

### Discussion

Adenocarcinoma of the anal canal is rare. The tumour may present as an obvious mass lesion in the anal canal but may lie undetected in a perirectal abscess or a chronic anal fistula.<sup>4,5</sup> Biopsy of all perianal abscesses and fistulas is therefore recommended. The origin of adenocarcinoma of the anal canal is often debated but its common association with a perianal abscess or anal fistula prompted the suggestion that anal adenocarcinomas arise in a pre-existing anal fistula. Other theories suggest that it may originate in reduplication of the gut, implantation from a rectal carcinoma, or in anal glands of the anal transitional zone.<sup>4,5</sup> The association of this patient's adenocarcinoma with an in situ lesion of the anal glands is strong evidence that some adenocarcinomas of the anal canal arise from anal glands. The association of anal gland carcinoma with in situ squamous carcinoma of the anal canal has not to our knowledge been reported previously and is reminiscent of similar lesions in the uterine cervix.<sup>6</sup> The association of HPV with squamous carcinomas of the anal canal has been established in recent years. Koilocytes were identified in this patient's tissues, although dot blot hybridisation showed only weak positivity for HPV subtypes 16, 18. This may be due to the presence of low viral copies and the relatively low sensitivity of dot blot hybridisation. It is tempting to postulate that, as in the uterine cervix, HPV infection may have an aetiological role in both

glandular and squamous carcinomas of the anal canal.

### Conclusion

The presentation of the invasive lesion as a non-healing anal fistula reinforces the need to biopsy these lesions to exclude malignancy. The identification of an in situ carcinoma of anal glands lends support to one theory that anal adenocarcinoma may arise from anal glands. Most interestingly, simultaneous glandular and squamous carcinoma associated with HPV infection suggests an analogy with similar simultaneous lesions in the uterine cervix and may point to an aetiological role for HPV in primary adenocarcinoma of the anal canal.

- 1 Fenger C. Anal canal tumours and their precursors. *Pathology Annual Part 1*. East Norwalk, Connecticut: Appleton & Lange, 1988:45-66.
- 2 Talbot R. Changing nature of anal cancer. *BMJ* 1988;297:239-40.
- 3 Schneider A, Grubert T. Diagnosis of human papilloma virus infection by recombinant DNA techniques. *Clin Obstet Gynaecol* 1989;32:127-40.
- 4 Zaren HA, Delone FX, Lerner HJ. Carcinoma of the anal gland; case report and review of the literature. *J Surg Oncol* 1983;23:250-4.
- 5 Nelson RL, Prasad ML, Abcarian H. Anal carcinoma presenting as a perirectal abscess or fistula. *Arch Surg* 1985;120:632-5.
- 6 Anderson MC. Premalignant and malignant disease of the cervix. *Obstetrical and gynaecological pathology* Vol I 3rd ed. Edinburgh: Churchill Livingstone, 1987:289.

## Combined high grade sarcoma and serous ovarian neoplasm

C Allen, M Stephens, J Williams

### Abstract

**A case of an ovarian serous epithelial neoplasm of borderline type admixed with sarcomatous elements is reported. This combination seems to be extremely rare with only four cases previously reported. It may represent a type of collision tumour or the development of a sarcoma in a growth with borderline differentiation.**

### Case report

#### CLINICAL FEATURES

A 63 year old woman (gravida 2 para 2) with no relevant medical history presented with a two

week history of lower abdominal pain and distension. Examination showed a tender, firm mass arising from the pelvis and extending to the left of the umbilicus. At laparotomy she was found to have ascites, a left ovarian tumour, equivalent in size to a 20 week gestation, probable tumour in the smaller right ovary, and probable deposits within the pouch of Douglas, omentum, and para-aortic nodes. Most of the left ovary was removed with difficulty. The right ovary was adherent to bowel and could not be dissected free. Lymph nodes, omentum, and peritoneum were not sampled. The patient was subsequently given chemotherapy. She died 11 months after diagnosis.

