Correspondence

Foam cells and histiocytes in endometrial stromal tumours

Sir: The excellent paper of McCluggage et al.\(^1\) reports one case of low-grade endometrial stromal sarcoma with trabecular sex cord-like areas. In these areas the tumour cells had a rhabdoid morphology. Among the tumour cells there were nests of foam cells, which were considered by authors to be histiocytes. Both ultrastructural and immunohistochemical studies concentrated on the cells of the stromal sarcoma, including those of the sex cord-like areas. However neither the immunophenotype nor the ultrastructure of the foam cells were determined.

Recently we have studied a very similar case in a 52-year-old woman with a 120 $\times$ 140 mm solid mass in her left ovary. A hysterectomy, including both tubes and ovaries, was performed. The ovarian tumour was a fibrothecoma. In the uterus several intramural leiomyomas were found. A submucosal, white-yellow nodule, 25 mm in diameter was also present, which corresponded to a low-grade stromal sarcoma with frequent sex cord-like areas and others composed of epithelioid cells in a trabecular pattern. Some of these epithelioid cells showed intracytoplasmic inclusions and rhabdoid phenotype. Contiguous to the tumour cells, there were numerous groups of cells with large and multivacuolated cytoplasm (Figure 1). Moreover aggregates of histiocytic cells, with clear, not foamy, cytoplasm were also present. In the immunohistochemical study, stromal sarcoma cells and sex cord-like cells were strongly positive for vimentin (Biogenex), AE1-AE3 (Biogenex) (Figure 2) and 8,18-anti-cytokeratin (Medac) antibodies. Focally, the cells of the stromal sarcoma, were also positive with anti-smooth muscle actin (Biogenex) and anti-desmin (Biogenex) antibodies, but negative were the rhabdoid cells of the trabecular areas. Likewise, the foam cells showed cytokeratins (Figure 2) and vimentin in their cytoplasm. However, only some isolated foamy cells were positive for smooth muscle actin. The clear, non-vacuolated cells had an immunophenotype according with its histiocytic morphology, being stained with anti-vimentin and KP1 (Dako) antibodies. Staining for EMA (Dako), CEA (Dako) and S100 protein (Biogenex) antigens were negative in all these cellular types.

The presence of foam cells in endometrial stromal tumours and sex cord-like uterine tumours has been reported\(^2,3\). They may be also present in hyperplasias and carcinomas of the endometrium\(^4\). On electron microscopy abundant lipid cytoplasmic vacuoles and membrane junctions were observed, being partially surrounded by basal membrane and containing scanty lysosomes\(^3,5\). Based on these findings, the foam cells have been classified as specialized, transformed stromal cells instead of true histiocytes. The foam cells in our case showed immunohistochemical features similar to other stromal tumour cells and were negative with the KP1 antibody. So endometrial stromal sarcomas can present tumour foam (non-histiocytic) cells, true histiocytes or, like our case, both cellular types.

D.Suarez Vilela
F.M.Izquierdo Garcia

Servicio de Anatomı´a Patolo´gica
Hospital de Leon
Altos de la Nava s/n
León
Spain

Figure 1. Groups of foam cells close to sex cord-like trabeculae.

Figure 2. Stromal sarcoma cells (left) and foam cells (right) positive with anticytokeratin antibody (AE1-AE3).

Histopathology 1998, 32, 568–578

Composite early carcinoma (ordinary adenocarcinoma, carcinoid, microglandular-goblet cell carcinoid, neuroendocrine mucinous carcinoma) of the stomach

Sir: Mixed or composite gastric carcinomas are rare tumours, characterized by an intimate admixture of glandular and endocrine components with frequent histologic transitions. We have observed a case of composite early gastric carcinoma that showed areas of tubular adenocarcinoma, carcinoid, microglandular-goblet cell carcinoma and areas of mucinous adenocarcinoma with neuroendocrine differentiation.

