Title	MR evidence of characteristics in symptomatic osteoarthritis of the temporomandibular joint: increased signal intensity ratio on proton density-weighted images of bone marrow in the mandibular condyle
Author(s) Alternative	Yajima, A; Sano, T; Otonari-Yamamoto, M; Otonari, T; Ohkubo, M; Harada, T; Wakoh, M
Journal	Cranio : the journal of craniomandibular practice, 25(4): 250-256
URL	http://hdl.handle.net/10130/668
Right	

MR Evidence of Characteristics in Symptomatic Osteoarthritis of the Temporomandibular Joint: Increased Signal Intensity Ratio on Proton Density-Weighted Images of Bone Marrow in the Mandibular Condyle

Aya Yajima, D.D.S.; Tsukasa Sano D.D.S., Ph.D.; Mika Otonari-Yamamoto, D.D.S., Ph.D.; Takamichi Otonari, D.D.S., Ph.D.; Mai Ohkubo, D.D.S., Ph.D.; Takuya Harada, D.D.S., Ph.D.; Mamoru Wakoh D.D.S., Ph.D.

0886-9634/2504-250$05.00/0, THE JOURNAL OF CRANIOMANDIBULAR PRACTICE,

Manuscript received March 29, 2007; revised manuscript received June 12, 2007; accepted June 20, 2007

Address for correspondence:
Dr. Aya Yajima
Dept. of Oral and Maxillofacial Radiology
Tokyo Dental College
1-2-2 Masago, Mihama-ku
Chiba 261-8502
Japan
E-mail: ayayajima@tdc.ac.jp

ABSTRACT: The purpose of this study was to clarify the presence of pain and a correlation between pain and characteristics of signal intensity of mandibular bone marrow in temporomandibular joints (TMJ) with osteoarthritis (OA). A total of 196 joints in 98 patients with TMJ disorders were examined using magnetic resonance imaging (MRI). A pain score and signal intensity on mandibular bone marrow were analyzed in the TMJ with OA. TMJ with OA showed a higher degree of pain compared to those without ($p<0.05$). During opening, the joints in the higher signal intensity group showed a significantly higher degree of pain compared to the joints in the lower signal intensity group in those with OA on proton density weighted images ($p<0.05$). It was concluded that TMJ with osteoarthritis is related to pain and that a symptomatic osteoarthritic TMJ can accompany bone marrow changes in the condyle, showing an increased signal on proton density weighted images.

Dr. Aya Yajima *received her D.D.S. degree from Tokyo Dental College, Chiba, Japan in 2005. She is a graduate school student of oral and maxillofacial radiology at the same college.*

Osteoarthritis is radiographically characterized by irregularities of the articular surfaces, osteophytosis, deformity, etc., and is frequently seen in joints with longstanding disk displacement without reduction.[1,2] It is suggested as a source of pain, but it is also often seen in asymptomatic subjects.[1,3-6] Magnetic resonance (MR) evidence of bone marrow abnormality of the mandibular condyle may be related to osteoarthritis,[7,8] but there has been no report of such bone marrow changes in symptomatic osteoarthritis of the temporomandibular joint (TMJ). The purpose of this study was to clarify a presence of pain and a correlation between pain and characteristics of signal intensity of bone marrow in the mandibular condyle in osteoarthritis of the TMJ.

Materials and Methods

The subjects included 104 consecutive patients with TMJ symptoms and dysfunction referred for MRI. Six of the patients were excluded from the study. In five, it was difficult to place the regions of interest (ROI) because of body movement artifact in one case, and a diagnosis of

synovial chondromatosis based on the images obtained in another. A total of 196 joints were studied. The mean age of the patients, 74 women and 24 men, was 36.7 years, with a range of 13 to 76. Immediately prior to MRI, the patients rated their degree of pain, using a visual analog scale (VAS) in which the maximum is 100 and the minimum 0, while both chewing and during opening. The degree of pain was scored separately for the left and right joints.

MRI was performed using the 1.5 Tesla MR imager (Magnetom Symphony, Siemens, Erlangen, Germany), using a double loop array coil. Bilateral proton density-weighted TMJ images were obtained at both closed- and open-mouth positions by using a fast-spin echo sequence (**Table 1**).

