LETTERS TO THE EDITOR

Tipp-ex fluid: convenient marker for surgical resection margins

The accurate recognition of resection margins in surgical material can present problems in routine practice. A need exists for a convenient, cheap, ready mixed, and easily used marker substance, which may be kept for long periods for use when required. Tipp-ex white fluid (Tipp-ex Ltd, Camberley, Surrey), a solution of titanium dioxide and polyacrylate in trichloroethane, is a standard office material used to blank out typing errors. The solution comes in a 30 ml plastic bottle, with a brush applicator on the cap (cost about £0.99 a bottle). The bottle can be kept beside the cut up area and remains usable for many months. Sections taken through a specimen, such as a breast lump, can be kept in the correct orientation on the cutting board and the outer edge marked with Tipp-ex before being placed in the cassette. The fluid requires no drying time and does not spread when applied to blotted tissue, whether the cassette is placed in a dry rack or directly into formalin. Clear and reliable marking is achieved and the thick white line is clearly seen grossly and on the slide. The material appears as a granular black line under the microscope (figure). Although "lifting" of the marker from the surface, probably due to tissue shrinkage, may produce a slight gap between tissue and marker, this does not present any problem in practice as the orientation and contour of the marker are clearly preserved. Toxicity is not a problem and the function of the processing equipment is unaffected.

While not replacing more sophisticated techniques for extensive surface marking, the use of Tipp-ex can satisfy most requirements of the pathologist in routine practice.

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Giant inflammatory polyposis in ulcerative colitis presenting with protein losing enteropathy

Patients with inflammatory bowel disease may show colonic inflammatory polypos which develop from regenerating mucosa surrounded by areas of ulceration. In extreme cases this results in giant inflammatory polyposis (colitis polyposa), which may present with abdominal pain or discomfort, rectal bleeding, a palpable abdominal mass or intestinal obstruction. We believe that this is the first case of giant inflammatory polyposis to present as a severe protein losing enteropathy.

A 40 year old man presented with diarrhoea and rectal bleeding. Proctosigmoidoscopy showed inflamed, oedematous rectal mucosa and a biopsy specimen active, non-specific colitis. This was initially controlled with sulphasalazine, but after four months symptoms returned and he was given prednisolone 40 mg/day. The dose was gradually reduced, but after six months the colitis worsened, his serum albumin concentration fell from 31 to 11 g/l and he developed gross oedema with restlessness and confusion. Colonoscopy showed a relatively normal rectum with a huge polyloid tumour-like mass bulging into the lumen of the sigmoid colon.

Subtotal colectomy was performed with anastomosis of the rectum to the hepatic flexure. Most of the 56 cm segment of resected colon showed gross polyoid mucosal hyperplasia (figure), with finger-like fronds up to 2 cm high. The proximal 4-3 cm seemed to be relatively normal while the distal 5 cm was inflamed, though not polyoid. The bowel wall and mesentry were not thickened.

Histological examination showed mildly active chronic ulcerative colitis with a mixed mucosal inflammatory infiltrate but no ulceration. Glandular architectural disruption and Paneth cell metaplasia extended to the distal resection margin. Superimposed on this was florid regenerative polyp formation.

Inflammatory polypos have been seen in 12-5-19%, of patients with ulcerative colitis, in a smaller proportion of those with Crohn's disease and less commonly in diverticular disease, ischaemic colitis, amoeobiasis and schistosomiasis. They are usually located in the transverse and descending colon and typically appear as fairly flat mucosal islands up to 1-5 cm in height. Occasionally, in association with ulcerative colitis and Crohn's disease, giant inflammatory polyposis, with polyps more than 1-5 cm high, is seen. In most cases radiological and endoscopic appearances have suggested a villous tumour. Previous reports have described either a normal or a moderately reduced serum albumin, but we believe that this is the first case to present with a severe protein losing enteropathy.

Protein losing enteropathy is seen in association with extensive mucosal ulceration, such as severe active inflammatory bowel disease, lymphatic obstruction, such as intestinal lymphangiectasia, and in increased desquamation of intestinal epithelial cells, such as coeliac disease when there is an increased rate of cell turnover, and Menetrier's disease when the epithelial surface area is greatly increased. Mucosal ulceration was not a feature in our patient and therefore cannot be held responsible for the very low serum albumin concentration. In giant inflammatory polyposis, however, there is obviously an analogy with Menetrier's disease as there is a very considerable increase in epithelial cell surface area, and the rate of cell turnover may also be increased.

