Radiographic Assessment of Asymmetry of the Mandible

Per-Lennart Westesson, Ross H. Tallents, Richard W. Katzberg, and Jeffrey A. Guay

PURPOSE: To assess the relationship between mandibular asymmetry and disorders of the temporomandibular joint. METHODS: We used advanced imaging of the temporomandibular joint to distinguish different causes of mandibular asymmetry. MR imaging and arthrography were applied to the temporomandibular joints of 11 patients presenting with mandibular asymmetry. RESULTS: Condyle hyperplasia was identified as the cause of the asymmetry in 5 patients. In the other 6 patients the mandibular condyle was normal on the long side, but the short side of the face demonstrated a small condyle head, short condyle neck associated with disk displacement, internal derangement, and degenerative joint disease of the temporomandibular joint. CONCLUSIONS: These observations suggest that both condyle hyperplasia on the long side of the mandible and disk displacement and degenerative joint disease of the temporomandibular joint on the short side can cause mandibular asymmetry. It was concluded that MR imaging or arthrography can be valuable for understanding the cause of mandibular asymmetry and be effective in treatment planning.

Index terms: Mandible; Temporomandibular joint, abnormalities and anomalies; Temporomandibular joint, magnetic resonance


The most prominent facial features of mandibular asymmetry include a shift of the chin to the short side and prominence of the mandibular (gonion) angle on the long side (1). Dental features may include an open bite on the long side, shift of the mandibular midline away from the long side, cross-bite on the short side, and a tilt of the frontal occlusal plane (1–5). There are several obvious causes of mandibular asymmetry such as trauma with fracture, tumors, and congenital anomalies. In many cases, however, the cause of mandibular asymmetry leading to facial deformity is unclear on physical examination (6, 7).

Previous radiographic studies have suggested that the long side has an enlarged condyle head and an elongated condyle neck (1, 6). In some circumstances, histologic samples have been acquired and shown abnormal cellular activity of the elongated side (8–10). Internal derangement of the temporomandibular joint as a cause of shortened condyle head and neck was first reported in children in 1985 (11). Decreased condyle growth and/or degenerative joint disease secondary to disk displacement was documented to result in facial asymmetry in a series of pediatric patients (11).

We have evaluated eleven young women with prominent mandibular asymmetry on physical examination. These patients were evaluated by detailed physical examination, tomography, radionuclide skeletal imaging, and arthrography or magnetic resonance (MR) imaging of both temporomandibular joints. The objective of this investigation was to describe our findings based on this detailed assessment concerning the relationship between mandibular asymmetry and temporomandibular joint disorders.

Materials and Methods

Eleven patients presented with prominent mandibular asymmetry on physical examination (Table 1). These patients were prospectively selected over a 2-year period from a consecutive series of about 300 patients seeking...
<table>
<thead>
<tr>
<th>Chin Shift Toward the Short Side</th>
<th>Pain</th>
<th>Dental Findings</th>
<th>Tomographic Findings</th>
<th>Arthrography or MR imaging</th>
<th>Radionuclide Imaging</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>+</td>
<td>+</td>
<td>Shift of mandibular dental midline toward short side</td>
<td>Elongated condyle neck</td>
<td>Normal</td>
<td>Normal</td>
<td>Not performed</td>
</tr>
<tr>
<td>+</td>
<td>+</td>
<td>Shift of mandibular dental midline toward short side; lateral open bite on long side</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>+</td>
</tr>
<tr>
<td>+</td>
<td>-</td>
<td>Shift of mandibular dental midline toward short side</td>
<td>Elongated condyle neck</td>
<td>Decreased joint space</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>+</td>
<td>+</td>
<td>Shift of mandibular dental midline toward short side; lateral open bite on long side; cross bite on short side</td>
<td>Elongated condyle neck</td>
<td>Decreased joint space</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>+</td>
<td>+</td>
<td>Shift of mandibular dental midline toward short side</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
<td>+</td>
</tr>
<tr>
<td>+</td>
<td>+</td>
<td>Shift of mandibular dental midline toward short side</td>
<td>Normal</td>
<td>Flattened condyle</td>
<td>Normal</td>
<td>Disk displacement without reduction</td>
</tr>
<tr>
<td>+</td>
<td>+</td>
<td>Shift of mandibular dental midline toward short side; lateral open bite on long side; cross bite on short side</td>
<td>Normal</td>
<td>Central erosion</td>
<td>Normal</td>
<td>Disk displacement without reduction</td>
</tr>
<tr>
<td>+</td>
<td>+</td>
<td>Shift of mandibular dental midline toward short side; cross bite on short side</td>
<td>Normal</td>
<td>Thin condyle, neck flattened sclerotic condyle</td>
<td>Disk displacement without reduction</td>
<td>Not performed</td>
</tr>
<tr>
<td>+</td>
<td>+</td>
<td>Shift of mandibular dental midline toward short side; cross bite on short side</td>
<td>Normal</td>
<td>Flattened condyle</td>
<td>Normal</td>
<td>Disk displacement with reduction</td>
</tr>
<tr>
<td>+</td>
<td>+</td>
<td>Shift of mandibular dental midline toward short side</td>
<td>Normal</td>
<td>Small flat condyle</td>
<td>Normal</td>
<td>Disk displacement without reduction</td>
</tr>
<tr>
<td>+</td>
<td>+</td>
<td>Shift of mandibular dental midline toward short side; lateral open bite on long side</td>
<td>Normal</td>
<td>Small condyle</td>
<td>Normal</td>
<td>Disk displacement without reduction</td>
</tr>
</tbody>
</table>
treatment for symptoms related to orofacial pain or temporomandibular joint pain and dysfunction. The criterion for inclusion was prominent mandibular/facial asymmetry on physical examination. All of the patients included were female; they ranged in age from 12 to 32 years, with a mean age of 24 years. The clinical assessment included an evaluation of signs and symptoms of facial pain and/or evidence for internal derangement of the temporomandibular joint. No patient had a history of mandibular or condylar fracture.

