

Correspondence

Small cell melanoma

In their recent paper on small cell malignant melanoma,¹ Blessing and co-workers report a series of 15 melanocytic lesions that, on the basis of their histology, were considered to constitute a new variant of naevoid melanoma—melanoma resembling naevus.

I am concerned about the lack of metastases in the reported series. Only documentation of metastasis constitutes formal proof that the lesions are diagnosed correctly as melanomas; histological resemblance to some features of melanoma by itself can never provide the necessary conclusive evidence. In addition, I cannot agree with the authors that some of the features of these lesions—such as vascular proliferation, lymphocytic infiltrate, and lentiginous junctional component—constitute supportive evidence of malignancy. Furthermore, the authors point out that in some respects the lesions resembled benign naevi; it is unclear why the resemblance to melanoma would be more relevant than the resemblance to naevus. From the illustrations provided in the paper, I am not sure that I would issue a confident diagnosis of melanoma.

One needs to have more cases with follow up to obtain a better picture of the possible malignant potential of these lesions. If no metastases are encountered in an expanded series, then the message of the paper would be very different and similar to the one of Sophie Spitz in her classic paper on what was then termed juvenile melanoma²: the lesions under study resemble melanoma in some respects, but are devoid of malignant potential. Careful correlation of histology with follow up data in a large series is the only way to solve this issue and to know how to interpret such lesions correctly.

W J MOOI

Department of Histopathology, The Netherlands
Cancer Institute, Plesmanlaan 121, 1066 CX
Amsterdam, The Netherlands

1 Blessing K, Grant JJH, Sanders DSA, *et al.* Small cell malignant melanoma: a variant of naevoid melanoma. Clinicopathological features and histological differential diagnosis. *J Clin Pathol* 2000;53:591–5.

2 Spitz S. Melanomas of childhood. *Am J Pathol* 1948;24:591–609.

The authors reply

We would like to thank Professor Mooi for taking an interest in our recent article describing small cell melanoma as a variant of classic melanoma.¹ The points that he makes are of course entirely relevant and are, we feel, generally covered in the manuscript; indeed, the title reflects the fact that the diagnosis might be contentious. Therefore, only the main points of his letter will be discussed.

He rightly comments that there is a lack of metastases in our cases. However, apart from one lesion that measured 1.1 mm, all were less than 1.0 mm, with a mean of 0.63 mm, and therefore the clinical behaviour of these cases is not unexpected. It is of course possible that “small cell melanoma” may have an inherently less aggressive behaviour. In addition, although we accept that metastasis is the

gold standard for the diagnosis of malignancy, histopathologists readily accept basal cell carcinoma as a malignant epithelial neoplasm without metastatic potential and, indeed, those who accept the concept of the radial growth phase in melanoma are quite happy to call these potentially non-metastasising lesions melanomas.

Professor Mooi expresses discontent over the features such as lentiginous junctional growth pattern, lymphohistiocytic infiltrate, and vascular proliferation as being supportive of malignancy. We agree that no feature in isolation is indicative of malignancy and that all features, clinical and histopathological, should be taken into account before reaching a diagnosis. However, two of these features are cited by major texts, and a lentiginous melanocytic growth pattern in an older patient (mean age, 48.6 years) in the absence of trauma is in our opinion supportive of at least in situ disease. The relative importance placed on these features may depend on whether one accepts the entity of dysplastic naevus, and here also we have a controversial entity with the problem of lack of reproducible histological features and disagreement over whether the lesion, if it exists, is a precursor to or risk marker of subsequent melanoma.

Melanocytic lesions comprise a heterogeneous group in which the biological behaviour of some of the more common entities is clearly understood. However, we believe it is essential for the less common entities (such as small cell melanoma) to be recognised and grouped with similar lesions so that accurate conclusions regarding their biological behaviour can be made; unless the entity that we have labelled “small cell melanoma” is clearly defined we will never collect the long term follow up data that will enable an assessment of its true biological potential. Until then, it is important that we all keep an open mind and in the words of the English philosopher Bertrand Russell who on being asked if he would be willing to die for his beliefs replied: “Of course not. After all, I may be wrong.”²

If “small cell melanoma” is the next Spitz naevus that’s OK by us.

K BLESSING
JJH GRANT
D S A SANDERS
M M KENNEDY
A HUSAIN
P COBURN

Department of Pathology, University of Aberdeen,
University Medical Buildings, Foresterhill, Aberdeen
AB25 2ZD, UK

1 Blessing K, Grant JJH, Sanders DSA, *et al.* Small cell malignant melanoma: a variant of naevoid melanoma. Clinicopathological features and histological differential diagnosis. *J Clin Pathol* 2000;53:591–5.

2 Bertrand Russell (1872–1970), English philosopher, mathematician, essayist and social reformer. *The Times book of quotations*. Glasgow: Harper Collins, 2000.