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Invasive adenocarcinoma of the breast. Lumpectomy resection margin marked with Tipp-ex white fluid (granular black line) (Haematoxylin and eosin.)

Colon extensively affected by giant inflammatory polyposis which has spared the proximal 6cm. The distal quarter is only slightly affected.
Follicular mucinosis, mycosis fungoides, and acute myeloid leukemia

Follicular mucinosis is a skin condition characterised by papulo-follicular lesions and histologically by mucinous degeneration of the hair follicles. Alopecia and lymphoma may occur, and often mycosis fungoides may develop subsequently. In published series, totalling 153 patients, progression to lymphoma occurred in 20%.

Case report
A 60 year old man was admitted with an itchy papulo-nodular rash, multiple follicular abscesses, and alopecia totalis. Small lymph nodes were palpated in the neck only and there was no hepatosplenomegaly. Previous medical history included alcohol abuse and pulmonary tuberculosis. Investigations showed haemoglobin at 9.8 g/l, white cell count of 4.2 x 10^9/l with normal white cell proportions, and a platelet count of 202 x 10^9/l. Red cells were dimorphic, with normocytic and hypochromic forms. Skin biopsy specimens showed mucinous degeneration around hair follicles characteristic of follicular mucinosis. Atypical lymphoid cells were also seen in the dermis and infiltrating the epidermis (figure), with a lymphoid infiltrate also seen at the epidermal-dermal junction, as found in mycosis fungoides.

A computed tomography scan showed hilar, splenic, paraaortic and paracaval lymphadenopathy suggestive of lymphoma. No lymphomatous infiltratation was seen on bone marrow examination, but there was noticeable dyserythropoiesis and abnormal megakaryocytes as well as 39%, myeloblasts and 21%, lymphocytes. Ringed sideroblasts were not found. The blasts were myelomonocytic; Sudan black and naphthol fast esterase positivity confirmed a diagnosis of acute myelomonocytic leukaemia (FAB: M4). Chromosome analysis of the bone marrow showed 66%, abnormal mitoses with hetero- ploidy, 47-48 or more chromosomes, breakages and aneuploidies. Although haemoglobin electrophoresis was normal, haemoglobin H inclusions were detected after incubation with Brilliant cresyl blue.

Immunofluorescence studies of the bone marrow showed that the blasts were positive for the myeloid markers CD13, 14, and 33. In the mononuclear layer 52% of cells were positive for lymphoid markers—that is, CD19 (B cell) 26%, CD2 (T cell) 26%—of which 29%, were CD4 and 3%, CD8 positive. Immunoperoxidase studies on the skin biopsy specimens showed CD4 positivity at the epidermal-dermal junction, characteristic of mycosis fungoides. CD13 and 14 positive cells were also present in the dermis. These probably represented blast cells as morphologically recognisable neutrophils and monocytes were scarce.

One week after diagnosis myeloblasts were seen in the blood in rapidly increasing numbers. Remission induction was attempted with a standard seven day regimen (daunorubicin, cytosine, arabinoside and thioguanine). He had a succession of infectious episodes and died subsequent to a chest infection, with mucormycosis and upper respiratory infection; a post mortem examination showed fungal pneumonia.

Comments
This patient had follicular mucinosis, mycosis fungoides, and acute myeloid leukaemia. The association between the two skin disorders is well recognised, but there is only one report linking dysmyelopoiesis with the Sézary syndrome. More generally, an association between lymphoproliferative and myeloproliferative disorders is now recognised.

In this patient the presence of H inclusions in the red cells indicated leukemic transformation of an early progenitor or stem cell. It seems unlikely that the skin and marrow disorders were unconnected. Primitive myeloid cells were present in the dermis; in the bone marrow the normal predominance of CD8 compared with CD4 lymphocytes was replaced by a CD4:8 ratio of 11:1. This suggests that an abnormal lymphoid proliferation was present in the bone marrow in addition to acute myeloid leukaemia.

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