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Abbreviations:
ACL = anterior cruciate ligament
ETL = echo train length
FOV = field of view
SE = spin echo

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Increased Subcortical Patellar Signal Intensity at T2-weighted MR Imaging: A Subacute Finding after Knee Injury[1]

PURPOSE: To investigate the temporal prevalence of a rim of increased signal intensity in the subcortical part of the patella at T2-weighted magnetic resonance (MR) imaging after arthroscopic knee surgery or knee injury.

MATERIALS AND METHODS: The prospective and retrospective components of the study were performed after receiving approval from the institutional research ethics board. Written informed consent was obtained for the prospective component of the study. The need for informed consent was waived for the retrospective component of the study. The authors performed a prospective evaluation of serial MR images of the knee of four patients (three men and one woman; age range, 28–53 years; mean age, 36.8 years) after arthroscopic anterior cruciate ligament (ACL) reconstruction. They also performed a retrospective review of MR images of the knee from 90 consecutive patients (59 male and 31 female patients; age range, 13–66 years; mean age, 34.6 years) referred for the evaluation of an ACL injury. All imaging studies were evaluated in consensus by two musculoskeletal radiologists for the presence of a rim of increased signal intensity in the subcortical part of the patella at T2-weighted imaging. The presence of this signal intensity pattern was correlated with the time to imaging from surgery or joint injury. Logistic regression modeling was performed with increased subcortical signal intensity as the outcome and a second-degree polynomial for elapsed time as the predictor.

RESULTS: All four patients who underwent imaging prospectively after ACL surgery showed increased T2-weighted signal intensity in the subcortical part of the patella 12 weeks after surgery. None of the patients showed these signal intensity changes at 6 or 24 weeks. A subcortical rim of increased T2-weighted signal intensity was seen in the patella in 24 (27%) of the 90 consecutive patients who underwent imaging for the assessment of possible ACL injury. Significant independent differences were seen in the prevalence of increased T2-weighted signal intensity in the subcortical part of the patella when related to time between injury and MR imaging ($P = .002$), with an increase in prevalence as time since injury increased and a decrease in prevalence in patients who underwent MR imaging more than 20 weeks after injury ($P = .013$).

CONCLUSION: Increased T2-weighted signal intensity in the subcortical part of the patella appears to be a subacute transient MR finding seen after knee surgery or injury.
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Various causes of increased intraosseous signal intensity at T2-weighted magnetic resonance (MR) imaging are well recognized at imaging of the knee. The cause of such signal intensity changes include bone bruising, fracture, osteonecrosis, osteoarthritis, and reactive edema secondary to infection or neoplastic disease (1,2). Although the underlying conditions associated with increased intraosseous T2-weighted signal intensity change are well recognized in many instances, the pathophysiologic mechanism(s) responsible for the increased signal intensity itself may be less clearly understood (2). We have observed the

presence of a thin curvilinear peripheral rim of increased signal intensity in the subcortical part of the patella at T2-weighted MR imaging in several patients referred for assessment of possible joint derangement after knee trauma and in patients who have undergone imaging after knee joint surgery. Our hypothesis was that the finding of increased subcortical signal intensity was a transient finding, seen subacutely after knee injury, and that the probability of identifying this finding would first increase and then decrease as the interval between knee injury and MR imaging increased. Thus, the purpose of our study was to investigate the temporal prevalence of the MR imaging finding of a rim of peripheral increased signal intensity in the subcortical part of the patella at T2-weighted MR imaging after arthroscopic knee surgery or knee injury.

MATERIALS AND METHODS

Both the prospective and retrospective components of our study were performed after obtaining approval from the institutional research ethics board. Written informed consent was obtained for the prospective component. The need for informed consent was waived by the ethics board for the retrospective component of the study. All MR examinations were performed with a 1.5-T MR unit (Signa; GE Medical Systems, Milwaukee, Wis) and dedicated quadrature extremity coils.