Clear cell carcinoma of the ovary arising in a mucinous cystadenoma

I read with interest the recent case report by Drs Dutt and Berney “Clear cell carcinoma of the ovary arising in a mucinous cystadenoma”.¹ As these authors state, ovarian clear cell carcinomas often arise in endometriosis with a quoted incidence of associated pelvic endometriosis in 50–70% of cases and an incidence of endometriosis in the same ovary of 25%.² This is undoubtedly a gross underestimate of the association between ovarian

endometriosis and clear cell carcinoma because in many cases the tumour will overgrow and completely obliterate the endometriosis. However, my personal experience is that when ovarian clear cell carcinomas are extensively sampled (with special reference to cystic areas) the frequency of endometriosis in these neoplasms is substantially higher and indeed the origin of the tumour in an endometriotic cyst can be identified in most cases.

Drs Dutt and Berney might consider the possibility that the pre-existing cystic areas in their case, in fact, represent an ovarian endometriotic cyst with mucinous metaplasia. Figure 1 looks like the picture often seen in ovarian endometriotic cysts with mucinous metaplasia and fig 2 (right hand side) looks like the atypical changes sometimes seen in such cysts. The single cell lining of clear cells (fig 2, left hand side) is often seen in clear cell carcinomas arising in endometriotic cysts. Mucinous metaplasia can be extensive in ovarian endometriotic cysts³ and, in such instances, a diagnosis of mucinous cystadenoma may be considered. In addition, borderline mucinous tumours may arise in endometriotic cysts.^{3,4} Within the ovary a definitive diagnosis of endometriosis (especially an endometriotic cyst) is often difficult because there may be secondary changes in both the glandular and stromal elements. In particular, in endometriotic cysts, typical endometrial type stroma is often sparse or absent altogether and instead the stroma usually has a fibrous appearance.

There is evidence in the literature that many ovarian endometriotic cysts are, in fact, benign neoplasms and are not related to usual pelvic/abdominal endometriosis. Ovarian endometriotic cysts are often solitary and not associated with generalised pelvic/abdominal endometriosis. Studies, using X linked polymorphic markers, have demonstrated that ovarian endometriotic cysts may be monoclonal,^{5,6} supporting the hypothesis of a benign neoplasm, and DNA aneuploidy has been found in atypical areas.⁷ Because benign and borderline serous and mucinous cystadenomas are common but the corresponding endometrioid neoplasms are rare, it may be that endometriotic cysts with and without atypia correspond to benign and borderline endometrioid cystadenomas, respectively. This would provide an explanation for the coexistence of ovarian endometrioid and clear cell carcinomas with endometriosis.

W G McCLUGGAGE

Department of Pathology, Grosvenor Road, Belfast
BT12 6BA, Northern Ireland
glenm.mccluggage@bll.n-i.nhs.uk

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4 Rutgers JL, Scully RE. Ovarian mixed epithelial papillary cystadenoma of borderline malignancy of Mullerian type: a clinicopathologic analysis. *Cancer* 1988;61:546–54.

5 Nilbert M, Pejovic T, Mandahl N, *et al.* Monoclonal origin of endometriotic cysts. *Int J Gynecol Cancer* 1995;5:61–3.

6 Yano T, Jimbo H, Yoshikawa H, *et al.* Molecular analysis of clonality in ovarian endometriotic cysts. *Gynecol Obstet Invest* 1999;47:41–5.

7 Ballouk F, Ross J S, Wolf BC. Ovarian endometriotic cysts. An analysis of cytologic atypia and DNA ploidy patterns. *Am J Clin Pathol* 1994;102:415–19.

The authors reply

We are grateful to Dr McCluggage for his interest in our paper. We would like to reiterate our report that in this case there were no identifiable endometriotic components: the tumour appeared to be a classic benign mucinous cystadenoma.

He suggests that the mucinous areas might represent an endometriotic cyst with complete mucinous metaplasia. After extensive sampling (as reported), we found no areas of endometriosis. In fact, as we stated, there was a mucin filled multicystic area lined by typical picket fence mucinous cells. This area comprised one third of the total tumour, which was 24 cm in maximum diameter. This seems incompatible with an endometriotic origin. The benign mucinous areas in the tumour are illustrated (fig 1).



Figure 1 Multicystic area of tumour with benign mucinous epithelium, mucin within cysts, and an absence of endometrial stroma.

We entirely agree that clear cells forming a single cell lining are common in clear cell carcinomas arising in endometriotic cysts, but this represents simply a change to clear cell morphology, and cannot be taken to impute the derivation of the benign lesion.

Dr McCluggage suggests that endometriotic cysts can show a lack of endometrioid stroma or that they can be "fibrous". If a multicystic endometriotic cyst has global mucinous metaplasia and a complete lack of endometrioid stroma, then we can only say that in our eyes this would be taken to be a mucinous cystadenoma.

To suggest that a clear cell tumour always arises from an endometriotic cyst seems to be too didactic a viewpoint. Our case demonstrated that other pathogeneses may give rise to clear cell carcinoma.