The axial scout view was obtained parallel to the occlusal plane. The views were selected according to the position on the long axis where the widest area was available between the lateral and medial poles. Corrected sagittal images were obtained perpendicular to the long axis and axial view. The images were saved as DICOM (D-I-C-O-M) files and read for a measurement of signal intensity using a public domain software (ImageJ 1.32j NIH, USA) for image analysis. We calculated a signal intensity of bone marrow adjacent to the top of the condyle to examine a relationship between osseous change and marrow changes in the condyle. The decision as to which images were suitable for ROI selection on condylar marrow was made by a consensus of two oral and maxillofacial radiologists. ROI was selected only in images where the entire area between the mandibular condyle and the lower border of the mandible could be imaged. The locations and sizes of ROI had to be consistent on all images from different joints to guarantee higher reliability.[9] The average area for condylar marrow was calculated from a total of 20 images to determine the size of the ROI. This resulted in a 4.5 mm^2 area for condylar marrow. The signal intensity ratio (SI) was calculated using the signal intensity of the condylar marrow on the ROI (SIC) and of the gray matter on a ROI (SIM) as follows: SI=SIC/SIM.

To obtain the measurement, a 5.9 mm^2 of ROI was defined and placed over the gray matter as a reference point. It was placed closest to the mandibular condyle on a line perpendicular to the top of the condyle in the gray matter (**Figure 1**). On the basis of the MRI findings, the status of the TMJ was classified as described in **Table 2**.

A pain score was compared between joints with osteoarthritis and those without. In joints with osteoarthritis, another pain score was also compared between joints with a higher or lower signal intensity of bone marrow of the condyle.

A statistical analysis was performed using Wilcoxon's rank sum test to compare the difference between the two groups. A probability of less than 0.05 was considered statistically significant.

Informed consent was obtained from each patient in accordance with the Declaration of Helsinki. This study was a retrospective study, and approval was obtained from our Institutional Review Board in advance.

Table 1
MR Imaging Parameters

Time of repetition/time of echo (msec)	3300/14
Number of signal averages	1
Field of view (cm)	150x150
Slice thickness (mm)	3
Matrix	512x512
Scan time (sec)	171
Flip angle	180°
GAP thickness (mm)	0.6
Turbo factor (echo train length)	3

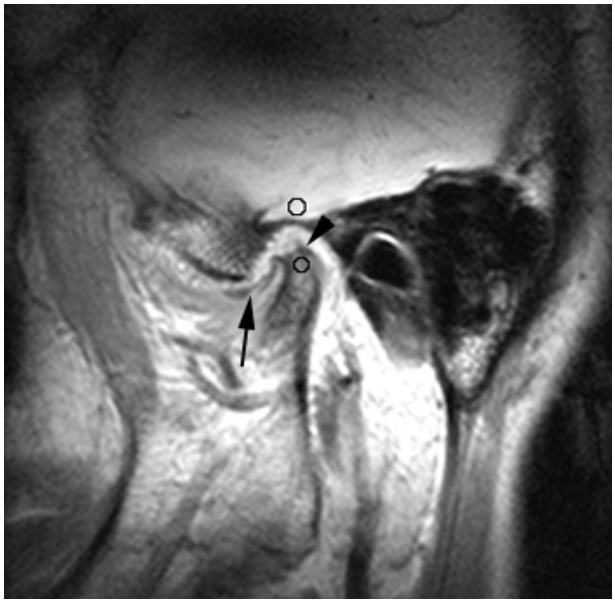

Figure 1
Painful TMJ with disk displacement without reduction associated with osteoarthritis from 58-year-old woman. On corrected sagittal proton density-weighted MR image, the disk (arrow) is anteiorly displaced and the condyle shows an erosion on the top (arrowhead). The figure shows circles; one is ROI in gray matter and another is a ROI in the bone marrow of the mandible. This joint was classified into the higher signal intensity group according to the signal intensity ratio calculated using the above two RCMs

Table 2
Criteria for Interpretation of the MR Images

Inclusion criteria for joints without osteoarthritis

Osseous components with evidence of normal cortical bone and any of disk position criteria, as follows:

- Disk located with its posterior band superior to the condyle in the closed- and open-mouth positions (normal disk position)
- Posterior band of disk located anterior, medial, or lateral to the normal position on top of the condyle in the closed-mouth position and normal condyle located between the anterior and posterior bands of the disk in the open-mouth position images (disk displacement with reduction)
- Posterior band of disk located anterior, medial, or lateral to the condyle during all mandibular movements (disk displacement without reduction)

Inclusion criteria for joints with osteoarthritis

Osteoarthritis evident as osteophytes, erosions, or deformity of the condyle and/or temporal component with evidence of disk displacement without reduction

Results

The results are summarized in **Tables 3**-**7**. There was a significantly higher degree of pain on chewing in the 40 joints with osteoarthritis than in the 156 joints without (p=0.04) (**Table 3**).

