David and coworkers [1] have recently reported a large series of non-small cell lung cancer (NSCLC) with visceral pleural invasion (VPI). They concluded that VPI was not associated with survival for tumors smaller than 5 cm, but showed negative effects on disease-free survival for tumors larger than 5 cm. This conclusion prompted us to raise an interesting question: does the varied effect of VPI on survival with different tumor sizes associate with VPI classification?

Patients with VPI can be further classified into two groups, PL1 and PL2, representing patients without and with visceral pleural surface invasion (VPSI). VPSI as an independent factor for poor prognosis and recurrence in NSCLC has been reported [2, 3]. Large tumor size was related to more frequent occurrence of VPSI in large tumors.

The poor prognostic effect of VPSI may be explained by the rapidity with which lung cancer cells in a subpleural location invade the pleura and disseminate throughout the pleural cavity. Once these cells exfoliate into the pleural cavity, preformed stomas that connect subpleural lymphatics with the pleural space could account for the systemic dissemination.

Concerning the standard definitions and evaluations of VPSI we previously reported [2], we would like to share with readers more detailed descriptions for processing the specimens with VPSI. Despite careful inspection and sampling of the retracted pleural surface with all for section of the retracted areas, serial microsections for definitive diagnosis are recommended to avoid the probably missing microscopic foci of VPSI. Immunohistochemical studies of thyroid transcription factor-1 protein and calretinin instead of elastic stain are necessary for differential diagnosis in those questionable cases associated with pleural hemorrhage and scanty floating tumor cells beyond the disrupted mesothelium. The accuracy of pathologic diagnosis should be emphasized because it may influence the patient’s postoperative treatment and outcome.

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References

Could Difference of Smoking Rate Between Men and Women Affect the Risk Difference of Acute Kidney Injury According to Sex?

To the Editor:

We have read the article by Mehta and colleagues [1] that showed women had a lower risk of acute kidney injury (AKI) than men at lower nadir hematocrit on cardiopulmonary bypass. In this study, the researchers considered the relationship between several preoperative risk factors like diabetes mellitus and AKI in men and women separately. How about smoking?

Smoking might increase risk of kidney failure, especially in men [2]. Although the pathophysiology of the association between smoking and kidney failure has not been fully revealed, smoking affects endothelial cell function, leading to vasoconperfusionstriction and vascular damage [3]. Reactive oxygen/nitrogen species generated from smoking might alter erythrocyte membrane physicochemical properties, changing the tissue [4]. The formation of carboxyhemoglobin after smoking causes a left shift of the hemoglobin-oxygen dissociation curve [5], which cause changes of tissue perfusion. The smoking prevalence of men was higher than women in Italy [6]. In conclusion, we should ask whether the smoking rate was different between males and females. Smoking could affect kidney function and tissue perfusion, so it acts as a bias in a study that evaluates whether AKI risk is lower in women than in men.

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