J Clin Pathol 1990;43:608–610

LETTERS TO THE EDITOR

Salivary duct adenocarcinoma

Salivary duct adenocarcinoma is a distinctive but rarely reported clinicopathological entity.[1-4] The tumour is defined as a high grade malignancy and shares similar histopathological features with the infiltrating duct carcinoma of the breast. The neoplasm is thought to arise from the excretory ducts of major salivary glands, primarily the parotid gland.[4]

We recently observed a typical case with an extremely aggressive clinical course. A 56 year old man presented with a tumour mass on the left side of his neck. The tumour was 6 cm in maximum diameter. Physical examination along the lower neck showed that he had lymphadenopathy. Radical parotidectomy and bilateral lymphadenectomy were performed. Macroscopically, the mass was a firm, indurated, and infiltrating neoplasm with necrotic areas. Peripheral areas of undamaged parotid gland were observed. Microscopic study (figure) showed the tumour to be an uncapsulated neoplasm composed mainly of cellular lobes and nests with solid, trabecular, or cribiform arrangement. Comedo necrosis was seen focally. A desmoplastic reaction was present in the stroma. Proliferating cells exhibited prominent atypia and mitoses. Lymph node metastases showed similar features. The patient died of neoplastic dissemination eight months later.

First described by Kleinsasser *et al* in 1968,[1] salivary duct adenocarcinoma is rare. To date, about 30 cases have been reported.[1-4] Its scarcity is reflected by the fact that no specific mention about this neoplasm appears in the largest series of salivary gland tumours reported by Eveson and Cawson in 1985.[5]

The hypothesis proposed by Batsakis about the histogenetic development of salivary gland and its derived tumours has been widely accepted.[6,7] According to this author,[7] myoepithelial cells play a decisive part in the development of salivary gland tumours, and probably modify their prognosis. In this sense salivary carcinomas in which myoepithelium plays an active part are, like those in the breast, low grade neoplasms arising from the intercalated duct unit.[7] On the other hand, excretory duct and its presumptively derived tumours, such as duct adenocarcinomas, squamous carcinomas, and mucoepidermoid carcinomas, are devoid of a myoepithelial component,[4,7] and characteristically, all of them take an aggressive clinical course.[7]

Morphologically, salivary duct adenocarcinoma closely resembles its mammary counterpart. In this sense Garland *et al* believe that epithelial nest arrangement and comedo-type central necrosis are the most useful criteria for diagnosis,[3] but other patterns such as solid, cribiform, desmoplastic, and papillary are also seen.

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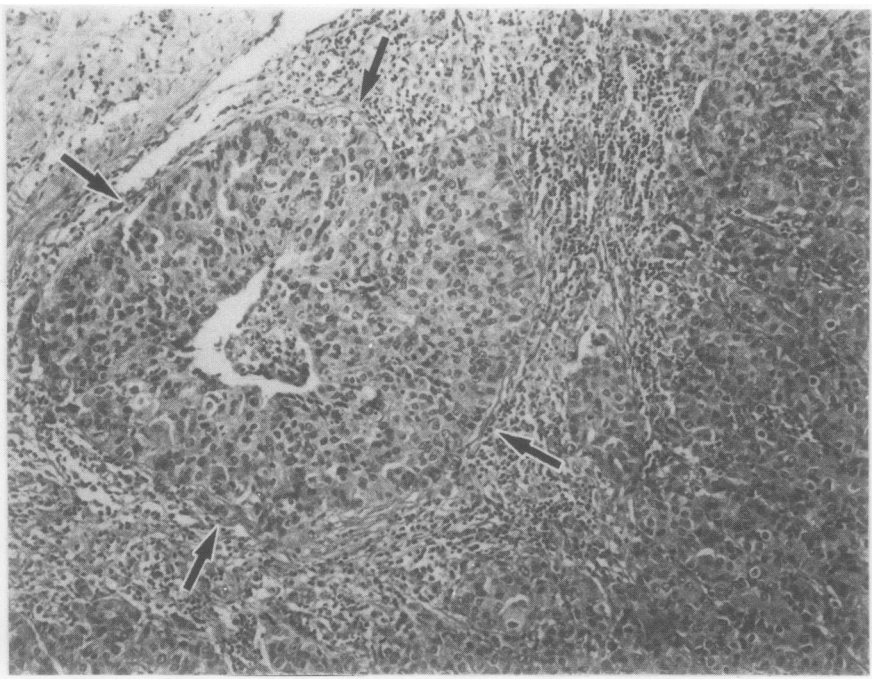

Microscopic view of a neoplastic duct showing comedo necrosis (arrows). (Haematoxylin and eosin.)