Joints with osteoarthritis showed a higher mean of the signal intensity ratio, but there was no statistically significant difference in the signal intensity ratio between joints with osteoarthritis and joints without (**Table 5**). In 40 joints with osteoarthritis, the joints were divided into higher or lower signal intensity groups according to the median (1.23) of the signal intensity ratio. During opening, the joints in the higher signal intensity group showed significantly higher degrees of pain compared to the joints in the lower signal intensity group (p=0.03) (**Table 7**).

Discussion

This study showed increased pain in joints with osteoarthritis. Several studies have found that osteoarthritis was not a significant factor in TMJ pain,[1,3-6] whereas joints with osteoarthritis are quite likely related to it.[5,10-12] Osteo-arthritis is present in a large proportion of older people and is usually completely asymptomatic. Symptoms related to temporomandibular dysfunction decrease with age and are often remitting and self-limiting.[13-16] Kurita, et al.[10] suggested that the presence of TMJ pain on mandibular movement is not a reliable predictor of osteoarthritis of the TMJ. Mechanical and chemical stimuli are proposed as possible causes of pain in and around the osteoarthritic joint.[10]

The discrepancy that can occur between findings obtained by imaging and patient symptomatology highlights the need for effective study to determine which findings are significant. One study[10] proposed that degenerative changes that are not evident in radiographic examination are also an important factor in joint pain. Joint pain is likely to occur in pathogenetic conditions in which condylar osteoarthritic change takes place.[10]

There are several other possible sources of TMJ pain, such as impingement and compression,[17] inflammatory changes[3] in the retrodiskal tissue, inflammatory changes in the synovial membrane fluid resulting in joint effusion,[18,19] and capsulitis.[20] Possible causes of pain in and around an osteoarthritic joint may include other factors that influence various components of the joint and periarticular structure, such as increased pressure, destruction, or inflammatory mediators.[10] Several studies suggest an association between osteoarthritis and bone marrow abnormalities of the condyle. One study[8] suggests that abnormalities suggestive of edema or osteonecrosis in the bone marrow are initially separate entities from osteoarthritis.

In the present study, we focused on the changes of condylar bone marrow in osteoarthritis on proton density-weighted images that are routinely imaged during clinical work at our institute to detect disk abnormality.[21]

This study identified a relationship between increased pain and increased proton density-weighted signal intensity in the upper portion of the mandibular condyle in joints with osteoarthritis using quantitative analysis.

In advanced cases of osteoarthritis, microscopic foci of necrosis are frequently found immediately adjacent to the joint surface.[7] Small infarcts can occasionally occur, but

Table 3
Association Between the Presence of Osteoarthritis and Pain Score on Chewing

Age		10-19	20-29	30-39	40-49	50-59	60-69	70-79
Mean	OA	6.75 (4)	9.23 (7)	24.25 (12)	8.50 (4)	15.00 (6)	34.50 (2)	11.20 (5)
	without OA	4.88 (26)	8.10 (50)	8.97 (31)	3.20 (10)	20.67 (9)	17.26 (27)	3.33 (3)
SD	OA	8.44	8.48	26.02	10.62	21.64	-	13.91
	without OA	6.77	15.39	15.34	2.71	31.55	19.19	-

OA: Osteoarthritis
SD: Standard deviation
Number of joints in parentheses

Table 4
Association Between the Presence of Osteoarthritis and Pain Score During Opening

Age		10-19	20-29	30-39	40-49	50-59	60-69	70-79
Mean	OA	22.50 (4)	13.29 (7)	25.42 (12)	17.00 (4)	20.00 (6)	36.50 (2)	28.40 (5)
	without OA	9.19 (26)	10.58 (50)	15.35 (31)	5.90 (10)	19.44 (9)	23.59 (27)	42.30 (3)
SD	OA	22.07	16.09	24.24	19.00	23.53	-	15.21
	without OA	11.37	16.16	20.57	5.82	30.99	22.55	-