The patient was a 60-year-old woman. Approximately 2 months prior to admission she developed epigastric pain. Gastric endoscopy revealed a small protuberant lesion with ulcer in the posterior wall of the gastric body, and a subsequent biopsy showed adenocarcinoma. A partial gastrectomy with perigastric lymph node dissection was performed. Postoperative recovery was uneventful, and the patient has been asymptomatic without evidence of recurrence or metastasis for the following 36 months. The resected stomach had a protuberant lesion with ulceration measuring 20×15 mm in the posterior wall of the body. On histological examination the tumour appeared to occupy the mucosa and to infiltrate deeply in the submucosa without extension to the muscularis propria. It was diagnosed as early gastric cancer. Four distinct components of the tumour were readily recognizable showing features of (intestinal type) tubular adenocarcinoma, carcinoid, microglandular-goblet cell carcinoid and mucinous adenocarcinoma. In the deeper part of the mucosa the tumour cells were arranged in a classic carcinoid pattern of solid nests and trabeculae, composed of polyhedral cells with uniform round-to-oval nuclei and finely granular eosinophilic cytoplasm (Figure 1a). The adenocarcinomatous glands were more abundant in the upper half of the mucosa (Figure 1b), and showed no intracytoplasmic mucin. The carcinoid pattern merged in many areas with the adenocarcinomatous pattern (Figure 1b).

microglandular-goblet cell carcinoid was mainly situated in the submucosal layer (Figure 2a). The tumour cells were arranged in small clusters or trabecular cords composed of between three and 12 cells. The nuclei were generally thin and crescent shaped. They were compressed to the rim of the cells by abundant cytoplasm mucin. A peripheral orientation of the nuclei predominated within the cellular clusters (Figure 2a). The cells resembled normal goblet cells of the gastrointestinal tract except for nuclear compression. Tumour cells invaded submucosal lymphatics (Figure 2b). Mucinous adenocarcinoma was found in the submucosa (Figure 2c). It showed extracellular lakes of mucin in which tumour cells were present in small clusters together with neuroendocrine cells (Figure 2c). The mucin stained mainly with Alcian blue, but some traces of diastase–periodic acid–Schiff positive material were also present. The acid

Figure 2. a, Trabeculae of neoplastic goblet cells within the submucosa (haematoxylin and eosin). b, Microglandular-goblet cell carcinoid tumour within submucosal lymphatics. Cluster of serotonin-positive neuroendocrine cells in intimate association with goblet cells (immunoperoxidase–serotonin with haematoxylin counterstain). c, Mucinous adenocarcinoma in the submucosa. Numerous chromogranin A-positive neuroendocrine cells, floating in the mucus lakes (immunoperoxidase–chromogranin A with haematoxylin counterstain).
mucin was of non-sulphated sialomucin type, blue staining with high iron diamine–Alcian blue. The Grimelius silver staining demonstrated that the majority of neuroendocrine cells were argyrophilic, whereas the argentaffin reaction of Fontana–Masson was negative. The six lymph nodes found were free of tumour deposit. Very strong immunoreactivity for chromogranin A was identified in carcinoid nests as well as in neuroendocrine cells of the microglandular-goblet cell carcinoid and the mucinous carcinoma (Figures 1b and 2c). The results of an immunohistochemical search for the presence of specific endocrine products revealed a moderate number of serotonin-immunoreactive cells within the neuroendocrine portion of the tumour (Figure 2b), but no evidence of gastrin, somatostatin, pancreatic polypeptide, VIP, ACTH, glucagon, or calcitonin. In the non-malignant gastric mucosa there was a nonerosive, non-specific gastritis without evidence of Helicobacter pylori infection. Chromogranin-positive cells were singly scattered and did not form nodular aggregates.

Our case was not associated with atrophic gastritis, metaplastic epithelium, dysplasia or with multiple proliferative lesions, such as intramucosal carcinoid and endocrine cell proliferations of the micro-nodular and linear type, which are currently regarded as carcinoid precursor changes.[1][2]. Therefore, we suggest that the composite early gastric carcinoma arises de novo from pluripotential neck stem cells, which are capable of endocrine and epithelial differentiation. The intimate admixture of the different cell types and patterns within the tumour is also compatible with this hypothesis.

In 1991, Yang and Rotterdam[4] reported one case of mixed carcinoid-adenocarcinoma of the stomach and reviewed 20 cases from the literature, approximately evenly divided between well differentiated and poorly differentiated adenoendocrine cell carcinoma. Recently, a composite carcinoma of the stomach consisting of endocrine and mucous epithelial cells with interspersed amphicrine cells was reported[5]. The present case differs from that reported previously in its range of histopathological patterns including areas of carcinoid, microglandular-goblet cell carcinoid, tubular adenocarcinoma and neuroendocrine mucinous adenocarcinoma.