Histopathology  
Department, Central  
Pathology Laboratory,  
Hartshill, Stoke on  
Trent ST4 7PA

C Allen  
M Stephens

Department of  
Radiotherapy, North  
Staffordshire Royal  
Infirmary, Hartshill,  
Stoke on Trent  
J Williams

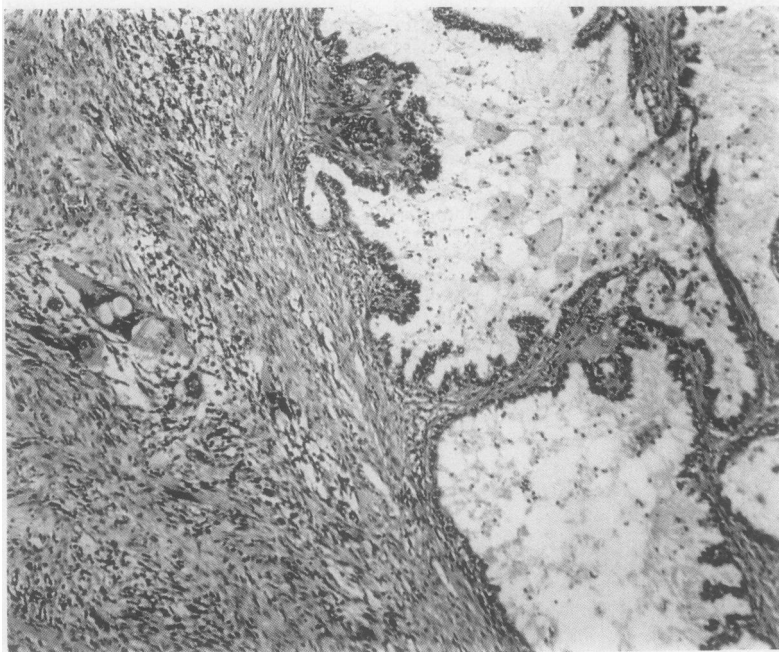
Correspondence to:  
Dr M Stephens

Accepted for publication  
27 August 1991

#### PATHOLOGICAL FINDINGS

Macroscopically the left ovary measured 17 cm in diameter, with a solid and papillary area contiguous with a myxoid, focally haemorrhagic area measuring up to 12 cm in maximum dimension.

Histologically the solid and papillary component was a papillary and adenofibromatous serous neoplasm of borderline malignancy with no evidence of stromal invasion. The myxoid component was a high grade sarcoma consisting of pleomorphic spindle cells and round cells with abundant eosinophilic cytoplasm. Cross-striations were not seen. Immunohistochemistry showed this component to be negative for epithelial markers (CAM 5-2, CEA, and EMA) but positive for vimentin and desmin, consistent with rhabdomyoblastic differentiation. For the most part the two components were clearly separate (figure), but there was one small focus where clearly sarcomatous stroma intervened between epithelial elements.



*Juxtaposition of sarcomatous and adenofibromatous areas with multinucleated rhabdomyoblasts in stroma (haematoxylin and eosin).*

#### Discussion

This tumour is an admixed serous epithelial serous adenofibroma of borderline malignancy and sarcoma showing rhabdomyoblastic differentiation. Although there might have been a common origin for both components, it is also consistent with juxtaposition of two components of different origin. This combination seems to be extremely rare with only four previously reported cases.<sup>1-4</sup> A fifth case was found to be an undifferentiated carcinoma with spindle cell differentiation.<sup>3</sup>

The histogenesis of combined ovarian tumours remains uncertain and while some authors consider they are an early form of malignant mixed mesodermal tumour<sup>3,4</sup> (the histogenesis of which is equally unclear), others suggest they may be collision tumours.<sup>5</sup> We believe that the coexistence of a serous papillary adenofibroma with a high grade sarcoma showing rhabdomyomatous differentiation represents either a collision tumour or the development of a sarcoma in a pre-existing borderline tumour. The development of high grade or anaplastic tumour foci in tumours is recognised at other sites, such as pleomorphic salivary adenomas.

Although we have no specific supportive evidence, we suspect that sarcomatous change in ovarian epithelial tumours may be more common than previously published reports suggest, and we emphasise the need to sample carefully areas with an unusual gross appearance in otherwise typical ovarian tumours.

- 1 Von Numers C, Mikkonen R. Leiomyosarcoma arising in serious cystadenoma of ovary: report of a case. *Ann Chir Gynaecol Fenn* 1960 1949;240-4.
- 2 Watts AE, Lichtenstein I. Primary leiomyosarcoma associated with serous cystadenocarcinoma of the ovary. *Gynaecol Oncol* 1977;5:81-6.
- 3 Clarke TJ. Sarcoma-like mural nodules in cystic serous ovarian tumours. *J Clin Pathol* 1987;40:1443-8.
- 4 McCullough J, Batman P, Parker D. Sarcoma-like nodules in cystic ovarian tumours. *J Clin Pathol* 1988;41:1028-9.
- 5 Prat J, Fox H. Mesenchymal tumours of the ovary. In: Fox H, ed. *Haines and Taylor Obstetrical and Gynaecological Pathology*. Edinburgh: Churchill Livingstone, 1987: 697-713.