Alcian blue: reliable rapid method for marking resection margins

Biopsy specimens taken from women with mammographic abnormalities found on screening require careful macroscopic and microscopic evaluation.[1] Accurate identification of resection margins is vital, and several methods of marking resection margins have been reported,[2-4] each of which has several disadvantages. India ink is messy and takes a long time to dry. Artists' pigments permit differential marking but are expensive and being radio-opaque are unsuitable for specimens that are subsequently *x* rayed.

In our laboratory breast lumps are *x* rayed whole after overnight fixation in 10% buffered formalin. They are then dipped into a container of 1% alcian blue (BDH Limited, Poole, Dorset) for a few seconds, removed, and dried with paper towels. Despite drying, sufficient stain remains on the outer surface (figure). The specimen is then sliced and the slices *x* rayed. After routine processing the alcian blue is clearly visible along the resection margins in sections stained with haematoxylin and eosin.

We have found this system of marking to be reliable, quick, and cheap. Tissue can be *x* rayed after marking and the alcian blue is clearly visible in the stained sections. Dipping the specimen into alcian blue gives a more uniform covering and is quicker than painting with a brush. Dipping into alcian blue, however, is not suitable for specimens that have been incised in the operating theatre (a practice we deplore); for these, the alcian blue can be painted on with a brush.

This method has proved so successful in our laboratory that we now use it routinely on any specimen where excision margins are relevant and likely to be difficult to assess.

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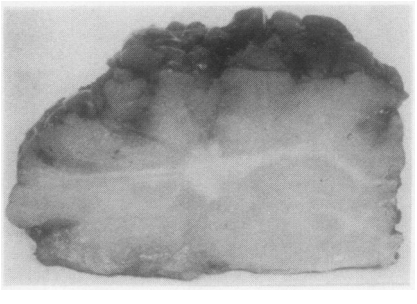

Cross section of a specimen showing staining of resection margins and central tumour.

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Congenital malaria in one identical twin

Congenital malaria is rare in Britain. We report uniovular twins, only one of whom developed congenital *Plasmodium vivax* infection.

A healthy primigravid woman delivered full term twins four months after her arrival in England from India. The delivery was normal, and the placenta was undamaged and consistent with a monozygotic pregnancy. Haemoglobin estimations at birth suggested a twin-to-twin transfusion (18·0 g/dl and 26·5 g/dl for twins I and II, respectively). Twin II therefore had one third of her blood volume exchanged with plasma protein fraction; thereafter her progress was uneventful.

When reviewed one month later, the mother and both twins had no fever and physical examination showed no abnormal physical signs. Repeat estimates of haemoglobin concentration were 7·5 g/dl (twin I) and 13·0 g/dl (twin II). Trophozoites and gametocytes of *Plasmodium vivax* were observed on the blood film of twin I (parasitaemia 0·3%) but not in repeated thick and thin blood films from Twin II. Scanty infestation with *Plasmodium vivax* was also present in the mother (thick film only). (These findings were later confirmed by the Malaria Reference Laboratory at the Hospital for Tropical Medicine and Hygiene.) IgM antimalarial antibodies were detected in serum from twin I but not from twin II (table), consistent with congenital infection in the former; IgG antibodies, presumably of transplacental origin, were present in both. Chloroquine was administered to the mother and both twins (10 mg base/kg initially, repeated after six hours, and then daily for five days), and all three were well when reviewed three months later.