Prospective Study: Patients and Imaging

From January 2003 to December 2003, four consecutive patients were prospectively examined after undergoing arthroscopic hamstring–anterior cruciate ligament (ACL) graft reconstruction (three men, one woman; age range, 28–53 years; mean age, 36.8 years). Consecutive patients undergoing ACL graft surgery were enrolled in this study. Patients with preoperative clinical and MR imaging documentation of complete ACL rupture underwent surgery 3–56 weeks after the initial knee injury. No other selection criteria were used. Preoperative MR imaging in these patients was performed 2–40 weeks (mean, 26 weeks) before surgery and consisted of the following imaging sequences: sagittal intermediate-weighted fast spin-echo (SE) imaging (repetition time msec/echo time msec of 4000/32, echo train length [ETL] of eight, 31.25-kHz bandwidth, 4.0-mm-thick sections with no gap, 14-cm field of view [FOV], and 512 × 256 matrix), T2-weighted fast SE imaging with fat saturation (3500/68.4, ETL of eight, 20.83-kHz bandwidth, 4.0-mm-thick sections with no gap, 14-cm FOV, and 256 × 224 matrix), coronal intermediate-weighted fast SE imaging (3500/37, ETL of eight, 31.25-kHz bandwidth, 4.0-mm-thick sections with no gap, 14-cm FOV, and 512 × 256 matrix), and transverse T2-weighted fast SE imaging with fat saturation (3500/68.4, ETL of eight, 20.83-kHz bandwidth, 4.0-mm-thick sections with a 1.0-mm gap, 14-cm FOV, and 256 × 192 matrix). In all patients, surgery was performed by one orthopedic surgeon (E.B.) with 12 years of clinical experience and subspecialty training. Surgery was performed by using a similar technique, with use of tourniquets and limb exsanguination for control of intraoperative bleeding. All patients were treated postoperatively, with gradual progressive weight bearing—as tolerated—on the treated limb. Three patients underwent MR imaging at 6, 12, and 24 weeks after surgery; one patient underwent MR imaging at 12 and 24 weeks after surgery. This patient was not successfully imaged at 6 weeks after surgery due to initial hospital restrictions in elective outpatient imaging at the time of the regional severe acute respiratory syndrome outbreak in Toronto, Canada.

MR imaging sequences performed in this patient group included sagittal intermediate-weighted fast SE imaging (4000/32, ETL of eight, 31.25-kHz bandwidth, 4.0-mm-thick sections with no gap, 14-cm FOV, and 512 × 256 matrix), T2-weighted fast SE imaging with fat saturation (3500/68.4, ETL of eight, 31.25-kHz bandwidth, 4.0-mm-thick sections with no gap, 14-cm FOV, and 256 × 224 matrix), coronal intermediate-weighted fast SE imaging (3500/37, ETL of eight, 31.25-kHz bandwidth, 4.0-mm-thick sections with no gap, 14-cm FOV, and 512 × 256 matrix), transverse T2-weighted fast SE imaging with fat saturation (3500/68.4, ETL of eight, 20.83-kHz bandwidth, 4.0-mm-thick sections with a 1.0-mm gap, 14-cm FOV, and 256 × 192 matrix), and transverse and sagittal T1-weighted fast SE imaging with fat saturation (716/11, ETL of four, 31.25-kHz bandwidth, 4.0-mm-thick sections with no gap, and 256 × 192 matrix) after the intravenous administration of 1 mmol/kg gadodiamide (Omniscan; Amersham Health, Buckinghamshire, England). A superior-inferior phase-encoding direction was used for the sagittal images, and a right-left phase-encoding direction was used for transverse and coronal images.

Retrospective Study: Patients and Imaging

We retrospectively reviewed MR images of the knee obtained between January 2002 and July 2003 in 90 consecutive patients without a history of prior knee surgery. The patients had been referred by one orthopedic surgeon (E.B.) for the evaluation of possible ACL injury. There were 59 male (66%) and 31 female (34%) patients aged 13–66 years (mean age, 34.6 years). All patients were examined on an elective ambulatory outpatient basis at one MR imaging facility.