Cephalometrically corrected linear tomograms were obtained of both temporomandibular joints and mandible in the lateral plane with the jaw closed and opened. The presence of degenerative joint disease was recorded if there was evidence of erosion of the condyle head, flattening of the condyle head, and osteophyte formation as previously described (12). Anteroposterior radiographs of the facial skeleton were also acquired in all patients. The long versus the short side of the mandible was determined by measuring the distances from the top of the condyle (condylion) to the angle of the mandible (gonion). A difference of left and right side greater than 3 mm was considered mandibular asymmetry. All measurements were made in the cephalometrically corrected tomograms and in anteroposterior radiographs.

The soft-tissue structures of the temporomandibular joint were evaluated either by arthrography or MR. Temporomandibular joint arthrography was performed as previously described (13, 14). Briefly, this entails injecting a small amount of contrast material into the lower joint space using a 3/4-in 23-gauge scalp vein needle under fluoroscopic control. Patients were then evaluated by spot film radiography, video fluoroscopy, and, in some cases, arthrotomography. The arthrograms were interpreted as described in the literature (13, 15), and the position of the disk was classified as normal or anteriorly displaced with or without reduction.

The MR images were obtained as previously described (16) using 3-in surface coils placed over both right and left temporomandibular joints and with simultaneous imaging of both sides in the sagittal closed-jaw and sagittal opened-jaw positions using a 3-mm section thickness. Additionally, coronal images were acquired of both right and left temporomandibular joints in the closed-mouth position. In this study we used T1-weighted images (600–1000/11–20/1 [repetition time/echo time]). The images were evaluated for alterations in condyle shape and changes in signal intensity of the cancellous portion of bone and for the position and function of the disk. Thus, if the posterior band of the disk was located over the condyle at the closed-mouth position, the disk was described as normal. If the disk was displaced anteriorly or sideways from its superior position over the condyle, it was noted as disk displacement. Thus in the coronal plane of imaging, if the disk was shifted beyond the lateral or medial pole of the condyle, the disk was described as displaced in a sideways fashion (17).

Radionuclide skeletal imaging was performed using technetium-99m methylene diphosphonate and planar γ imaging of both right and left temporomandibular joints with the jaw closed and opened in a technique that has been previously described in detail (18). These images were evaluated for increased uptake of the radionuclide based on previously described techniques (18).

Results

Two distinct patient groups were identified. One consisted of those with an apparent unilateral condyle hyperplasia causing mandibular facial asymmetry (Fig 1). In these patients the condyle head was significantly larger than the contralateral side. The other group comprised those with a normal joint on the long side and a short condyle and condyle neck on the short side secondary to internal derangement and degenerative joint disease (Fig 2). Radiographic analysis of the patients in this study showed a long condyle and/or condyle neck of the long side of the face in five patients, suggesting condyle hyperplasia (Table 1 and Fig 1). The remaining six patients showed a short, deformed condyle with signs of degenerative joint disease of the short side of the face (Fig 2). Arthrography or MR showed disk displacement without reduction in the six patients with a short condyle, all associated with degenerative joint disease (Fig 2). In the joints with condyle hyperplasia there were no signs of internal derangement on the long or short side of the face (Fig 1). One patient had bilateral internal derangement, and the most pronounced internal derangement was demonstrated in the joint on the short side.

Panoramic tomograms and bilateral arthograms from an asymptomatic volunteer without clinical evidence of facial or mandibular asymmetry are shown in Figure 3 for comparison. Bilateral tomograms and arthograms of the patient with facial asymmetry secondary to degenerative joint disease and internal derangement are shown in Figure 4. It was not possible on clinical examination to differentiate these two patient groups. Tomography consistently showed signs of degenerative joint disease in the short condyles. Signs of degenerative joint disease were not seen in any of the patients with condyle hyperplasia.