OA: Osteoarthritis
SD: Standard deviation
Number of joints in parentheses

Table 5
Difference in Signal Intensity Ratio in Joints With Osteoarthritis and Without

		Signal Intensity		
	No. joints	Mean	SD	Median
Joints with OA	40	1.35	0.45	1.2
Joints without OA	156	1.24	0.52	1.1

OA: Osteoarthritis
SD: Standard deviation
Note: There was no significant difference between joints with osteoarthritis and without (Wilcoxon's rank sum test)

again they are found at the joint surface and only in joints with advanced osteoarthritis.[22] This may be in accordance with the results in the present study that MR evidence of marrow changes in symptomatic osteoarthritis was seen adjacent to the top of the condyle.

Increased pain with bone marrow alterations such as edema and osteonecrosis has been well established for other joints, such as the femoral head,[23] knee,[24] wrist,[25] and shoulder.[26]

Several MRI studies describe abnormalities in the mandibular condyle with an appearance similar to osteonecrosis of other joints.[19, 27-30] This leads to the assumption that osteonecrosis can also affect the mandibular condyle.[27-30] This issue was discussed long before MRI was available,[32] but it has remained controversial because no histological correlations have been available.

Table 6
Association Between Signal Intensity Ratio and Pain Score in Joints With Osteoarthritis on Chewing

Age		10-19	20-29	30-39	40-49	50-59	60-69	70-79
Mean	<1.23	0	24.00	14.00	-	27.13	1.00	19.80
		(1)	(1)	(3)	(0)	(8)	(2)	(5)
	≥1.23	-	16.00	9.56	22.00	5.33	-	-
		(0)	(5)	(9)	(3)	(3)	(0)	(0)
SD	<1.23	-	-	-	-	29.75	-	24.13
	≥1.23	-	14.52	10.52	-	-	-	-

SD: Standard deviation
Number of joints in parentheses

Larheim and Westesson, et al. analyzed core biopsies from 50 mandibular condyles and correlated histological findings with MR signal patterns categorized as normal, edema, or osteonecrosis (**Tables 7-9**). They found that edema and osteonecrosis occurred in the marrow of the condyle.[7] An earlier study[32] found that joints with bone marrow abnormalities in the mandibular condyle were markedly more painful than those without. Emshoff, et al.[3,33] evaluated risk factors for TMJ pain as a function of MRI findings. Significant increases in the risk of TMJ pain occurred with disk displacement without reduction in combination with osteoarthrosis and bone marrow edema. However, there is no description of criteria for bone marrow edema judged on MRI in that study. There has been no quantitative study on the signal intensity of the mandibular condyle in joints with osteoarthritis.

Normal bone marrow has high signal intensity at T1, proton density, and T2-weighted images because of its fat content. Any process that alters or replaces marrow fat will decrease T1 and the proton density-weighted signal. The T2 signal strength will depend on the type of tissue that replaces the normal marrow. Necrotic tissue, hematoma, and inflammatory debris will have higher T2 signals than neoplastic tissue. Fibrotic or solerotic tissue has low signals on T1, proton density, and T2-weighted sequences. Lang, et al.[34] studied patients after lumbar fusion and found high signal intensities on T1-weighted images in patients with a solid fusion. A localized area of high signal intensity seen within the spinal cord on T2-weighted or proton density spin-echo (SE) images in cases of compression of the spinal canal and cord is described.[35] In a brain study on proton density, the edema is bright.[36] The bone marrow categories with edema and osteonecrosis do not include high signal but low signal intensity on proton density-weighted images (**Table 7**). This is not in accordance with our results, showing

Table 7
Association Between Signal Intensity Ratio and Pain Score in Joints With Osteoarthritis During Opening

Age		10-19	20-29	30-39	40-49	50-59	60-69	70-79
Mean	<1.23	0	26.00	26.00	-	33.50	1.50	27.20
		(1)	(1)	(3)	(0)	(8)	(2)	(5)
	≥1.23	-	18.80	15.67	23.33	25.00	-	-
		(0)	(5)	(9)	(3)	(3)	(0)	(0)
SD	<1.23	-	-	-	-	26.71	-	23.79
	≥1.23	-	21.78	15.28	-	-	-	-