In all patients, MR imaging sequences included sagittal intermediate-weighted fast SE imaging (4000/32, ETL of eight, 31.25-kHz bandwidth, 4.0-mm-thick sections with no gap, 14-cm FOV, and 512 × 256 matrix), sagittal T2-weighted fast SE imaging with fat saturation (3500/68.4, ETL of eight, 20.83-kHz bandwidth, 4.0-mm-thick sections with no gap, 14-cm FOV, and 256 × 224 matrix), coronal intermediate-weighted fast SE imaging (3500/37, ETL of eight, 31.25-kHz bandwidth, 4.0-mm-thick sections with no gap, 14-cm FOV, and 512 × 256 matrix), and transverse T2-weighted fast SE imaging with fat saturation (3500/68.4, ETL of eight, 20.83-kHz bandwidth, 4.0-mm-thick sections with a 1.0-mm gap, 14-cm FOV, and 256 × 192 matrix). In all patients, a retrospective chart review was performed by one of the authors (T.I.P.) to determine the interval between the initial knee injury and MR imaging. The interval between injury and imaging was categorized as acute if it was 0–4 weeks, subacute if it was 5–20 weeks, and chronic if it was more than 20 weeks. One MR imaging study from each of these 90 patients was used for the data analysis.

Image Evaluation

All images were reviewed by two musculoskeletal radiologists (L.M.W., T.I.P.) with 10 and 2 years of subspecialty training and experience, respectively; agreement was reached by consensus. Readers were aware of the clinical indication for MR imaging but were blinded to patient identification and other clinical information, including interval between surgery or injury and MR imaging. All imaging studies were assessed for the presence or absence of a rim of increased signal intensity paralleling the subcortical margins (articular and nonarticular) of the patella on transverse and/or sagittal T2-weighted MR images. The signal inten-

TABLE 1
Prospective Study: Presence and Absence of Increased T2-weighted Signal Intensity in the Subcortical Part of the Patella after Surgery

Patient No.	Time after Surgery (wk)		
	6	12	24
1	Absent	Present	Absent
2	Absent	Present	Absent
3	Absent	Present	Absent
4	NA	Present	Absent

Note.—NA = not applicable.

sity was compared with that of normal fatty marrow in the patella, distal femur, and proximal tibia. Comparison was also made with the adjacent subcutaneous fat to ensure that the findings were not related to artifact secondary to heterogeneous fat suppression. To avoid potential false-positive findings due to volume averaging with adjacent joint fluid, a continuous subcortical rim of increased T2-weighted signal intensity was required to be seen on two contiguous transverse or sagittal images to be classified as present. In addition, the enhancement features of the subcortical margins of the patella were assessed in the prospective study group on the contrast material–enhanced T1-weighted images obtained with fat saturation.

The imaging assessment of the retrospective imaging cohort referred for MR imaging assessment of possible ACL injury also involved the assessment of the presence or absence of MR imaging findings of ACL injury, posterior cruciate ligament injury, collateral ligament injury, meniscal tears, and joint effusion in all patients. A cruciate or collateral ligament injury was defined as MR imaging findings of focal or diffuse discontinuity of ligament fibers, abnormal contour of the ligament, or abnormal signal intensity of the ligament and surrounding region (3–7). Meniscal tears were defined as abnormalities in meniscal morphology (ie, fragmentation or truncation) and/or findings of increased intrameniscal signal intensity extending to one or more of the meniscal articular surfaces seen on at least two contiguous imaging sections with short-echo-time MR imaging (8–10). The presence or absence of a joint effusion was defined as more than 5-mm anterior-posterior distention of the suprapatellar recess measured in the midline of the joint or more than 10-mm anterior-posterior distention of the lateral joint recess measured on the last lateral sagittal image of the lateral recess of the joint (11).

