature.

We believe that the present scenario of CHL is much more homogeneous than the one depicted for ALCL. In fact, most if not all examples of CHL have a germinal centre derivation, as shown by molecular data and B cell variant cyclin D1 gene expression, which is absent in ALCL. There is no consistent lack of ALK protein expression. We believe that the WHO classification is useful in discussing the different biological aspects of the disease in the light of modern molecular biology.

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References

Cytopathology of Bone and Soft Tissue Tumours

Layfield LJ. (£115.00.) Oxford University Press, 2002. ISBN 0 19 513236 X.

“Fine needle aspiration (FNA) is only now gaining acceptance as a primary modality for the study of musculoskeletal lesions.” So opens the preface to this book, and immediately the reader needs to choose how they are going to receive this statement. If you are sceptical then read the rest of the preface for its outline of the justification of FNA in this context, prepared to stay on the ship if persuaded; if you instantly agree then dive straight into the first chapter. It is an introduction, but with more than just technical aspects and analysis of rates of success or failure; the cost aware can read how many dollars are saved by FNA compared with open biopsies. Of more value are the tables and diagrams summarising cellular features in benign and malignant lesions, and algorithms for diagnosis. If you are going to use FNA for this purpose then get a copy of these tables on to the wall in front of you.

Most of the book works systematically through soft tissue and then bone tumours, using a standard pattern giving a description or overview, histological findings, cytological findings, problems in diagnosis, and a summary of key features. The text is clearly written, with good descriptions of the cellular features, and numerous illustrations: bullet points give added clarity where appropriate.

So what are you hoping to achieve by reading this book?

Are you a histopathologist who is an occasional cytopathologist? All the illustrations will look the same, and you should not expect to make a useful comment most of the time.

In summary, this is a well written text on a difficult subject, with clearly presented information. Should you show this book to your clinical colleagues to encourage them to use FNA more often? It depends on whether you are an enthusiast.

J Goepel

The Hospital Autopsy, 2nd ed.


Didactic information in a textbook cannot substitute for practical experience in gaining skill and dexterity in the performance of autopsies. However, the editors have produced a commendable syllabus, making most of the potential of a textbook in this area. This text is almost unique in this respect; it should be of major importance to trainees, but also presents useful reference material for established practitioners.

The first five chapters cover essential preparatory information for the autopsy, starting with the history and comments on the future of where autopsies are going. Autopsies and the law are then covered, in up to date and comprehensive detail (although the detail is likely to be UK specific). The ethical and religious aspects of autopsies are adequately covered. There then follows a valuable chapter on biological safety, which links through to a following chapter on autopsy suite design and construction.

The group of chapters that follow (5–8) cover the practical conduct of routine autopsies, which are well described and illustrated. The next group of chapters (9–12) cover specialist dissection and circumstances, such as examination of the nervous system, and fetal, perinatal, infant, and maternal autopsies.

There then follows a group of chapters (13–15) that are a very valuable source of information on ancillary investigations such as toxicology, microbiology, and even immunological analyses.

The final group of chapters (16–18) round off the syllabus by discussing clinical demonstration, autopsy report formulation and teaching, reconstruction of the body, and the role of the autopsy in clinical audit. It might have been slightly more logical if reconstruction of the body had been included with the routine autopsy techniques.

Allowing for the limitations that a textbook cannot teach practical technique, that chapters on autopsy and the law are jurisdiction specific, and that information on autopsy consent procedures is liable to become out of date as the regulatory environment moves on, it is difficult to conceive of a better syllabus within a textbook of this size. I warmly commend it as a bench book in all histopathology departments, whether trainees are present or not. Even for experienced practitioners, there is useful information on ancillary investigations, and advice on autopsy suite design of the calibre presented here is difficult to find elsewhere in any single source.

T Stephenson

Handbook of Toxicologic Pathology, 2nd ed


My first impression on being asked to review this “hand book” was that I would need big hands to hold it for long! I found this a most impressive text consisting of two volumes each of about 800 pages. The handbook is a multiauthor text and has now appeared in second edition.

Volume one describes various principles of toxicology whereas the second volume looks at organ specific toxicology. I found that some chapters were particularly good, such as those describing heavy metal toxicology and radiation damage. The handbook gives a wealth of information written by experts in their field. Colour photographs may have assisted the text but presumably at the expense of production costs.

In summary, I believe this to be a most comprehensive reference book covering the topic of toxicological pathology thoroughly. Although specialised, it may well find a place in the general histopathology or chemical pathology laboratory. More likely, it would be used in a specialist toxicology department or larger library.

M Crook

Full details of events to be included should be sent to Maggie Butler, Technical Editor JCP. The Cedars, 16 Queen Street, Castle Hedingham, Essex CO9 3HA, UK; email: maggie.butler2@btopenworld.com

ACP Management Course for Pathologists, 2003

10–12 September 2003, Hardwick Hall Hotel, Sedgefield, County Durham, UK

Further details: Ms Valerie Wood, ACP Central Office, 189 Dyke Road, Hove, East Sussex, BN3 1TL, UK. (Tel +44 01273 775700; Fax +44 01273 773303; email valerie@pathologists.org.uk)
Food for thought

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