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 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—describing small cell melanoma as a variant of naevoid melanoma. Clinico-pathological features and histological differential diagnosis. J Clin Pathol 2000;53:591–5.

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


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, 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 0.8 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 the 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 lentigious 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 possibility of reproduction 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.” If “small cell melanoma” is the next Spitz naevus that’s OK by us.

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

Clear cell carcinoma of the ovary leading to a mucinous cystadenoma


I feel that 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 the 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 lentigious 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 possibility of reproduction 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.” If “small cell melanoma” is the next Spitz naevus that’s OK by us.

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

Clear cell carcinoma of the ovary arising in a mucinous cystadenoma


We read with interest the case report by Mathers and O'Donnell on squamous carcinoma: an earlier report! 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 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).

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. The 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. 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.

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

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

References


The authors reply

We thank Pai et al for their interest in our paper “Squamous carcinoma of the skin with a rhabdoid phenotype.” We apologise for our omission of their previous paper 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

Correction


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