Statistical Analysis

Statistical analysis of the findings from the retrospective image review cohort was performed to assess potential predictors of the finding of transient increased T2-weighted signal intensity within the subcortical part of the patella. To test this hypothesis, we fitted a logistic regression model with increased subcortical T2-weighted signal intensity as the outcome and a second-degree polynomial for elapsed time as the predictor. The second-degree polynomial was chosen a priori to allow for the probability of increased subcortical T2-weighted signal intensity to increase and then decrease with elapsed time since injury. The independent effect of patient age in predicting increased T2-weighted signal intensity was assessed by adding a term for age to this regression model. The Hosmer-Lemeshow goodness-of-fit test was used to check goodness of fit of our logistic regression models. The robustness of the second-degree polynomial for elapsed time in the logistic regression models was checked by comparing the results to models with cubic splines for elapsed time. For further exploratory analyses, we used the χ^2 test to assess the relationship of increased T2-weighted signal intensity with other demographic variables and MR imaging findings. A P value of less than .05 was taken to indicate statistically significant difference. All analyses were performed by using statistical software (S-Plus 6.2, 2003; Insightful, Seattle, Wash).

RESULTS

Prospective Patient Group

Results are summarized in Table 1. None of the four patients had increased T2-weighted signal intensity in the subcortical part of the patella on the preoperative MR imaging studies (obtained 2, 30, 36, and 42 weeks before surgery, respectively). None of the three patients who were prospectively examined after undergoing hamstring–ACL reconstruction (examined at 6, 12, and 24 weeks after surgery) showed evidence of increased T2-weighted signal intensity in the subcortical part of the patella at MR imaging performed 6 weeks after surgery. All three patients showed evidence of increased subcortical T2-weighted signal intensity at 12 weeks, and all showed disappearance of the subcortical increased signal intensity at 24 weeks (Fig 1). For the fourth patient examined prospectively and imaged at 12 and 24 weeks, MR imaging studies illustrated increased subcortical T2-weighted signal intensity within the patella at 12 weeks after surgery, with resolution of the signal intensity changes at 24 weeks. Findings of contrast enhancement after the intravenous administration of a gadolinium chelate in all four patients mirrored both the anatomic distribution and temporal presence or absence of increased signal intensity in the subcortical part of the patella on T2-weighted images.

Retrospective Patient Group

Results for the retrospective patient group are summarized in Table 2. At MR imaging, increased T2-weighted signal intensity in the subcortical part of the patella was identified in 24 (27%) of the 90 patients (Fig 2). In 14 patients (16%), the interval between injury and MR imaging was more than 365 days. Increased T2-weighted signal intensity in the subcortical patella was identified in two (14%) of these 14 patients. Results of logistic regression analysis showed a significant quadratic term ($P = .002$) for the time between injury and MR imaging. This association is illustrated in Figure 3, which shows the predicted prevalence of increased T2-weighted signal intensity in the subcortical part of the patella versus time from injury. It can be seen that the prevalence of the increased subcortical T2-weighted signal intensity *(a)* is low with imaging at shorter times following injury (acute), *(b)* increases as time between injury and imaging increases (subacute period), and *(c)* decreases in patients who undergo imaging at least 20 weeks after injury ($P = .013$).

After adjusting for time since injury in the logistic regression analysis, we found a moderate decrease in the odds of increased T2-weighted signal intensity in the subcortical patella with increasing age ($P = .04$), with the odds of visualizing the increased T2-weighted signal intensity decreasing by a factor of 0.65 per decade.

In evaluating the logistic regression analysis, we found no lack of fit with the Hosmer-Lemeshow test ($P > .3$), and the results obtained by using a cubic spline for elapsed time were qualitatively similar and not significantly different from those obtained with the second-degree polynomial.