SD: Standard deviation
Number of joints in parentheses

Table 8
Histological Classification of Core Biopsies of Mandibular Condyle Adopted from Larheim, et al.[7]

Histological Classification		Description of microscopic features
I. Normal	a.	*Without reacive bone formation*: Normal marrow architecture, preservation of the hematopoietic marrow, normal trabecular, and cortical cone architecture.
	b.	*With reactive bone formation*: Normal marrow architecture, preservation of the hematopoietic marrow, evidence of increased bone cell activity, increased bone remodeling, and reactive bone formation.
II. Edema	a.	*Early*: Edema fluid with serum protein exudate within marrow interstitium, preservation of hematopoietic marrow elements, no evidence of reticulin fibrosis.
	b.	*Late*: Edema fluid with serum protein exudate within marrow interstitium, preservation of hematopoietic marrow elements, focal evidence of disruption of marrow interstitium and reticulin fibrosis.
III. Osteonecrosis	a.	*Early*: Loss of hematopoietic marrow elements, loss of normal marrow stroma and marrow fat, no evidence of reticulin fibrosis.
	b.	*Late*: Loss of hematopoietic marrow elements, loss of normal marrow stroma and marrow fat, evidence of reticulin fibrosis.

increased proton density-weighted signals seen on symptomatic osteoarthritis. Larheim, et al.[7] described a histological classification of core biopsies of the mandibular condyle. Histological evidence of bone marrow edema was also found where there was no evidence of osteonecrosis, suggesting that edema is a precursor in osteonecrotic development, as in other joints.[7,37] Another study[3] suggested that pain is more severe in TMJ with marrow edema of the mandibular condyle than in those with osteonecrosis. The early stage of edema category includes edema fluid with serum protein exudate within marrow interstitium, preservation of hematopoietic marrow elements, and no evidence of reticulin fibrosis (**Table 8**). The present result, increased proton density-weighted signals in TMJ with symptomatic osteoarthritis, may reflect this early stage of edema.

This study concluded that TMJ with osteoarthritis is related to pain, and a symptomatic osteoarthritic TMJ can accompany bone marrow changes in the upper portion of the condyle adjacent to osseous changes, showing increased signals on proton density images.

References

1. Westesson P-L, Rohlin M: Internal derangement related to osteoarthritis in temporomandibular joint autopsy specimens. *Oral Surg Oral Med Oral Pathol* 1984; 57:17-22.

Table 9
MR Classification of Bone Marrow of the Mandibular Condyle Adopted from Larheim, et al.[7]

MR Classification		Signal intensity patterns of bone marrow of the mandibular condyle
I. Normal		Homogenous bright proton density signal and homogenous intermediate T2 signal
II. Edema		Decreased proton density signal and increased T2 signal
III. Osteonecrosis	a.	Decreased proton density signal and decreased T2 signal; sclerosis pattern.
	b.	Combination of areas with decreased proton density and increased T2 signal with other areas with decreased proton density and decreased T2 signal; combined edema and sclerosis pattern.