D M BERNEY
N DUTT

Histopathology Department, St Bartholomew's Hospital, West Smithfield, London EC1A 7BE, UK

Rhabdoid phenotype in cutaneous squamous carcinoma: an earlier report!

We read with interest the case report by Mathers and O'Donnell on squamous carcinoma of the skin with a rhabdoid

phenotype.¹ The authors have indeed beautifully demonstrated the squamous histogenesis in their tumour. However, we are surprised by their statement that this is the "first case of cutaneous malignant rhabdoid tumour showing clear squamous histogenesis".

During their literature search, the authors appear to have missed our paper on a similar topic.² In 1996, we published a report of two squamous carcinomas with a rhabdoid phenotype. One of them was in the skin of an 85 year old man. The neoplasm had areas of conventional squamous carcinoma as well as large areas with a rhabdoid phenotype. The rhabdoid cells had diastase resistant periodic acid schiff material. The rhabdoid cells were positive for cytokeratin, epithelial antigen, and vimentin, but negative with antibodies to desmin, S-100 protein, and HMB45.

Our abstract clearly contained the words "skin", "squamous carcinoma", and "rhabdoid phenotype" and should have been picked up on a MEDLINE search.

S A PAI

Department of Pathology, Manipal Hospital, Airport Road, Bangalore 560 017, India
s_pai@vsnl.com

A M BORGES
C S SOMAN

Department of Pathology, Tata Memorial Hospital, Parel, Bombay (Mumbai) -400012, India

- 1 Mathers ME, O'Donnell M. Squamous cell carcinoma of skin with a rhabdoid phenotype: a case report. *J Clin Pathol* 2000;53:868-70.
- 2 Pai SA, Vege DS, Borges AM, et al. Rhabdoid phenotype in squamous carcinoma: a report of two cases. *Indian J Cancer* 1996;33:161-4.

The authors reply

We thank Pai *et al* for their interest in our paper "Squamous carcinoma of the skin with a rhabdoid phenotype".¹ We apologise for our omission of their previous paper² in our review of the literature. However, we are pleased to hear that other authors have described rhabdoid differentiation within a squamous carcinoma of skin, as we feel this represents an important phenotype, which is predictive of a poor clinical outcome.

M E MATHERS

Department of Histopathology, Royal Victoria Infirmary, Queen Victoria Road, Newcastle upon Tyne NE1 4LP, UK

M O'DONNELL

Department of Histopathology, Freeman Hospital, High Heaton, Newcastle upon Tyne NE7 7DN, UK

- 1 Mathers ME, O'Donnell M. Squamous cell carcinoma of skin with a rhabdoid phenotype: a case report. *J Clin Pathol* 2000;53:868-70.
- 2 Pai SA, Vege DS, Borges AM, et al. Rhabdoid phenotype in squamous carcinoma: a report of two cases. *Indian J Cancer* 1996;33:161-4.

Calendar of events

Full details of events to be included should be sent to Maggie Butler, Technical Editor JCP, The Cedars, 36 Queen Street, Castle Hedingham, Essex CO9 3HA, UK; email: maggiebutler@pilotree.prestel.co.uk

BSCC Annual Scientific Meeting

9-11 September 2001, Majestic Hotel, Harrogate, UK

Further details: BSCC Office, PO Box 352, Uxbridge UB10 9TX, UK. (Tel +44 01895 274020; fax +44 01895 274080; email lesley.couch@psilink.co.uk)

5th International Course on Bone Marrow Biopsy Pathology

Palermo, 3-6 November 2001

Further details: Vito Franco, Istituto di Anatomia Patologica, Università di Palermo, Italy. (Tel +39 091 6553534; fax +39 091 6553521; email: vfranco@unipa.it; website: www.unipa.it/bmcourse)

Current Concepts in Surgical Pathology

12-16 November 2001, The Four Seasons Hotel, Boston, Massachusetts, USA

Further details: Department of Continuing Education, Harvard Medical School, PO Box 825, Boston, MA02117-0825. (Tel +1 617 432 1525; Fax +1 617 432 1562; email hms-cme@harvard.edu; web page <http://www.med.harvard.edu/conted/>)

41st St Andrew's Day Festival Symposium on Therapeutics

6-7 December 2001, Royal College of Physicians, Edinburgh, UK

Further details: Eileen Strawn, Symposium Coordinator. (Tel +44 0131 225 7324; fax +44 0131 220 4393; email 2.strawn@rcpe.ac.uk; website www.rcpe.ac.uk)

Correction

Immunohistochemistry for MSH2 and MHL1: a method for identifying mismatch repair deficient colorectal cancer. *J Clin Pathol* 2001;54:484-7.

In the text the MSH2 and MHL1 genes were sometimes mistakenly written as MSH1 and MHL2, respectively. The authors apologise for this error.



Clear cell carcinoma of the ovary arising in a mucinous cystadenoma

W G McCluggage

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