The presence of knee joint effusion ($n = 45$, 50%) was not a statistically sig-

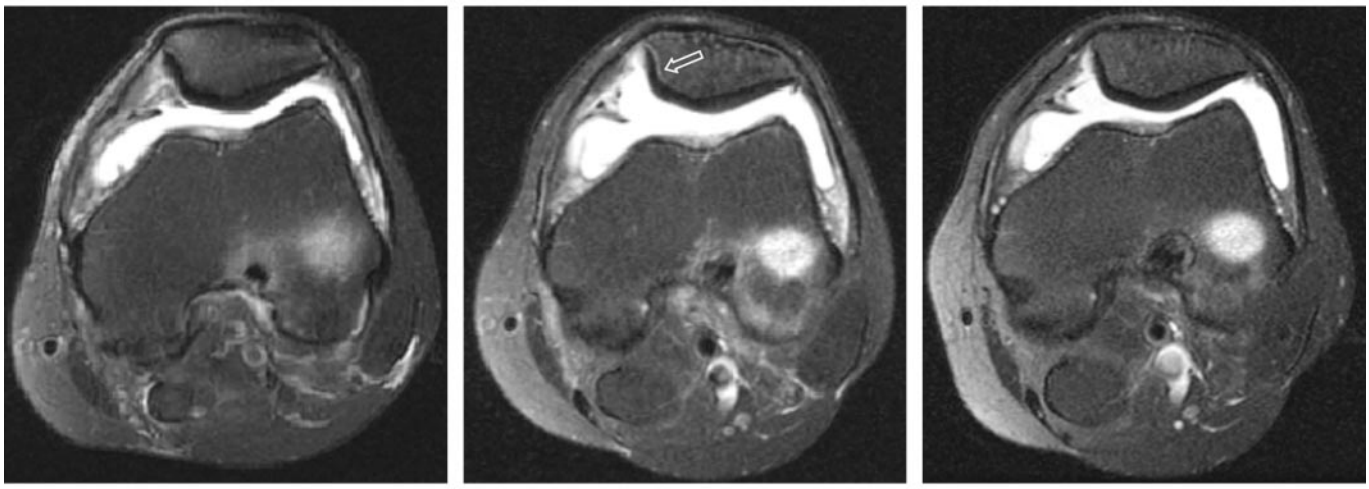

Figure 1. Transverse T2-weighted fast SE MR images obtained with fat saturation (3500/68.4) at **(a)** 6, **(b)** 12, and **(c)** 24 weeks after ACL graft reconstruction in a 31-year-old man. Increased peripheral T2-weighted signal intensity (arrow) in the subcortical part of the patella is seen only in **b**.

nificant predictor of the presence of increased peripheral T2-weighted signal intensity in the subcortical part of the patella ($P = .48$). Neither was the maximal amount of fluid identified in the midline sagittal image of the suprapatellar bursa ($P = .17$; fluid volume range, 0–16 mm; mean volume ± standard deviation, 5.26 mm ± 4.02).

The prevalence of increased peripheral T2-weighted signal intensity in the subcortical patella was not significantly related to the presence of MR imaging signs of injury to the ACL ($n = 59$, 66%) ($P = .80$), posterior cruciate ligament ($n = 10$, 11%) ($P > .999$), medial collateral ligament ($n = 19$, 21%) ($P > .999$), lateral collateral ligament ($n = 4$, 4%) ($P = .57$), or meniscus ($n = 48$, 53%) ($P = .80$). Patient sex ($P > .999$) and laterality of injury (right or left) ($P = .82$) were also not statistically significant predictors of visualizing subacute increased T2-weighted signal intensity in the subcortical part of the patella.

TABLE 2
Number of Patients with Increased Subcortical T2-weighted Signal Intensity

Parameter	Time between Injury and MR Imaging (wk)			Total
	0–4	5–20	20+	
No. of patients	25	48	17	90
No. of patients with increased subcortical signal intensity	2 (8)	19 (40)	3 (18)	24 (27)

Note.—Numbers in parentheses are percentages.