### Solitary fibrous tumour of the submandibular gland

**Sir:** We describe a solitary fibrous tumour (SFT) within the submandibular salivary gland, a rare location. Although most evidence favours an origin from mesenchymal cells[1][2], the histogenesis of these tumours is still uncertain. An immunohistochemical analysis of cellular markers has been performed and, to further investigate the nature of the lesion, the extracellular matrix has also been examined.

A 46-year-old woman noticed a painless swelling in the right submandibular region, that had appeared about 1 year before. Physical examination revealed a well circumscribed, nontender nodule in the right submandibular gland, and the patient underwent surgical excision of the affected gland. The resected submandibular gland contained a well circumscribed, firm, grey-pinkish 23 × 19 mm nodule with a central brownish cystic cavity (Figure 1). Microscopically, the lesion comprised a patternless proliferation of spindled-to-ovoid cells associated with a fibrous matrix (Figure 1). Vascularity was prominent, and in some fields suggested a haemangiopericytic pattern. Tumour cells exhibited a diffuse, strong immunoreactivity for vimentin and CD34 antigen, and a moderate cytoplasmatic staining for bcl-2 protein. Apart from vascular basement membranes, the tumour tissue resulted negative for laminin and type IV collagen. However, in focal areas, a patchy pericellular staining for both of these substances was seen in the tumour (Figure 2). The abundant fibrous matrix interposed among tumour cells

---

**R.A.Caruso**

**M.F.Heyman**

**L.Rigoli**

**C.Inferrera**

Department of Human Pathology and *Medical Genetics, University of Messina,*

---


was immunoreactive mainly for type III collagen (Figure 2) and, to a lesser degree, for fibronectin and tenascin. Scant CD68-positive macrophages were present, whereas antibody to Factor XIIIa stained many histiocytes interspersed among tumour cells.

The location of SFT within the salivary glands is very unusual, and only occasional examples have been documented in the literature. On ultrastructural examination, presence of basement membrane material around neoplastic elements has been described in some but not all SFTs. We found in the present case a focal patchy immunostaining for both basement membrane components type IV collagen and laminin around tumour cells. This finding agrees with the ultrastructural evidence of basement membrane in some SFTs reported in literature and, in turn, could be in keeping with the postulated differentiation of the tumour cells towards myofibroblasts which are known to be partly enveloped by a basement membrane. Most of extracellular matrix was immunoreactive for type III collagen, fibronectin and tenascin, all of which are mesenchymal-type matrix substances that have been found in lesions with fibroblastic/myofibroblastic differentiation. In our case the only immunohistochemically detectable cytoskeletal filament was vimentin that, in absence of any ultrastructural evidence, is not specific to substantiate a myofibroblastic character of the tumour cells. Due to their capability to undergo morphofunctional modulation in response to environmental stimuli, fibroblasts seem to represent an extraordinarily heterogeneous cell population, among which the myofibroblastic phenotype is only one type.

As observed in other SFTs, our tumour exhibited a vimentin-CD34-bcl-2-positive phenotype and, in addition, comprised a population of factor XIIIa-immunoreactive cells. The latter have often been found in CD34-positive tumours. Occurrence of vimentin-CD34-positive mesenchymal cells has been observed in both adult and fetal normal submandibular glands (M.Guarino, F.Giordano, and F.Pallotti, unpublished observations). Whether these fibroblast-like cells are the origin of the SFT we described, or whether the vimentin-CD34-positive immunophenotype has emerged during tumour development from relatively more undifferentiated mesenchymal cells, is speculative.

Figure 1. Submandibular gland parenchyma (top) is seen at the periphery of the tumour (bottom). Tumour cells have elongated nuclei and poorly defined cytoplasm (inset) (haematoxylin & eosin).

Figure 2. Immunostaining for extracellular matrix components discloses a patchy pericellular reactivity for type IV collagen. The intercellular matrix shows widespread staining with anti-type III collagen antibody (inset) (ABC method on paraffin sections).


Thymoma arising with a thymolipoma

Sir: On review of a series of thymomas from the personal consultation files of one of the authors (JR), we encountered a unique case of a hitherto undescribed occurrence: a thymoma arising within a thymolipoma.

The patient was a 67-year-old, otherwise healthy female who was found to have an anterior mediastinal mass on radiographic examination. No symptoms of myasthenia gravis were reported. At thoracotomy, a 100 mm, well circumscribed ovoid mass was found attached to the pericardium. Following resection, the patient was free of tumour recurrence for 10 years, after which she was lost to follow-up.