Since 1980, less than 20 cases of congenitally acquired malaria have been reported in Britain. A case of congenital malaria has previously been described in the second-born of non-identical twins[1]; premature placental separation was implicated. We believe ours to be the first reported case of congenital malaria in one of a pair of identical twins. As both twins were probably exposed to *Plasmodium vivax* during birth, the exchange transfusion of the second twin may have removed infected erythrocytes and thus protected against the development of clinically important parasitaemia. It seemed reasonable to suppose that both twins could be infected, however, and so both were treated. As congenitally acquired malaria does not involve passage of sporozoites, an exoerythrocytic cycle does not occur. Chloroquine alone was therefore the treatment of choice.[1,2]

Malarial immunofluorescent antibody test reactions in two identical twins

	Twin I	*Twin II*
IgG	++++	+++
IgM	+++	—

Reactions were graded visually from absent (−) to very strong (++++). The malarial antigen used was from *Plasmodium fieldi*.[1]

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Survival of Helicobacter pylori in water and saline

Helicobacter (Campylobacter) *pylori* is a ubiquitous human parasite and the most common and important cause of gastritis.[1] The natural reservoir for this organism is probably the human stomach, but the mode of transmission from person to person remains unknown. Although *H pylori* has never been isolated from an environmental source, recent studies[2] have shown that, like *Campylobacter jejuni*,[3] it may survive in fresh water microcosms in a viable state for more than 10 days and as viable, non-culturable coccoid bodies for up to one year.

We studied the survival of *H pylori* (NCTC) 11916) in sterile distilled water, physiological saline, and artificial seawater.[4] Viable counts were determined using a standard microbiological dilution method, with inoculation on to a selective isolation medium for campylobacters (modified New York City medium). Plates were incubated under microaerophilic conditions at 37°C for three to four days. Colonies were counted and expressed as colony forming units per millilitre (cfu/ml) and also tested for rapid urease, oxidase, catalase, and Gram reactions to confirm the identity of surviving organisms. Counts were repeated at daily intervals from suspensions stored at room temperature and at 7°C in the dark. Experiments were performed in duplicate.

At 7°C, *H pylori* (NCTC 11916) remained viable for culture when suspended in distilled water for a period of between 11–14 days, saline for 16 days, and artificial seawater for between three and seven days (figure). Suspensions stored at room temperature, however, became non-culturable within one day of inoculation in distilled water and seawater, and within three days in physiological saline. Similar results were found with *H pylori* (NCTC 11639) and a recently isolated strain.

The non-culturable bacterial suspensions contained coccoid forms of the organism, which have been shown to be viable.[2] Coccoid cells of *C jejuni* may be transformed into culturable, spiral forms by animal passage.[3] Water borne outbreaks of campylobacteriosis have been described,[3] and urease positive campylobacters have been isolated from the roots of aquatic plants, from freshwater and seawater.[5]

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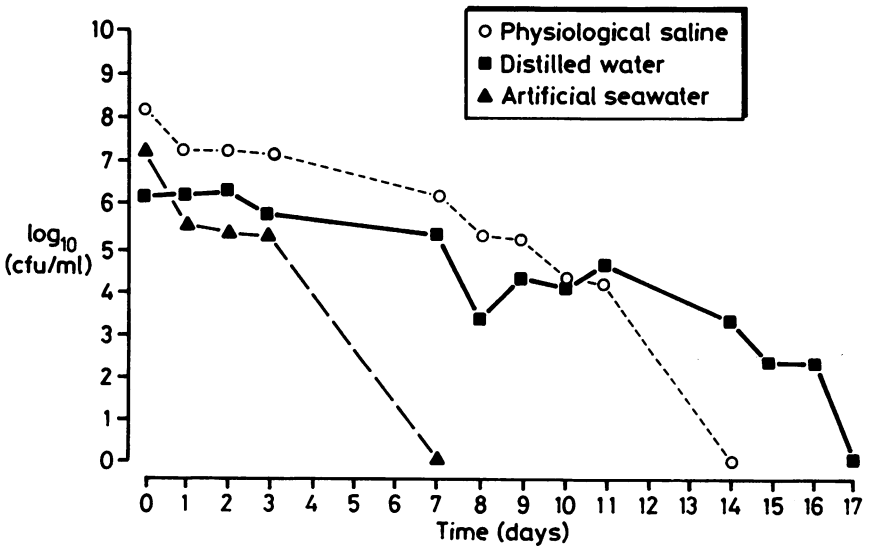

Survival of H pylori (NCTC 11916) in distilled water, physiological saline, and artificial seawater at 7°C.