2. Westesson P-L: Structural hard tissue changes in temporomandibular joints with internal derangement. *Oral Surg Oral Med Oral Pathol* 1985; 59:220-224.
3. Emshoff R, Brandlmaier I, Bertram S, Rudisch A: Risk factors for temporomandibular joint pain in patients with disk displacement without reduction. A magnetic resonance imaging study. *J Oral Rehab* 2003; 30:537-543.
4. Sano T: Recent developments in understanding temporomandibular joint disorders. Part 1: bone marrow abnormalities of the mandibular condyle. *Dentomaxillofac Radiol* 2000; 29:7-10.
5. Emshoff R, Innerhofer K, Rudisch A, Bertram S: The biological concept of "internal derangement and osteoarthrosis": a diagnostic approach in patients with temporomandibular joint pain? *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2002; 93:39-44.
6. Wiberg B, Wanman A: Signs of osteoarthrosis of the temporomandibular joints in young patients: a clinical and radiographic study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1998; 86:158-164.
7. Larheim TA, Westesson P-L, Hicks DG, Eriksson L, Brown DA: Osteonecrosis of the temporomandibular joint: correlation of magnetic resonance imaging and histology. *J Oral Maxillofac Surg* 1999; 57:888-898.
8. Sano T, Westesson P-L, Larheim TA, Rubin SJ, Tallents RH: Osteoarthritis and abnormal bone marrow of the mandibular condyle. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1999; 87:243-252.
9. Ogura A, Maeda F, Miyai A, Hongoh T: *Accuracy of contrast-to-noise ratio measurement for magnetic resonance clinical images*. Nippon Hoshasen Gijutsu Gakkai Zasshi 2004; 60:1543-1549. (pub. in Japanese)
10. Kurita H, Kojima Y, Nakatsuka A, Koike T, Kobayashi H, Kurashima K: Relationship between temporomandibular joint (TMJ)-related pain and morphological changes of the TMJ condyle in patients with temporomandibular disorders. *Dentomaxillofac Radiol* 2004; 33:329-333.
11. Emshoff R, Rudisch A, Innerhofer K, Bosch R, Bertram S: Temporomandibular joint internal derangement type III: relationship to magnetic resonance imaging findings of internal derangement and osteoarthrosis. An intraindividual approach. *Int J Oral Maxillofac Surg* 2001; 30:390-396.
12. Bertram S, Rudisch A, Innerhofer K, Pumpel E, Grubwieser G, Emshoff R: Diagnosing TMJ internal derangement and osteoarthritis with magnetic resonance imaging. *J Am Dent Assoc* 2001; 132:753-761.
13. Pereira FJ Jr, Lundh H, Westesson P-L, Carlsson LE: Clinical findings related to morphologic changes in TMJ autopsy specimens. *Oral Surg Oral Med Oral Pathol* 1994; 78:288-295.
14. Lundh H, Westesson P-L, Kopp S: A three-year follow-up of patients with reciprocal temporomandibular joint clicking. *Oral Surg Oral Med Oral Pathol* 1987; 63:530-533.
15. Rasmussen OC: Description of population and progress of symptoms in a longitudinal study of temporomandibular arthropathy. *Scand J Dent Res* 1981; 89:196-203.
16. Randolph CS, Greene CS, Moretti R, Forbes D, Perry HT: Conservative management of temporomandibular disorders: a posttreatment comparison between patients from a university clinic and from private practice. *Am J Orthod Dentofacial Orthop* 1990; 98:77-82.
17. Isberg A, Isacsson G, Johansson A-S, Larson O: Hyperplastic soft tissue formation in the temporomandibular joint associated with internal derangement. *Oral Surg Oral Med Oral Pathol* 1986; 61:32-38.
18. Westesson P-L, Brooks SL: Temporomandibular joint: relationship between MR evidence of effusion in the presence of pain and disk displacement. *Am J Roentgenol* 1992; 59:559-563.
19. Schellhas KP, Wilkes CH: Temporomandibular joint inflammation: comparison of MR fast scanning with T1- and T2-weighted imaging techniques. *Am J Roentgenol* 1989; 153:93-98.
20. Farrar WB, McCarthy WL Jr.: Inferior joint space arthrography and characteristics of condylar path in internal derangements of the TMJ. *J Prost Dent* 1979; 41:548-555.
21. Rao VM, Dalinka MK, Mitchell DG, Spritzer CE, Kaplan F, August CS, Axel L, Kressel HY: Osteopetrosis: MR characteristics at 1.5 T. *Radiol* 1986; 161:217.
22. Milgram JW: *Osteonecrosis due to traumatic and idiopathic causes in radiologic and histologic pathology of non-tumorous diseases of bones and joints*. Northbrook, IL: Northbrook Publishing Co., 1990.
23. Thickman D, Axel L, Kressel HY: Magnetic resonance imaging of avascular necrosis of the femoral head. *Skeletal Radiol* 1986: 15:133.
24. Wilson AJ, Murphy WA, Hardy DC, Totty WG: Transient osteoporosis: transient bone marrow edema? *Radiol* 1988; 167:757-760.
25. Ekerot L, Eiken O: Idiopathic avascular necrosis of the scaphoid. Case report. *Scand J Plast Reconstr Surg* 1981; 15:69.
26. Dzioba RB, Quinlan WJ: Avascular necrosis of the glenoid. J *Trauma* 1984; 24:448.
27. Schellhas KP, Wilkes CH, Omlie MR, Peterson CM, Johnson SD, Keck RJ, Block JC, Fritts HM, Heithoff KB: The diagnosis of temporomandibular joint disease: two-compartment arthrography and MR. *Am J Roentgenol* 1988; 151: 341-350.
28. Schellhas KP: Internal derangement of the temporomandibular joint: radiologic staging with clinical, surgical, and pathologic correlation. *Magn Reson Imaging* 1989; 7:495-515.
29. Schellhas KP, Wilkes CH, Fritts HM, Omlie MR, Lagrotteria LB: MR of osteochondritis dissecans and avascular necrosis of the mandibular condyle. *Am J Roentgenol* 1989; 152:551-560.
30. Schellhas K: Temporomandibular joint injuries. *Radiol* 1989; 173:211-216.
31. Reiskin AB: Aseptic necrosis of the mandibular condyle: a common problem? *Quintessence Int* 1979; 2:85-89.
32. Sano T, Westesson P-L, Larheim TA, Takagi R: The association of temporomandibular joint pain with abnormal bone marrow of the mandibular condyle. *J Oral Maxillofac Surg* 2000; 58:254-257.
33. Emshoff R, Brandlmaier I, Bertram S, Rudisch A: Relative odds of temporomandibular joint pain as a function of magnetic resonance imaging findings of internal derangement, osteoarthrosis, effusion, and bone marrow edema. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2003; 95:437-445.
34. Lang EK: Renal, perirenal, and pararenal abscesses: percutaneous drainage. *Radiol* 1990; 174:109.
35. Takahashi M, Sakamoto Y, Miyawaki M, Bussaka H: Increased MR signal intensity secondary to chronic cervical cord compression. *Neuroradiol* 1987; 29:550-556.
36. Ray H: *MRI The basics*. 2nd ed. Lippincott Williams & Wilkins: A Wolters Kluwer Co., 2004.
37. Sano T, Westesson P-L, Yamamoto M, Okano T: Differences in temporomandibular joint pain and age distribution between marrow edema and osteonecrosis in the mandibular condyle. *J Craniomandib Pract* 2004; 22:283-288.