On cut section, most of the 100×60×40 mm lesion was lobulated, soft and yellow, consistent with adipose tissue. At one pole, a 25 mm firm, circumscribed while nodule was identified (Figure 1).

Microscopically, the larger lesion comprised an even admixture of approximately 40% unremarkable mature adipose tissue and approximately 60% normal thymic parenchyma. Both thymic cortex and medulla containing Hassal’s corpuscles were present (Figure 2a). In contrast, the 25 mm nodule was a cellular lesion divided by thin fibrous bands emanating from a fibrous capsule (Figure 2b). It comprised plump epithelioid cells having pale chromatin, prominent nucleoli and pink ill-defined cytoplasm, along with equal numbers of dispersed mature lymphocytes. Mitoses were scarce. Perivascular serum lakes and areas representing medullary differentiation were identified. We categorized this encapsulated thymoma as predominantly mixed under the Lattes–Bernatz classification and cortical under the Müller–Hermelink classification. A thin rim of normal lobulated thymic tissue uninvolved by either thymolipoma or thymoma was microscopically identified at the periphery.

Thymolipoma is usually asymptomatic and incidentally identified as a large, well circumscribed anterior mediastinal mass. Reported cases have reached 16 kg and 360 mm³. Because the mass conforms to the shape of the adjacent pericardium, it can simulate cardiomegaly radiographically². Thymolipoma is distinguished from the more common mediastinal lipoma by the even admixture of thymic parenchyma with adipose tissue throughout and the increase in thymic parenchyma relative to normal for the patient’s age.

While several theories have been proposed, the pathogenesis of thymolipoma remains controversial. One theory, citing the haphazard mixture of thymic parenchyma and fat, holds that it is a thymic hamartoma¹. A malformative theory³ is supported by a reported case in which parathyroid tissue was admixed with a tumour resembling thymolipoma. This case suggests possible origin from aberrant development of the third pharyngeal pouch. Other theories suggest that thymolipoma represents fatty regression of a thymoma or of a previously hyperplastic thymus. Perhaps the most intriguing theory is of thymolipoma representing a benign tumour of specialized thymic stroma⁴ which maintains its relationship with the thymic epithelium as it grows. In this scenario, thymolipoma would be analogous to other tumours of specialized stroma such as fibroadenoma of the breast and adenofibroma of the uterus. To further support this analogy, cases of a malignant tumour of specialized thymic stroma, thymoliposarcoma, have been reported⁵. In these, liposarcoma expands the stroma between lobules of thymic parenchyma, again preserving the relationship of thymic stroma to epithelium.

This unique case of thymoma arising within thymolipoma may be interpreted in several ways. One can suggest that it strengthens the link between thymoma and thymolipoma that is supported by their common association with myasthenia gravis, though myasthenia gravis is far more commonly associated with thymic hyperplasia than with either of these two. If one views thymolipoma as a benign tumour of thymic stroma analogous to fibroadenoma of the breast, a thymoma arising within it would be somewhat analogous to the development of epithelial neoplasia in fibroadenoma, such as lobular carcinoma in situ. Of note, a case of thymolipoma showing diffuse thymic epithelial proliferation has been reported.


Jejunal hamartoma as a rare cause of gastrointestinal haemorrhage

Sir: Magnus Alsleben first described myoepithelial hamartomas or adenomyomas in the submucosa of the stomach in 1903. These tumours are very rare. They predominantly occur in the antrum, duodenum and then in decreasing numbers in the remainder of the small bowel. Adenomyomas are considered to be hamartomas since they contain muscular, glandular and fatty elements. We report the second case of a small bowel myoepithelial hamartoma causing gastrointestinal haemorrhage.

A 65-year-old man was referred to our hospital with a 4-day history of melaena. There were no other...
complaints. Laboratory tests showed only an anaemia. Oesophago-gastro-duodenoscopy and colon-radiography revealed no abnormalities. A small-bowel transit clearly showed a side standing mass in the distal part of the jejunum. At laparotomy, an intraluminal tumour was found at about 1 m from the ileo-caecal valve. A segment of small bowel containing the tumour was resected and a primary end-to-end anastomosis performed. Postoperative recovery and follow-up was uneventful; anaemia disappeared. Histologically the lesion was submucosal and composed of glands surrounded by muscular and connective tissue. The epithelium of the glands was cylindric in shape and cystically dilated with no signs of cytological atypia—a jejunal hamartoma (Figures 1 and 2).

