Candidal infection is uncommon in acute oesophatitis: evidence from a non-selected DGH population

Mucosal candida infection of the lower oesophagus is unusual except in certain groups of patients who are either immunosuppressed or who have other recognised causes of candidal infection. 1 Invasive oesophageal candidiasis occurs most often in the immunosuppressed, occurring in 10–20% of patients with myeloproliferative disorders or leukaemia and in up to 74% of patients with AIDS, with an increased frequency of infection in patients with endocrine disorders such as hyperparathyroidism. 2 Previous studies have shown that the incidence of candidal infection in 22,000 consecutive hospital admissions was 0.1% (27 cases), whereas it was found in less than 5% of a general population presenting with gastrointestinal complaints. 3 It is important to exclude invasive candidiasis as this is a major risk factor in the development of candidal septicemia, which can result as a direct effect of visceral wall complication. Complications of oesophageal candidiasis also include oesophageal stenosis and perforation, which may occur in the acute phase of infection, and which can be life threatening. Less commonly, pseudodiverticulosis may also result. 1,4

Candidal oesophagitis is caused most commonly by C. albicans, C. tropicalis, and C. krusei. 5 The diagnosis of invasive candidiasis requires a combination of clinical suspicion and laboratory investigation. Difficulties in diagnosis arise where the clinical presentation is non-specific or serology produces false positive results because of vulvovaginal candidiasis. Oesophageal brushings are useful for identifying mucosal surface candidal colonisation but are poor at detecting fungal invasion. 6

Here we present the results of a retrospective review of 60 cases of acute oesophagitis, with reassessment for candida by D-PAS staining. Ninety consecutive oesophageal biopsies coded on the laboratory computer SNOMED database as acute inflammation with or without ulceration were examined from the period 1997–1998. In our hospital few specimens are sent for mycological examination but are poor at detecting fungal invasion. 6

The aim of our study was to examine the method of routine reporting currently used in our laboratory when dealing with inflamed oesophageal biopsies—that is, without routine additional fungal stain—and to examine the efficacy of detecting candida on routine haematoxylin and eosin stained slides with an additional PAS or D-PAS stain. We showed that the addition of a routine fungal stain for endoscopic biopsy is likely to be of benefit in the detection of candida if the patient is not in an at risk group. If the patient is at risk of candida then combined endoscopic biopsy and brush cytology is likely to be much more useful than a biopsy alone for the detection of the organism. 5

Causes of candidal infection of the lower oesophagus include use of antibiotics, which may allow overgrowth or colonisation by Candida species, inhaled corticosteroids, reduced gastric acid output from H2 receptor blockers, proton pump inhibitors, or prior vagotomy. Alcoholism, diabetes, malnutrition, advanced age, and abnormal oesophageal motility associated with conditions such as scleroderma and achalasia have also been associated with candida oesophagitis. Stries, tures, obstructing tumours, or diverticula which cause stasis are also associated with fungal oesophagitis and may be a chronic mucosal cutaneous candidiasis syndrome. Nine patients (15%) in our study had D-PAS stains performed at the time of biopsy by the reporting pathologist and the majority of these additional stains were requested when there was evidence of acute ulceration, not just inflammation. The use of routine stains for candida is not justified unless the patient is immunosuppressed or falls into an at risk group. Routine investigation for oesophageal candidal infection should also include brush cytology.

Although the purpose of Weddell's trip was not discovery, his name has been retained by three eponyms: the Weddell seal, the Weddell sea, and Weddelite (calcium oxalate).

CARLOS ORTIZ-HIDALGO
Department of Pathology, The American British Cervical Cancer Hospital, Star 136 Eq. Observatorio, Mexico City 01120, Mexico


An assessment of the artefacts introduced by mounting two parallel sections from each histological level of cervical punch biopsies.

A previous study1 that compared different methods of orienting cervical biopsies has been extended to determine the optimal method of mounting the sections, cut from these biopsies, on the slide to ensure that they are clearly visible for histological examination under the coverslip.

In this department six levels have traditionally been cut from each cervical biopsy, with two adjacent sections from each level being mounted on either side of the midline along the long axis of the slide. This practice places the sections in close proximity to the edge of the coverslip where they may not be protected if the coverslip moves medially or where they may become obscured if mountant is squeezed from between the slide and the coverslip, and becomes smeared over the edge of coverslip.

Two hundred routine cervical punch biopsies in which duplicate sections from each level were mounted on either side of the midline were assessed prospectively up to July 1999. They were compared with 47 biopsies, mainly gastric, duodenal, and rectal, in which a single section from each level had been mounted at the midline of the slide. The slides were reviewed by a single pathologist who assessed the following features:

1 Is part of one or more of the sections from each level incompletely covered by the coverslip leaving it at least partially unprotected?

2 Is part of one or more of the sections from each level completely or incompletely covered by the mountant? As a result of being covered by mountant, are (3) one or more of the sections from each level partially or completely obscured?

This category was included to distinguish those cases in which, despite mountant having been smeared over the coverslip, it had retained its transparency, permitting the features in the underlying section to be assessed from those cases in which the mountant was no longer transparent and obscured the section.

The review by Soldan and Barbara1 on the risks of infection transmission by blood transfusion is an interesting and comprehensive article. However, there is little reference to prion disease and in particular new variant Creutzfeldt-Jakob disease (nvCJD) and the recently recommended control measures for blood donated in the United Kingdom. The only mention is in the last paragraph where they state “whether prion disease can be transmitted by transfusion is currently uncertain”.

