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Case report

Squamous cell carcinoma in a duplicate large intestine

A case report

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Summary. A case report of a squamous cell carcinoma arising in a closed duplication of the large intestine is presented. The aetiology of squamous cell carcinoma in this site is discussed. It is quite possible the epithelial changes described in this case are similar to those associated with squamous cell carcinoma in other anatomical sites. The unusual environment within the lumen of a closed duplication may be carcinogenic.

Key words: Large intestinal duplication – Squamous cell carcinoma

Introduction

A case of primary squamous cell carcinoma (SCC) arising in a congenital duplication of the large intestine is presented. This is a rare tumour and the aetiology is discussed with reference to the epithelial changes found in the colonic mucosa. It is possible the series of epithelial changes resulting in SCC at this site correspond to those found in the lung.

Case history

A 48 year old woman presented with a mass in the right iliac fossa. She complained of no bowel symptoms. At laparotomy a tumour was attached to the mesentery of the caecum and ascending colon. The ovaries were normal. A right hemicolectomy was performed and the patient made an uneventful recovery.

Results

Macroscopical appearances

A right hemicolectomy specimen in which 3 cm medial and parallel with the ascending colon in the mesentery was a firm ovoid mass measuring 8 cm × 7 cm. The mass when cut was partially cystic and contained a solid white area with foci of necrosis (Fig. 1).

Microscopy

Histology showed a closed duplicated segment of large intestine with all layers of the bowel wall represented. The lumen of the duplication corresponded to the cystic area within the mass and was lined by large intestinal and stratified squamous epithelium (Fig. 2). The squamous epithelium showed varying degrees of dysplasia ranging from mild to carcinoma in-situ. Arising from the dysplastic epithelium was a moderately differentiated keratinising SCC (Fig. 3) which infiltrated into but did not completely penetrate the muscularis externa. No glandular differentiation or mucin was demonstrated. All lymph nodes showed reactive features only. The remaining large and small intestine were of normal histological appearance.

Discussion

To date only six cases, including ours, have been reported of a malignant tumour arising within a duplication of the colon. Of these, two were adenocarcinomas, three were SCC and no histological diagnosis was given in the remaining case (Hickey and Corson 1981). Pure SCC of the colon and rectum are rare tumours with a reported incidence of 0.3–0.5 per 1000 malignancies of the large colon (Vezeridis et al. 1983). Two of the previously described SCC arising within a large bowel duplication have been found in areas of heterotopic epithelium of salivary and pancreatic type (Hickey and Corson 1981). The two reported adenocarcinomas both arose from colonic epithelium (Hickey and Corson 1981). Interestingly, all 6 cases have

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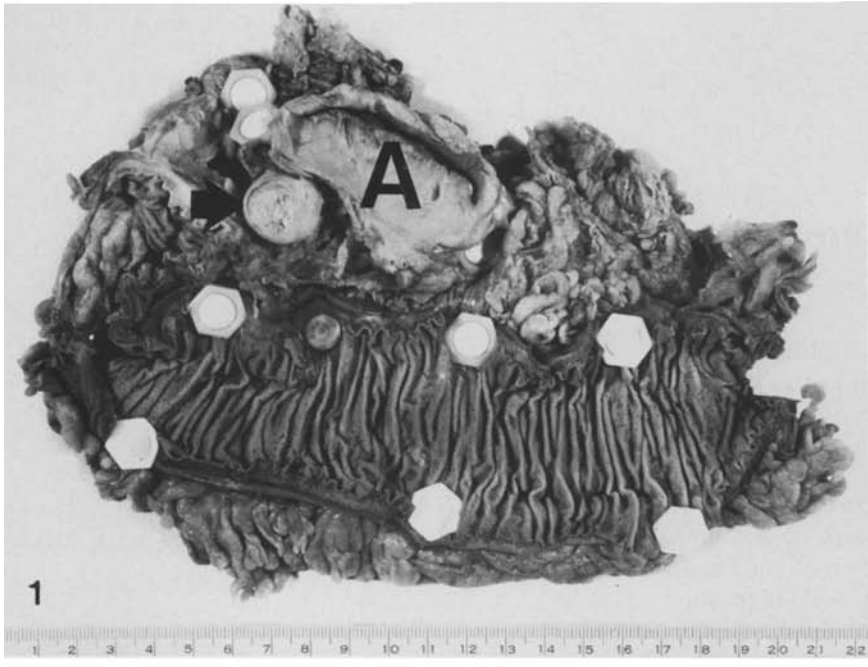