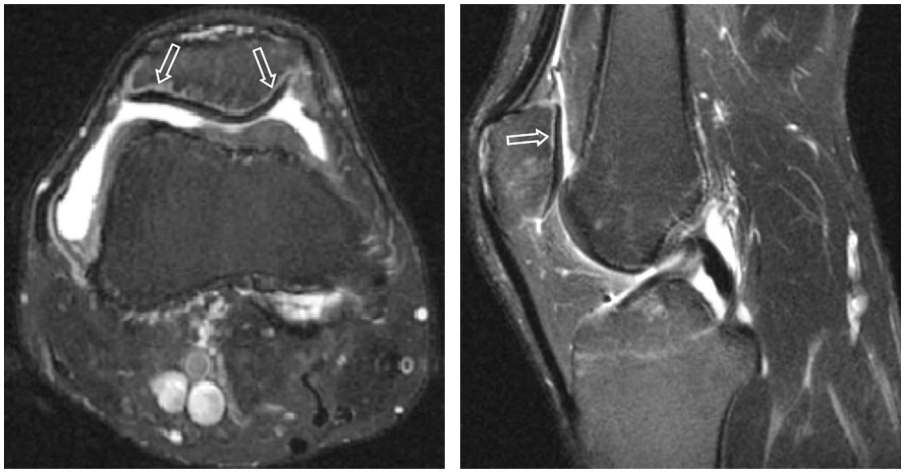

Figure 2. **(a)** Transverse and **(b)** sagittal T2-weighted fast SE MR images obtained with fat saturation (3500/68.4) in a 30-year-old man who was assessed for clinically suspected ACL injury. Increased peripheral signal intensity (arrows in **a**, arrow in **b**) is noted in the subcortical part of the patella.

DISCUSSION

Our results illustrate findings of transient development and subsequent resolution of a curvilinear area of increased T2-weighted signal intensity within the subcortical part of the patella in patients who underwent imaging within a 6-month period after arthroscopic ACL–hamstring graft reconstruction. Similar findings of increased T2-weighted signal intensity in the subcortical and subchondral parts of the patella were noted in 27% of patients who underwent imaging for the evaluation of clinically suspected ACL injury.

Although the finding appears to develop and regress in both patient groups, the time course of the finding after surgery (peaking at 12 weeks and resolving

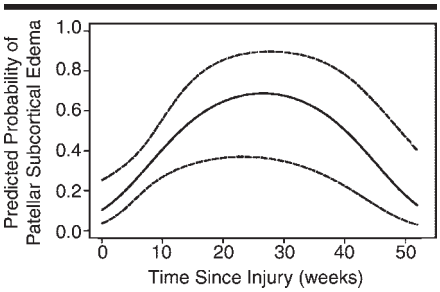

Figure 3. Graph shows the predicted probability of increased T2-weighted signal intensity in the subcortical part of the patella relative to time after injury in patients referred for MR imaging to assess possible ACL injury. Dotted lines represent the 95% confidence band.

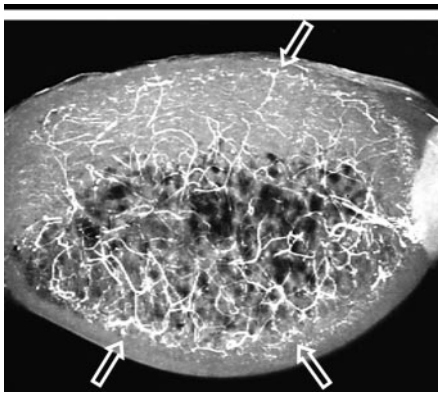

Figure 4. Microangiogram of normal canine patella (transverse section) depicts extensive intraosseous blood supply within the patella with numerous arcuate vessels (arrows) coursing through cancellous bone immediately beneath the articular cartilage and the ventral, medial, and lateral cortices. (Image courtesy of Paul Howard, DVM, MS, Vermont Veterinary Surgical Center, Colchester, Vt)

by 24 weeks) differs slightly from that found in patients with injury (peaking at approximately 27 weeks). This may reflect the longer period of disability endured and potential differences in ambulation and rehabilitation between the patients who had not undergone surgical treatment compared with those who had.

