Visceral pleura invasion and lung cancer: further clarifications

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Kang et al. [1] reported that visceral pleura invasion (VPI) was a factor of poor prognosis in T2 non-small cell lung cancer (NSCLC). In a previous study [2], we already stressed the poor prognosis of VPI which we also found correlated with more extensive mediastinal lymph node (LN) involvement and decreased survival rates. Contrary to these authors, we observed VPI to be more frequent in case of non-squamous carcinoma (and of adenosquamous carcinoma which others also underline [3]) and in tumours larger than 5 cm. Concerning this latter point there is an unclear area in their paper. In the ‘patient and method’ section, they took into account T2 lesions and in the results they provide lesions measuring more or less than 3 cm. In case of T2 lesions, VPI is present in 100% of tumours 3 cm or less, the tumours without VPI being T1. Perhaps the authors meant 5 cm and this was mistyped: in such case they must add the T1N0 subgroup to the T2 without VPI. The frequency of VPI will probably appear higher for tumours more than 5 cm.

Although not observing an increased rate of VPI in the non-squamous NSCLC patients, they postulated that the most probable reason for this was that most lesions were adenocarcinomas and hence located more peripherally. We suggest the same explanation, also available for adenosquamous carcinomas, for lesions larger than 5 cm, the size of which evolves towards the periphery increasing the likelihood of VPI.

Similar to us [2], they suggest as explanation for poor prognosis the rapidity with which NSCLC in a subpleural location invades the pleura and disseminates throughout the pleural cavity: pre-formed stomas that connect subpleural lymphatics with the pleural space could account for the lymphatic and then the systemic dissemination of the exfoliated tumour cells. Developing this explanation further, we also stressed [2,4] the analogy existing between the poor prognosis in case of VPI and that observed when tumour cells are identified in post-thoracotomy pleural lavage (PTPL).

In order to better understand these phenomena, we recently performed two other studies. We first demonstrated [4] that the presence of tumour cells within PTPL appeared correlated with VPI and particularly with the p2VPI subgroup (the one with tumour exposed on the pleural surface not having yet involved the parietal pleura). Secondly, we demonstrated in an anatomical study [5] that the lymphatic drainage of the medial portion of the diaphragmatic pleura travelled through the peritracheobronchial LN chains, so explaining the greater frequency of mediastinal LN involvement observed in case of VPI.

We agree with Hang et al. to consider VPI as a potential indication for adjuvant chemotherapy. The role that VPI and parietal pleural cell reabsorption may play in the evolution of NSCLC is an important and interesting topic. Such pathologic features, which may aid in the selection of patients for adjuvant chemotherapy, require further knowledge and research.

References