Mast cell density, microvessel density and their role in renal cell carcinoma
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Madam, I read the article by Guldur et al. entitled “The relationship of mast cells and angiogenesis with prognosis in renal cell carcinoma (J Pak Med Assoc. 2014; 64(3): 300-3)” with great interest. However, we have worries about the role of the Mast cell (MC) in neoplastic angiogenesis, metastasis and consequently progression of renal cell carcinoma (RCC). Additionally, human MC cytotoxicity against different tumour and vascular endothelial cells was recently demonstrated by us and others.1

Guldur et al. reported no significant relation between MC density (MCD) and microvessel density (MVD) in RCC.1 Then, increased MCD in tumoural tissue was interpreted as ‘MCs might have an effect on angiogenesis’.1 Although there have been several studies done in RCC,2 their results are confusing as well, similar to Guldur et al.1

In this study,1 detecting increased MCD in RCC suggested that MCs might play a role in tumour-associated angiogenesis, metastasis and acceleration of tumour growth. Nonetheless, in addition to the capability of MCs to kill tumour and endothelial cells, there was no significant correlation between MCD and neoangiogenesis in some mice studies and human cancers. Besides MCs, other inflammatory cells, e.g. tumour-associated macrophages, are blamed in tumour-associated angiogenesis. A significant correlation between prognosis, tumour-associated macrophages, and MVD was shown in several reports of RCC.2 In neoangiogenesis, individual characteristics of tumours are important, e.g. caveolin-1, hypoxia-inducible factor-1α, and matrix metalloproteinase expressions by RCC cells.3

It is tough to understand these contradictory results in this study with the literature. These conflicting findings may be lack of standardized scheme available to reliably assess the neoangiogenesis plus a striking heterogeneity in the endothelial cell marker expression among different tumours. Some researchers used colour image analysis, Doppler, and dynamic assessment by intravital microscopy to evaluate angiogenesis in RCC. Anti-CD105 demonstrated to be superior to anti-CD31 as a pan-endothelial marker to evaluate the role of neoangiogenesis in various carcinomas.4 Also, utilizing nonspecific endothelial cell markers may not reflect the angiogenic status for some tumours vascularizing without significant angiogenesis.5

Guldur et al. found a significant relation between MCD and the tumour stage, grade, diameter and metastasis.1 Even though these findings are very important for evaluating RCC, the authors did not comment. These findings make us think that MCD seems to be associated with tumour progression, metastasis and prognosis. However, no association has been mostly reported between the MCD, stage, grade, tumour diameter, or survival in RCC. Some studies also suggested MC cytotoxicity occurs in RCC through tumour-specific IgE. In conclusion, observing increased MCD/MVD in only tumour stroma with bad/good prognosis on histopathological specimens seems to be insufficient to explain their real role in that tumour tissue.

References