Correspondence

Small cell melanoma

In their recent paper on small cell malignant melanoma, 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 naevoid.

I am concerned about the lack of metas-
tases in the reported series. Only documenta-
tion of metastasis constitutes formal proof
that the lesions are diagnosed correctly as
melanomas; histological resemblance to some
features of melanoma by itself can never pro-
vide 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 lenticious junctional component—
describing small cell melanoma as a variant of
naevoid melanoma. Furthermore, the authors point
out that in some respects the lesions resembled
benign naevoid; it is unclear why the resem-
blance 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 naevus: the lesions under
study resemble melanoma in some
respects, but are devoid of malignant poten-
tial. Careful correlation of histology with fol-
lower up data in a large series is the only way to
solve this issue and to know how to interpret
such lesions correctly.

1 Blessing K, Grant JJH, Sanders DSA, et al. Small cell malignant melanoma: a variant of naevoid melanoma. Clinico-pathological gen-
tures and histological di-

2 Bertrand B, Bacon C, Modena N, et al. English philoso-


4 Rutgers JL, Scully RE. Ovarian mixed epithelial papillary cystadenoma of borderline malignancy of Mullerian type: a clinicopatho-


7 Nilbert M, Pajovic T, Mandahl N, et al. Mono-


K BLESSING
J J Grant
D S A Sanders
M M Kennedy
A Husain
P Coburn
Department of Pathology, University of Aberdeen, Aberdeen Medical Buildings, Foresterhill, Aberdeen AB25 2ZD, UK

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 metapla-
sia. 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) might be mucinous carcinomas arising in endometriotic cysts. Mucinous metaplasia can be extensive in ovarian endometriotic cysts and, in such instances, a diagnosis of mucinous cystad-

enoma might be considered. In addition, a few borderline mucinous tumours may arise in endometriotic cysts. Within the ovary a definitive diagnosis of endometriosis (es-
cially an endometriotic cyst) is often difficult because there may be no labelling of the re-

sion might be contentious. Therefore, only

Drs Dutt and Berney “Clear cell carcinoma of

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. 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, the only 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 possi-
ble that “small cell melanoma” may have an inherently less aggressive behaviour. In addi-
tion, although we accept that metastasis is the
gold standard for the diagnosis of malign-
nancy, histopathologists readily accept basal cell carcinoma as a malignant epithelial neo-

plasm 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
features such as lenticious junctional growth pattern, lymphocytic 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 lenticious 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 potential for reproduction of reproduc-
able 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 heteroge-
nous group in which the biological behav-
ior is 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 behav-
ior 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 Ber-
trand 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.”.

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

W J MOOI
Department of Histopathology, The Netherlands Cancer Institute, Plesmanlaan 121, 1066 GX Amsterdam, The Netherlands

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

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Clear cell carcinoma of the ovary

arising in a mucinous cystadenoma

I read with interest the recent case report by
Dr Dutt and Berney “Clear cell carcinoma of
the ovary arising in a mucinous cystadenoma.

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tures and histological di-


5 Nilbert M, Pajovic T, Mandahl N, et al. Mono-


6 Ballouk F, Ross J S, Wolf BC. Ovarian endomet-


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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 pockmark 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).

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 endometrioty cysts can show a lack of endometrioid stroma or that they can be “fibrous”. If a multicystic endometriotic cyst has global stroma and a complete lack of endometrioid 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, Tata Memorial Hospital, Parel, Bombay (Mumbai) 400011, India
A M BORGES
C S SOMAN
Department of Pathology, Manipal Hospital, Airport Road, Bangalore 560 017, India

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 They authors have indeed beautifully demonstrated the squamous histogenesis in their tumour. 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.1 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.

We object 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, Tata Memorial Hospital, Parel, Bombay (Mumbai) 400011, India
A M BORGES
C S SOMAN
Department of Pathology, Manipal Hospital, Airport Road, Bangalore 560 017, India

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


In the text the MSH2 and MLH1 genes were sometimes mistakenly written as MSH1 and MLH2, respectively. The authors apologise for this error.