Macroscopically the adenomyoma presents as a sessile polyp with a diameter ranging from 5 to 15 mm. It is a submucosal lesion containing glands lined by a columnar epithelium and smooth muscle fibres. Diagnosis is always made postoperatively, simply because most patients have no symptoms. Intussusception is the first complication in the majority of cases. High quality small-bowel transit radiography can only confirm a lesion in the small bowel. But that gives us a broad differential diagnosis. Even if the tumour can be reached by endoscopy, it is still situated submucosally. So in practice only resection and pathological investigation gives a definitive diagnosis.

On reviewing the literature we could only find 13 case reports describing small-bowel myoepithelial hamartomas: eight in the ileum, four in the jejunum and one in a Meckel’s diverticulum. Only one of these patients had anaemia as presenting symptom.

S.H. van Helden
G. Jutten
H. van Hoey
A.M. Dierick*

Departments of Surgery and
*Pathology, KLINa Brasschaat,
Heerlen,
The Netherlands


Genital carcinoma secondary to pagetoid spread from a pagetoid urothelial carcinoma in-situ

Sir:

Pagetoid spread of urothelial carcinoma to the genital tract is a rare phenomenon producing a pattern resembling that of Paget’s disease. We report a case of pagetoid urothelial carcinoma in-situ of the bladder associated with pagetoid spread to the vulva and the cervix. This was associated with bilateral ureteric and urethral pagetoid extension.

A 71-year-old woman presented in 1995 with painless haematuria. Following the diagnosis of carcinoma in-situ, she was treated for 3 months with BCG. Eighteen months later, a routine cervical/vaginal smear demonstrated malignant urothelial cells, although clinical examination was negative at this time and the patient was asymptomatic. As the haematuria and the pelvic pain dramatically increased, a radical cystectomy with hysterectomy and resection of the vagina was performed. The patient is well 3 months after surgery.

Typical carcinoma in-situ was seen in the original biopsies and the cystectomy specimen (Figure 1a). In the bladder, pagetoid changes was extensively present. Similar cells were present in the urothelium of both ureters and the urethra (Figure 1b). They were also identifiable in atrophic epithelium of the vaginal and cervix (Figure 2). There was no evidence of invasive disease. Staining for Alcian blue and PAS confirmed the absence of mucins. The malignant cells were positive for epithelial membrane antigen only and negative for CEA, S100, HMB45 and PSA.

Correspondence 575

Pagetoid changes in urothelial carcinoma in-situ are rare and only 0.6% of all the bladder carcinomas had focal pagetoid features. These changes occur either in pure CIS or in CIS areas accompanied with papillary and/or invasive carcinomas and these changes are often present in patients first treated with radiotherapy, with chemotherapy, by diathermy or by bacillus Calmette–Guerin. Pagetoid changes do not seem to have clinical or prognostic implications. The histogenesis of pagetoid changes in urothelial carcinoma remains unknown. However, non-specific local reaction of the neoplastic cells to injury or loss of E-cadherin expression have been proposed.

The association of genital Paget’s disease with bladder carcinoma has been previously described. Thus, there have been reported cases of Paget’s disease of the glans penis due to a urothelial carcinoma, but there were pagetoid changes in only one case of these bladder carcinomas, and only one case of these tumours was an in-situ carcinoma. Association of Paget’s disease of the vulva with bladder carcinoma has also been documented. Paget’s disease represented only 5% of the carcinomas of the vulva. In this study of 98 cases, only 25% of the cases of vulvar Paget’s disease were associated with other malignancies which were mainly gynaecological carcinomas, and only two cases were related to transitional bladder carcinomas. Three situations can explain the association of a Paget’s disease and a bladder tumour: the coincidental presence of two unrelated malignancies with no continuity between the two lesions; an extension of the Paget’s disease into the urinary tract; and, as in our case, a pagetoid extension to the vulva of the bladder carcinoma.

Our case is unusual because of the extent of the gynaecologic involvement. Pagetoid extension of an in-situ urothelial carcinoma into the vagina up to the cervix has not been previously described. This gynaecologic extension did not give rise to any symptoms and was detected by chance on a routine cervico-vaginal smear. Occult and asymptomatic pagetoid spread is yet known in other localizations, and the pathologist must be aware of the possibility of urethral, ureteral, or genital extension in such pagetoid urothelial carcinomas in-situ or infiltrating. As in our case, frozen section

Figure 1. a. Bladder specimen: urothelial carcinoma in-situ with large atypical cells dispersed in a pagetoid fashion. b. Pagetoid urothelial cells dispersed along the urethral mucosa (haematoxylin & eosin).

