We read with interest the recent report by Yildirim et al (issue 2, volume 56, 2008, Tuberk Toraks) regarding soft tissue sarcoma metastatic to pleural (1). We would like to share our experience. Our patient was synovial cell sarcoma of the leg metastatic to pleura. In the patient, the sarcoma recurred only in visceral and parietal pleura several times, and was surgically resected each time. However, he had no metastatic lesion other than pleura. In addition, he developed neither pleural effusion nor intrapulmonary metastasis. As the author described, it is generally accepted that pleural effusion do not develop when the pleural is involved by sarcomas because of the characteristic absence of lymphatic metastasis (2). In the case reported by the authors, however, massive pleural effusion was observed (1). We would appreciate hearing from the authors why this case developed pleural effusion. The authors described that malignant cells were cytologically identified in pleural fluid. Please explain or speculate the mechanism or speculation. According to the authors, the dominant pattern of metastases is hematogenous in most patients with usual soft tissue sarcoma (1). Did the present case have metastatic sites other than pleura? If not so, we would like to know why soft tissue sarcoma in some patients including this case metastasizes only in the pleural space.

REFERENCES
We greatly appreciate the comments made by Hiroaki Satoh and Kiyohisa Sekizawa on our paper. We shall try to respond to and clarify their doubts to the best of our ability. First, they questioned why this case developed pleural effusion. Most malignant tumours can produce pleural metastases, which are usually late events in the course of malignancy. As we know, an important feature of the parietal pleura is lymphatic stomata, i.e. openings between parietal pleural mesothelial cells (1). Pleural fluid is drained out of the pleural space predominantly through the stomata of the parietal lymphatics lying between the parietal mesothelial cells (2). It has been suggested that reduced pleural fluid outflow, secondary to tumour blockage of parietal stomas and the subsequent drainage paths, or lymphatic obstruction due to metastatic mediastinal lymphadenopathy, also contributes to fluid accumulation (3). The most likely explanation to us seems that a possible mechanism of pleural fluid for this case is blockage of these parietal stomas by tumour cells.

Secondly, there were no metastatic sites other than pleura. Nevertheless we could not evaluate lung parenchyma because of the massive pleural effusions. We do not explain why soft tissue sarcoma in some patients metastasizes only in the pleural space. Each type of cancer has a specific pattern of metastatic distributions. The mechanism of unusual distant metastasis for soft tissue sarcoma remains obscure. As discussed in the text, the dominant pattern of metastases for soft tissue sarcoma is hematogenous. We speculated that a possible mechanism of metastases for this patient may be tumour embolism. For this reason, soft tissue sarcoma may produce metastases only in the pleural space. The lungs, as generally accepted, are a common site of metastases for soft tissue sarcoma. In addition to this, it has been shown that only about 55-60% of patients with proven pleural metastases develop pleural effusions (4). However, in the original series by Songür et al., 2 of 400 patients with primary extremity sarcoma were found to have isolated pleural effusions (5).

We hope that further papers will be illustrative for this patient population.

We thank Hiroaki Satoh and Kiyohisa Sekizawa for their remarks and suggestions.

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