To the editor: Is this cutaneous angiomyolipoma truly an angiomyolipoma?

Sir,

We have read the article by Ammanagi *et al*.[1] with interest; however, we feel that some points need clarification. Firstly, the authors referred to angiomyolipoma (AML) as "hamartomatous" lesion; although this was the traditional view, it is now well accepted that these lesions are clonal and neoplastic; the same is true for other tumor-like lesions of tuberous sclerosis.[2,3] In fact, it is believed that the structural elements of AML, although recapitulating adipose tissue, vessels, and smooth muscle have a different and common origin in the putative epithelioid perivascular cell (PEC). The mere coexistence of adipocytes with vessels and smooth muscle is thus not sufficient to make diagnosis of AML. Second, we cannot agree that renal AML constitute separated entity from extrarenal AML. The lesions seen in the liver, retroperitoneum, lymph nodes, lung, or other locations share the morphology as well as lineage characteristics of renal AMLs.

More importantly, the photographs included in the paper are not truly compatible with the diagnosis given. As the authors them self state, the age of the patient and location of the lesion is unusual for an AML. In such a case, morphologic features and the immunophenotype of the tumor should be at least compatible with the diagnosis; however Figure 3 in the article by Ammanagi *et al*. shows large vascular-like spaces, connective (fibrous?) tissue, and adipocytes. No immunohistochemistry was used, hence a question may arise if the vascular spaces are truly vascular; differential diagnosis with a kind of urachal remnant is essential. In our opinion, the histologic picture as well the description is only distantly reminiscent of an AML. The diagnosis has to be confirmed by demonstration of PEC lineage (e.g., coexpression of smooth muscle and melanocytic markers, S-100 negativity in adipocytes, CD1a positivity or electron microscopy). We cannot plausibly declare that the lesion presented is not an AML; however, the authors didn't prove their diagnosis. If we could suggest the next step in investigation, if the AML diagnosis is unequivocally established, the analysis of TSC1 and TSC2 genes would be of extreme interest; in fact, as far as we know, there are no data about their status in AML located in the skin.

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