Figure 2. The carcinomatous pagetoid cells involved the epithelium of the vagina (a) and the epithelium of the cervix (b) (haematoxylin & eosin).
during surgery can help to verify the status of the surgical margins.

L.Arnould*  
L.Chalabreysse†  
C.Belichard‡  
J.Cuisenier‡  
C.Billerey§  
F.Collin*

Departments of *Pathology and  
‡Surgery, Centre G.F .Leclerc, Dijon;  
†Department of Pathology,  
University Hospital, Strasbourg; and  
§Department of Pathology,  
University Hospital, Besançon, France


Fortuitous diagnosis in a uterine leiomyoma of metastatic lobular carcinoma of the breast

Sir: Tumour-to-tumour metastasis is a rare, but well-described phenomenon. We present a case of metastasis, from a previously undiagnosed breast carcinoma, detected in a leiomyoma during routine microscopic examination of a uterus removed for treatment of menorrhagia.

A 54-year-old woman presented with irregular vaginal bleeding which was unresponsive to norethisterone. An uncomplicated total abdominal hysterectomy and bilateral oophorectomy was performed and the patient was discharged home on oestrogen patch hormone replacement therapy. The patient’s past medical history included investigations for breast lumpiness and an FNAC diagnosis of fibroadenosis on the left. In 1994 the patient had presented with indentation of the skin of the right breast near the nipple. A mammogram was reported as poorly visualized but showing no abnormality. No further investigations were performed.

Macroscopically the specimen weighed 320g and was unremarkable except for a 40 mm diameter fundal fibroid mass. Histological examination of one section showed a typical leiomyoma. However, another section showed an infiltrate of mitotically-inactive small rounded cells with mildly pleomorphic nuclei and a small amount of eosinophilic cytoplasm. These were orientated in single or ‘Indian’-file between the smooth muscle cells (Figure 1a). Further sections failed to reveal a similar infiltrate. The infiltrating round cells were positive for cytokeratin, CAM5.2 (Figure 1b) and negative for desmin on immunohistochemical staining. The small cell size, ‘Indian’-file pattern and immunohistochemical profile in this case allowed a diagnosis of leiomyoma with metastatic adenocarcinoma, probably lobular from the breast, to be made. This finding resulted, on re-examination of the patient, in the discovery of marked right-sided nipple and skin retraction and a 40 mm mass behind the nipple. Fine needle aspiration cytology, mammography and breast

Figure 1. a, H & E. b, Immunohistochemical staining with cytokeratin (CAM5.2).
ultrasound confirmed primary lobular breast carcinoma and no other metastases were seen on bone scintogram or abdominal ultrasound.

Histopathological examination of uterine leiomyomas may result in the diagnosis of unsuspected leiomyosarcoma, or atypical leiomyoma, the main differential diagnosis in this case. In addition, a number of cases have been reported of breast cancer metastases discovered incidentally within leiomyomas\textsuperscript{1–4}. However, in these cases the diagnosis of tumour-to-tumour metastasis was usually made at autopsy in the presence of numerous other metastases or represented an unusual site of recurrence of a known malignancy. Therefore the phenomenon was interesting but did not affect the patient’s management.

The histopathological and immunohistochemical findings in this case were characteristic of metastatic lobular carcinoma from the breast. Interestingly, the atypical cells were present in only one of the six sections examined. The primary lesion had previously remained undiagnosed despite presentation to a breast clinic, clinical examination on several occasions and admission to hospital for hysterectomy. In addition, the patient had been prescribed hormonal treatment for menorrhagia and, immediately following hysterectomy and bilateral oophorectomy, had been commenced on hormone replacement therapy. The histopathological diagnosis of metastatic lobular breast carcinoma enabled hormone replacement therapy to be discontinued and prompted referral for appropriate further investigation and management.

To our knowledge this is the first reported case in which a primary breast cancer was only diagnosed following the fortuitous finding of metastasis to a uterine leiomyoma. This case clearly illustrates the necessity for pathological investigation, with adequate sampling and thorough microscopic examination by a trained pathologist, of all surgical specimens, no matter how apparently routine.

R.D.Liebmann
K.D.Jones*
R.Hamid*
M.Lapsley

Department of Histopathology and
*Obstetrics and Gynaecology,
St. Helier Hospital,
Carshalton,
Surrey, UK