The pattern of increased T2-weighted signal intensity seen in our patients corresponds to the anatomic prominence of subcortical microvasculature within the patella. The microvascular blood supply of the patella has been previously illustrated with microangiography (12,13). Results of canine patellar studies have shown an extensive intraosseous blood supply within the patella with numerous arcuate vessels coursing through subcortical cancellous bone (Fig 4) (13). We speculate that the cause of this subcortical rim of increased T2-weighted signal intensity described in the current investigation may be directly or indirectly related to this microvascular network.

The increased prominence of microvascular architecture and possible capillary leakage may theoretically be accentuated in the setting of alterations in autonomic neurovascular regulation of blood supply with increased capillary flow or impairment of vascular outflow. The link between intraosseous vascular circulation and bone formation and resorption is well documented, with intraosseous hyperemia recognized as an essential physiologic component of corresponding deossification (14–16). Alterations in osseous vasoregulation and hyperemia have also specifically been recognized in the setting of acute bone resorption and disuse (17), which may vary in geographic pattern on the basis of the topographic and anatomic relationships of intraosseous vascular circulation (14).

Alterations in biomechanical load have also been proposed as a potential cause of altered intraosseous blood flow. Mechanical loading may serve a vital role in ensuring that cells within bone (particularly osteophytes) receive sufficient metabolic exchange (18). Investigators have hypothesized that changes in mechanical loading may deprive osteocytes of the metabolic supplies to which they have become accustomed, leading to a vasoregulatory physiologic response of hyperemia (17). It should be noted that such alterations in intraosseous vascularity may resolve with removal or cessation of the biomechanical stress or alteration implicated in their initial pathogenesis. Our study observations of transient temporal changes of T2-weighted signal intensity in the subcortical part of the patella after ACL reconstruction and knee injury may be suggestive of altered biomechanics and/or associated vasoregulatory changes in intraosseous subcortical patellar blood flow after knee surgery or knee injury as potential causes for the increased T2-weighted signal intensity changes described.

Microvascular accentuation within the subcortical aspects of the patella may alternatively arise as a result of impairment of vascular outflow, possibly related to venous congestion or thrombosis. Passive intraosseous venous congestion of microvascular patellar blood flow could theoretically occur in the setting of increased intraarticular pressure as a result of an articular joint effusion (19,20). Our data, however, did not show a significant relationship between the presence of a joint effusion and visualization of increased T2-weighted subcortical signal intensity changes.

Limitations of the current study include the fact that temporal follow-up studies were not available in the patient group referred for the assessment of ACL injury. Thus, the sequential evolution of the MR imaging changes described in the subgroup of patients with increased subcortical T2-weighted signal intensity and the possible time to resolution in this retrospective study group could not be directly assessed in individual patients. Other limitations include the fact that only patients suspected of having an ACL injury were specifically examined and that the specific mechanism of injury, the level of resultant dysfunction, ambulation, weight-bearing, and type or duration of possible rehabilitation in the retrospective study patient population were unknown. Assessment of this clinical information may have been valuable because these factors may have been important contributory features in the development of the subcortical T2-weighted signal intensity changes described. In addition, study of the retrospective review cohort relied on the patients' clinical charts for information regarding the timing of injury. It is possible that secondary injury may not have been recorded. This may explain why two of 14 patients with an injury-to–MR imaging interval of more than 365 days were observed to have increased T2-weighted signal intensity in the subcortical part of the patella. Another limitation of our study was the small number of patients examined prospectively after ACL reconstruction. Prospective evaluation of a larger series of patients would provide greater statistical confidence as to the frequency of subcortical patellar increased T2-weighted signal intensity observed in patients after ACL reconstructive surgery.

In conclusion, the finding of a rim of increased T2-weighted signal intensity in the subcortical part of the patella appears to be a transient MR finding observed in the subacute period after knee surgery or injury. We identified a correlation between the temporal and spatial distribution of this finding and acute trauma or surgery to the knee. To our knowledge, this observation has not been previously reported. At present, we can only speculate as to the cause of the finding, which may be physiologically related to alterations in subcortical vascularity in the patient groups studied.

