学 位 論 文 の 要 旨

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学　位　論　文　名	Brain Activity Changes in Somatosensory and Emotion-related Areas With Medial Patellofemoral Ligament Deficiency
発　表　雑　誌　名 （巻，初頁～終頁，年）	Clinical Orthopaedics and Related Research (in press)
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論 文 内 容 の 要 旨

INTRODUCTION

Background: Patellar instability with medial patellofemoral ligament (MPFL) deficiency is a common sports injury among young people. Although nonoperative and surgical treatment can provide stability of the patella, patients often have anxiety related to the knee. We speculate that neural dysfunction may be related to anxiety in these patients; however, the mechanism in the brain that generates this anxiety remains unknown.

The aim of the current study was to assess brain activity in patients with a chronic MPFL deficiency compared with control subjects who do not have a MPFL deficiency or any other knee disorders. We asked: (1) How does brain activity in patients with MPFL deficiency change in the areas related to somatic sensation against lateral shift of the patella? (2) How does patella instability, which can lead to continuous fear or apprehension for dislocation, influence brain activity in the areas related to emotion?

MATERIALS AND METHODS

Nineteen patients with MPFL deficiency underwent surgical reconstruction in our hospital

from April 2012 to March 2014. Excluding seven patients with osteochondral lesions, 12 patients (five males and seven females; mean age, 20 years) with MPFL deficiency were sequentially included in this study. Eleven control subjects (four males and seven females; mean age, 23 years) were recruited from medical students who had no history of knee injury. Diagnosis of the MPFL deficiency was made with MR images, which confirmed the rupture, and by proving the instability with a custom-made biomechanical device. Brain activity during passive lateral stress to the patella was assessed by functional MRI. Functional and anatomic images were analyzed using statistical parametric mapping. Differences in functional MRI outcome measures from the detected activated brain regions between the patients with MPFL deficiency and controls were assessed using t tests.

The study protocol was approved by the Ethics Committee of Shimane University and written informed consent was obtained from all subjects.

RESULTS AND DISCUSSION

Intergroup analysis showed less activity in several sensorimotor cortical areas, including the contralateral primary somatosensory areas(% signal change for MPFL group, 0.49%, versus 1.1% for the control group; $p<0.001$), thalamus (0.2% versus 0.41% for the MPFL versus control, respectively; $p<0.001$), and ipsilateral thalamus (0.02% versus 0.27% for the MPFL versus control, respectively; $p<0.001$), and ipsilateral Cerebellum (0.82% versus 1.25% for the MPFL versus control, respectively; $p<0.001$)in the MPFL deficiency group than in the control group. In contrast, The MPFL deficiency group showed more activity in several areas, including the contralateral primary motor area (1.06% versus 0.6% for the MPFL versus control, respectively; $p<0.001$) , supplementary motor area (0.89% versus 0.49% for the MPFL versus control, respectively; $p<0.001$), prefrontal cortex (1.09% versus 0.52% for the MPFL versus control, respectively; $p<0.001$) , inferior parietal lobule (0.89% versus 0.62% for the MPFL versus control, respectively; $p<0.001$), anterior cingulate cortex (0.84% versus 0.08% for the MPFL versus control, respectively; $p<0.001$), visual cortex (0.86% versus 0.14% for the MPFL versus control, respectively; $p<0.001$), vermis (1.18% versus 0.37% for the MPFL versus control, respectively; $p<0.001$), and ipsilateral prefrontal cortex (1.1% versus 0.75% for the MPFL versus control, respectively; $p<0.001$) than did the control group.

Similar to those with ACL deficiency, patients with MPFL deficiency showed diminished activation in the primary sensory area and thalamus, both of which play leading roles in input from proprioception in normal knees. The decreased activation of these cortical areas in the MPFL group could be attributed to the differentiation of the ascending afferent pathway from the

injured knee. We propose some theories regarding the possible response to this incitement. One is that the MPFL has a function as a mechanoreceptor, which could detect a lateral shift of the patella when the MPFL was elongated or shortened. Another theory is that the Golgi tendon end organ or muscle spindle senses the elongation or shortening of the patella tendon and/or quadriceps femoris muscle and/or joint capsule. We surmise that the reasons for altered brain activity in patients with MPFL deficiency is that the sensory function of tendons, joint capsule, and muscles may be affected by patella instability. Diminished activity of the ipsilateral cerebellum indicates a decreased ascending afferent pathway through the spinocerebellar tract, which is one of the somatosensory pathways. This finding also could show function of proprioception in the MPFL-injured knee. In addition, increased activity in the primary motor area, supplementary motor area, anterior cingulate cortex, and cerebellum, as seen in our patients, may not indicate a normal response to a lateral shift of the patella. We were concerned that apprehension regarding dislocation could be imprinted as a fear memory resulting from recurrent dislocation or a long posttraumatic interval in patients with MPFL deficiency. If the apprehension can become chronic, like pain, it may lead to persistent feelings of anxiety or instability, even with a stabilized knee after MPFL reconstruction.

This study suggests that specific brain-area activity is increased in patients with MPFL deficiency relative to that in controls. Further longitudinal research to assess brain activity and proprioception between patients pre- and post reconstructive knee surgery may reveal more regarding how patella instability is related to brain function. We hope that based on such research, a neural approach to improve patella-instability-related brain function can be developed.

CONCLUSION

Less activity in the contralateral somatosensory cortical areas suggested that MPFL deficiency may lead to diminished somatic sensation against lateral shift of the patella. In contrast, increased activity in the anterior cingulate cortex, prefrontal cortex, and inferior parietal lobule may indicate anxiety or fear resulting from patellar instability, which is recognized as an aversion similar to that toward chronic pain.