

Treatment of tenosynovial giant-cell tumour types

We read with great interest the international, retrospective, cohort study by Monique Mastboom and colleagues¹ reporting surgical outcomes of diffuse-type tenosynovial giant-cell tumours, and comments by William Tap on the importance of multidisciplinary care for this disease.² Mastboom and colleagues' study analysed surgical outcomes, with particular emphasis on postoperative recurrence and complications. Tap mentioned the usefulness of colony-stimulating factor 1 receptor inhibitors, a molecular targeted therapy, for patients with tenosynovial giant-cell tumours, and the importance of the indications by which medical oncologists select them.

First, because tenosynovial giant-cell tumours are benign neoplasms, recurrence itself might not be so problematic for patients because they do not concern life prognosis, but the functional impairment or complications from the disease or surgical or medical treatments should be taken into account for evaluation of treatment outcomes. Mastboom and colleagues' study reported the symptoms of 578 patients who had complete data before initial treatment and at final follow-up, and described that, although the proportion of patients with reported joint stiffness decreased after surgery from 14% to 8%, 10% of patients not reporting stiffness before surgery complained of it thereafter. Therefore, the total proportion of patients reporting stiffness increased from 14% to 18% postoperatively, suggesting that stiffness is attributable to the surgery itself in some cases.

Second, occasionally, patients with tenosynovial giant-cell tumours have indolent disease progression, with slow tumour growth or rare occurrence of osteochondral destruction of the involved joints. The biological features of tenosynovial giant-cell tumours

need to be considered when selecting the treatment modality. For instance, frequency of bone destruction in patients with tenosynovial giant-cell tumours might depend on the volume of the involved joints,³ which suggests the bone destruction in tenosynovial giant cell tumours might not be like that in rheumatoid arthritis (chemical), but mechanical in part. Thus, an active surveillance plan could be worthwhile. Only 76 (7%) of patients under active surveillance were included in Mastboom and colleagues' report and were excluded from the analyses during follow-up. Surgical intervention and its method should be carefully decided based on the predicted complications of the surgery and indolent biological features of tenosynovial giant cell tumours. In some cases, active surveillance might be a suitable choice.

In cases in which postoperative function and complications are predicted to be a concern and active surveillance could not be applicable due to the worsened symptoms and joint destruction (eg, hip),^{4,5} molecular targeted therapy could be a treatment choice.

Future studies on the role of active surveillance should be done by multiple institutions to clarify the significance of this treatment modality, and to maintain patients' function and quality of life.

We declare no competing interests.

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