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References

1. Eustace S, Keogh C, Blake M, Ward RJ, Oder PD, Dimasi M. MR imaging of bone oedema: mechanisms and interpretation. Clin Radiol 2001;56:4–12.
2. Zanetti M, Bruder E, Romero J, Hodler J. Bone marrow edema pattern in osteoarthritic knees: correlation between MR imaging and histologic findings. Radiology 2000;215:835–840.
3. Mink JH, Levy T, Crues JV III. Tears of the anterior cruciate ligament and menisci of the knee: MR imaging evaluation. Radiology 1988; 167:769-774.
4. Robertson PL, Schweitzer ME, Bartolozzi AR, Ugoni A. Anterior cruciate ligament tears: evaluation of multiple signs with MR imaging. Radiology 1994;193:829–834.
5. Lee JK, Yao L, Phelps CT, Wirth CR, Czajka J, Lozman J. Anterior cruciate ligament tears: MR imaging compared with arthroscopy and clinical tests. Radiology 1988; 166:861–864.
6. Vahey TN, Broome DR, Kayes KJ, Shelbourne KD. Acute and chronic tears of the anterior cruciate ligament: differential features at MR imaging. Radiology 1991;181: 251–253.
7. LaPrade RF, Gilbert TJ, Bollom TS, Wentorf F, Chaljub G. The magnetic resonance imaging appearance of individual structures of the posterolateral knee: a prospective study of normal knees and knees with surgically verified grade III injuries. Am J Sports Med 2000;28:191–199.
8. De Smet AA, Norris MA, Yandow DR, Quintana FA, Graf BK, Keene JS. MR diagnosis of meniscal tears of the knee: importance of high signal in the meniscus that extends to the surface. AJR Am J Roentgenol 1993; 161:101–107.
9. Mesgarzadeh M, Moyer R, Leder DS, et al. MR imaging of the knee: expanded classification and pitfalls to interpretation of meniscal tears. RadioGraphics 1993;13:489–500.
10. Firooznia H, Golimbu C, Rafii M. MR imaging of the menisci: fundamentals of anatomy and pathology. Magn Reson Imaging Clin N Am 1994;2:325–347.
11. Schweitzer ME, Falk A, Berthoty D, Mitchell M, Resnick D. Knee effusion: normal distribution of fluid. AJR Am J Roentgenol 1992;159:361–363.
12. Scapinelli R. Blood supply of the human patella: its relation to ischaemic necrosis after fracture. J Bone Joint Surg Br 1967;49: 563–570.
13. Howard PE, Wilson JW, Robbins TA, Ribble GA. Normal blood supply of the canine patella. Am J Vet Res 1986;47:401–403.
14. Allman RM, Brower AC. Circulatory patterns of deossification. Radiol Clin North Am 1981;19:553–569.
15. Laroche M. Intraosseous circulation from physiology to disease. Joint Bone Spine 2002;69:262–269.
16. De Lorimer AA, Minear WL, Boyd HB. Reflex hyperemic deossifications regional to joints of the extremities. Radiology 1946; 46:227–236.
17. Gross TS, Damji AA, Judex S, Bray RC, Zernicke RF. Bone hyperemia precedes disuse-induced intracortical bone resorption. J Appl Physiol 1999;86:230–235.
18. Piekarski K, Munro M. Transport mechanism operating between blood supply and osteocytes in long bones. Nature 1977;269:80–82.
19. Kofoed H, Lindenberg S. Effect of simulated joint effusion on subchondral haemodynamics and metabolism. Injury 1986;17:274–276.
20. Bunger C, Harving S, Hjermind J, Bunger EH. Relationship between intraosseous pressures and intra-articular pressure in arthritis of the knee: an experimental study in immature dogs. Acta Orthop Scand 1983;54:188–193.