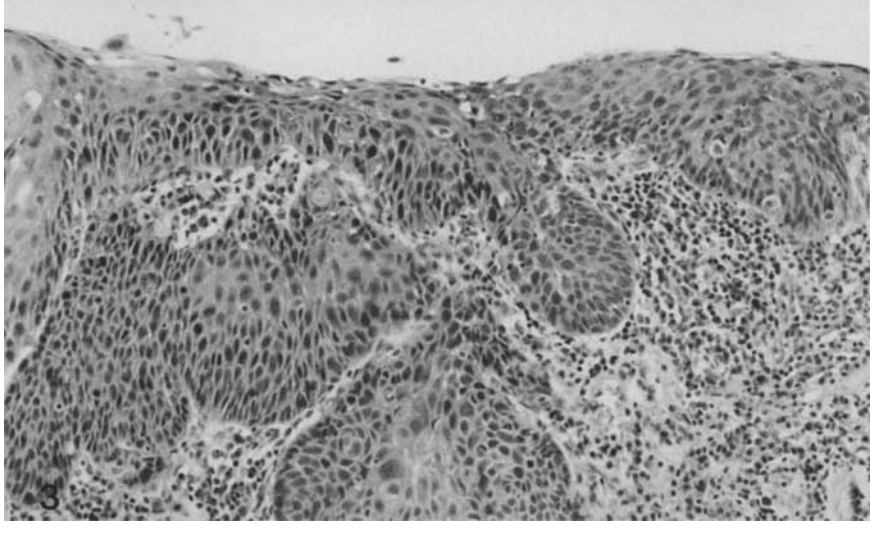

Fig. 1. Duplicated large intestinal segment showing the mass containing a compressed cystic area (A). An enlarged lymph node (*arrowed*) is present adjacent to the mass

Fig. 2. Part of the cyst lined by large intestinal epithelium on the right which merges with stratified squamous epithelium on the left. These overlie an infiltrating squamous cell carcinoma within the deeper tissues. Original magnification ×20

Fig. 3. Infiltrating squamous cell carcinoma originating from the overlying squamous epithelium which shows changes of carcinoma in-situ. Original magnification ×40

been found in women between the ages of 44 and 61 years and all were found in the right side of the colon.

The right and transverse colons have the highest incidence of SCC in the anatomically normal large bowel (Lundquest et al. 1988). These sites also correspond to the distribution of colonic duplications associated with SCC in the literature. It is of interest to note an adenomatous polyp containing SCC has been described in this part of the large bowel (Lundquest et al. 1988).

Several theories have been forwarded to explain the aetiology of SCC in the normal colon (Williams et al. 1979; Hickey and Corson 1981; Pitella and Torres 1982). Most consider squamous metaplasia to be the precursor of epithelial dysplasia which eventually leads to invasive SCC (Gusterson 1984). However, squamous metaplasia is extremely rare in the bowel (Morson and Dawson 1979). Squamous metaplasia has been found in 3 out of 750 adenomatous polypi of the large bowel and it was thought that the squamous component arose from and eventually replaced the adenomatous part of the polyp (Williams et al. 1979). This may provide a potential site from where SCC could develop and is in accord with the finding of squamous metaplasia and dysplasia in the epithelium surrounding a SCC in the large bowel (Morson and Dawson 1979). In the lung the sequence of changes in the bronchial mucosa associated with SCC includes metaplasia, dysplasia and carcinoma in-situ (Gusterson 1984). A similar sequence of epithelial changes are noted in longstanding ulcerative colitis where SCC is found in the colonic mucosa containing squamous metaplasia and this is the result of longstanding chronic inflammation (Morson and Dawson 1979). In this example SCC had arisen in colonic epithelium independent of adenomatous polypi.

Carcinogenesis is thought to involve a sequence of steps which is initiated by a specific carcinogen. The end result produces an abnormal pattern of differentiation and proliferation in the cells of the target tissue (Wright 1983). The genome of all cells contains the necessary genetic information to allow cell differentiation to follow several alternative patterns. The effect of the carcinogen will be to inhibit normal cell differentiation resulting in the accumulation of undifferentiated cells. An important feature of these cells is that, unlike the fully differentiated cell, they retain the capacity for division and proliferation (Ruddoch 1987). These cells may represent the precursors from which metaplastic and dysplastic epithelia originate (Robbins et al. 1984). The epithelial changes associated with the development of SCC in the lung are also found within the mucosa surrounding SCC in the colon (Morson and Dawson 1979). It is possible that a carcinogen in either the colon or a closed colonic duplication initiates abnormal cell differentiation resulting in squamous metaplasia and dysplasia. As a consequence of prolonged contact between the carcinogen and the colonic mucosa, dysplastic changes in the metaplastic epithelium may occur which ultimately lead to carcinoma, but it is not known why the resulting tumour is a SCC rather than an adenocarcinoma.

The environment inside a closed duplication may therefore, provide favourable conditions for the initiation of carcinogenesis. The prolonged contact of a carcinogen upon the lining mucosa may not, however, represent an important factor in this sequence because SCC are also found in the normal colon. Furthermore, the closed duplication will not come into contact with partly digested foodstuffs suggesting that in the development of SCC in the large intestine the carcinogen may not be present within dietary constituents.

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