Dr. Tsukasa Sano *received his D.D.S. degree from Showa University, Tokyo, Japan in 1987 and his Ph.D. degree from the same university in 1991. He was a visiting assistant professor in the Department of Radiology, University of Rochester Medical Center, Rochester, New York and currently is a professor and the chairman of oral and maxillofacial radiology, Tokyo Dental College, Chiba, Japan.*

Dr. Mika Otonari-Yamamoto *received her D.D.S. degree from Nagasaki University, Nagasaki, Japan in 1995 and her Ph.D. degree from Showa University, Tokyo, Japan in 2003. She was a research fellow in the Department of Radiology, Massachusetts Eye and Ear Infirmary, Harvard Medical School, Boston and is now an assistant professor of oral and maxillofacial radiology, Tokyo Dental College, Chiba, Japan*

Dr. Takamichi Otonari *received his D.D.S. degree from Tsurumi University, Yokohama, Japan in 1998 and his Ph.D. degree from Tokyo Dental College in 2006. He is an assistant professor of oral and maxillofacial radiology, Tokyo Dental College, Chiba, Japan.*

Dr. Mai Ohkubo *received her D.D.S. degree from Tokyo Dental College, Chiba, Japan in 1999 and her Ph.D. degree from Showa University, Tokyo, Japan in 2003. She is an assistant professor of oral and maxillofacial radiology, Tokyo Dental College, Chiba, Japan.*

Dr. Takuya Harada *received his D.D.S. degree from Tokyo Dental College, Chiba, Japan in 1993 and his Ph.D. degree from the same college in 1997. He is an assistant professor of oral and maxillofacial radiology, Tokyo Dental College, Chiba, Japan.*

Dr. Mamoru Wakoh *received his D.D.S. degree from Tokyo Dental College, Chiba, Japan in 1984 and his Ph.D. degree from the same college in 1988. He was a visiting professor in the Division of Radiology and Imaging Sciences, School of Dentistry, University of Louisville, Louisville, Kentucky. He is currently an associate professor of oral and maxillofacial radiology, Tokyo Dental College, Chiba, Japan.*