From 1995 to the end of March 1999, 40 cases of nvCJD had been reported in the United Kingdom, and it is currently not known how many people may be incubating it asymptptomatically. A recent working party report1 considers that the distribution of infectivity of nvCJD may be different from that of other forms of CJD, as in the former there may be more involvement of lymphoreticular tissues, possibly involving circulating lymphocytes. Evidence of nvCJD has previously been found in human tonsillar tissue.2

In Britain donated blood supplies are to be treated to reduce the risk of patients being infected with nvCJD because of the theoretical risk that it could be transmitted by white blood cells.3 Plasma which is used to manufacture plasma products—for example, as used in haemophilia—is currently obtained from donor units in the United Kingdom where material was not discovered, his name has been retained by them.4

“Better blood transfusion.”5

5 Warden J. Blood supplies to be treated to reduce CJD risk. BMJ 1998;317:232.

Authors’ response

We agree with the author of this letter that more comment about nvCJD and the adoption of measures to try to control its possible transmission by blood donated in the United Kingdom is warranted. The situation with regard to nvCJD, as described by Dr Dawson, was evolving during and since the time we wrote our review, and we welcome its addition to the record in this manner.

K SOLVAND
J BARBARA
PHLS, Colindale Avenue, London NW9, UK

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Book reviews


This book updates two related WHO publications, Basic Tests for Pharmaceutical Substances (1986) and Basic Tests for Pharmaceutical Dosage Forms (1991), and gives information on tests for a further 23 substances, 58 dosage forms, and four medicinal plant materials. The drugs/preparations listed are mainly from the WHO Model List of Essential Drugs (1997). Reagents, test solutions, and volumetric solutions not listed before are detailed in the present volume, although there is nothing on suppliers, and little on reagent stability, storage conditions, and so on. There is a cumulative index to the three books.

The tests are confined to a physical description of the material and simple colour and other reactions, and aim to facilitate identity testing of bulk supplies, for example when labelling is unclear, and to indicate whether gross degradation has occurred in certain substances. A lot of work has gone into this relatively short publication, as evidenced by the list (more than a page) of acknowledgements of individuals and collaborating centres. Is the book of interest to clinical/forensic laboratories? Probably not, except that some laboratories might find a few of the tests useful if asked to identify powders, and so on (see Flanagan RJ, et al. Basic analytical toxicology. Geneva: WHO, 1995). There are simpler ways of identifying tablets (for example, Ramsey JD, Woolley JM. TICZAC—a CD-ROM for the identification of tablets and capsules. The International Association of Forensic Toxicologists: Proceedings of the 35th Annual Meeting, Padova, 1997:174–82).

The tests are supplemented by a review of recent published non-WHO work on simple methods for identifying pharmaceuticals, including volumetric, spectrophotometric, and thin layer chromatographic (TLC) methods. It seems that more than 150 TLC procedures were developed by WHO collaborators in the early 1980s using some 40 different mobile phases, but this work was not published as it was thought necessary to try to reduce the number of mobile phases used. Publishing and updating books, data sheets, and so on is becoming easier as desk top and even electronic publishing become the norm. It remains to be seen how WHO will respond to this challenge—there is not even an email address for comments in the present volume.

R J FLANAGAN


The first edition of this book was published in 1990 with the subtitle A guide to the FAB classification. (The FAB group had by then published proposals for the classification of the acute leukemias, the myelodysplastic syndromes and the chronic lymphoid leukemias.) “This book is much more than an atlas,” quoted from a review of that edition, advertises this new volume from its back cover. How true. Much more than just a guide to the FAB classification too. It has been comprehensively revised and updated to include current immunophenotypic, cytogenetic, and molecular developments, and also consideration of scatterplot data from the new generation of automated counters. The book is both a comprehensive atlas of clearly reproduced photomicrographs of a whole range of common and rare leukemic material, and an elegant and enthusiastically written treatise on leukaemia classification. No classification of leukaemia can be perfect. The often competing criteria of easy and reproducible applicability, clinical relevance, and biological plausibility necessitate compromises. This work does not seek to champion or defend the FAB classifications, but explains them critically, and sets them in a contemporary context in the light of the newer technological developments. Cytogenetic and molecular discoveries have clarified many biological entities. Meanwhile, generations of careful morphologists, for whom Dr Bain is a contemporary standard bearer, have gone back to the microscopic appearances to find that much of the information was there for the want of looking. Thus we can predict from the preliminary appearances when we are going to find an 8;21 translocation or a 16 inversion, whether or not a Philadelphia negative case is going to be found to have a bcr rearrangement, and many more examples. For the clinical haematologist this is not only rapidly available free information, it is also fun, and there’s little enough of that around these days. Even the developing “Bugger the cytochemistry, what do the markers show?” school of haematology should be enticed back to their microscopes by this enthusiastic writing, and I commend this book unreservedly to all haematologists and trainees.

PETER CAREY

Practical Adult Cardiovascular Pathology Course

Royal Brompton Hospital, Imperial School of Medicine
6–7 March 2000

A “hands on” course approaching in detail the problems facing the diagnostic pathologist when dealing with cardiovascular pathology. Approaches to cardiac necropsy and sudden death will be emphasised. The course is aimed at trainees studying for the MRCPath.

Further details: Short Course Officer, National Heart and Lung Institute, Dovehouse Street, London SW3 6LY, UK; tel +44 (0)2073518172; fax +44 (0)2073518246; email: shortcourse. NHLI@IC.ac.uk

British Society for Clinical Cytology

BSCC Spring Tutorial: Cervical Cytology

Guy’s Hospital, London
7 April 2000

Lectures and workshops including “Invasive squamous cell carcinoma” (W Gray), “Borderline changes” (P Smith), and “Atrophic smears” (L Turnbull).

Further details: BSCC Office, EMI, Central Research Laboratories, Dawley Road, Hayes, Middx UB3 1HH, UK; tel +44 (0)181 6062511; fax +44 (0)181 6062565; email: lesley.couch@psilink.co.uk