Radionuclide imaging was abnormal on the long side in six patients, four with condyle hyperplasia and two with internal derangement. Radionuclide imaging was abnormal on the short side in five patients, two with internal derangement and three with normal joints (Table 1).
Fig. 1. Condyle hyperplasia.
A, Photograph and B, sagittal, and C, coronal MR images of the left condyle of a patient with deviation of the chin to the right. The MR images show elongation of the condyle and condyle neck (arrows) as the cause of mandibular asymmetry.
D, Surgical treatment with removal of the superior part of the condyle corrected the asymmetry and histology of the specimen. Superiorly there is a layer of highly cellular band of fibrous connective tissue (F), which displays active intramembranous osteogenesis. This is located superior to the more dense cortical bone (C). Brisk osteoblastic rimming (arrows) is evident along trabeculae adjacent to the zone of osteogenesis (hematoxylin and eosin stain; magnification, 100X).

Five of the six patients showing mandibular asymmetry associated with internal derangement had surgery. The arthrographic or MR findings of disk displacement was confirmed at surgery in these five patients. One of the patients with mandibular asymmetry caused by condyle hyperplasia (Fig 1) also had surgery and histologic evidence of active osteogenesis (Fig 1D).

Discussion

There are many causes of mandibular asymmetry (Fig 5). Principally they can be divided into two groups, namely those with an enlargement of the condyle and condyle neck on the long side and those with a decrease in size of the condyle and condyle neck on the short side. Several of the causes are obvious by history, physical examination, and plain film radiographic studies such as fractures, tumors, and congenital anomalies. In the majority of patients with mild and moderate mandibular asymmetry, however, the cause is not obvious at clinical examination. Enlargement of the condyle and the condyle neck on the long side has been recognized for a long time as a cause of mandibular asymmetry (1-10). Diminution of the condyle and condyle neck on the short side secondary to disk displacement, internal derangement, and degenerative joint disease has not, until recently, been recognized as a cause of mandibular asymmetry in children (11) and adults (19). The findings in this study support the hypothesis that disk displacement, internal derangement, and degenerative joint disease can cause mandibular asymmetry.

In the classical group having enlargement of the condyle and elongation of the condyle neck on the long side (ie, condyle hyperplasia) there was no evidence of disk displacement, internal derangement, or degenerative joint disease either on the long or the short side of the mandible. In the other group with shortening of the condyle and condyle neck on the short side there was radiographic evidence of temporomandibular joint disease on the short side in all patients. On the long side soft-tissue imaging did not reveal
Fig. 2. Internal derangement and degenerative joint disease.
A, Photograph and B–G, MR images of a patient with mandibular asymmetry and deviation of the chin to the left secondary to unilateral internal derangement and degenerative joint disease. The MR images of the right temporomandibular joint (B–D) show the disk (arrow) superior to the condyle in the sagittal plane. The coronal image (C) shows a slight medial shift of disk position (arrow). On the short side (E–G) the disk (arrows) is anteriorly displaced without reduction. There are erosive changes (arrowheads) of the condyle.

any abnormalities. There was no characteristic evidence on physical examination to distinguish these two cause of mandibular asymmetry. Therefore, soft-tissue imaging of the temporomandibular joint seems to be of utmost value for understanding the cause of mandibular asymmetry and to help in treatment planning.

The prevalence of mandibular asymmetry is unknown. The patients in this study were collected over a 2-year period. They were selected
based on the finding of mandibular asymmetry on physical examination. The patients represent a subgroup of about 300 patients seen for symptoms of temporomandibular joint disorders. All of the patients in this study with mandibular asymmetry presented with temporomandibular joint pain and dysfunction. Little is known about the relative frequency of different causes of mandibular asymmetry. Our clinical experience and a recent study of 100 patients with mandibular asymmetry (19) suggest that disk displacement, internal derangement, and degenerative joint disease could be one of the main causes for mild and moderate mandibular asymmetry.

Mandibular asymmetry caused by enlargement of the condyle and condyle neck on the long side has been subdivided into four subgroups (20) (Fig 5). These are congenital hemifacial hypertrophy, condyle hyperplasia, hemimandibular hyperplasia, and hemimandibular elongation. The five patients in this study with enlargement of the long side were all classified as having condyle hyperplasia (Fig 5). Hyperplasia and hypertrophy have sometimes been used interchangeably. This is incorrect. Hypertrophy indicates the enlargement or overgrowth of an organ or part of an organ caused by an increase in size of its constituent cells. Hyperplasia includes the abnormal multiplication or increase in number of normal cells in normal arrangement in a tissue. For most conditions of the mandibular condyle in this article, hyperplasia is the correct term.

In mandibular asymmetry secondary to internal derangement and degenerative joint disease it is unclear whether erosive changes of the condyle or hypoplastic growth of the entire condyle and condyle neck cause the asymmetry. The impression from imaging studies in this paper and our clinical experience suggest that diminution of the size of the condyle is frequently not limited to the condyle head but also involves the condyle neck. This implies that internal derangement and degenerative joint disease has an effect on the growth of the entire mandibular condyle and condyle neck region. This is supported by earlier studies on the growth of the mandibular condyle and mandibular ramus (21–23). Further study is needed before we have a complete understanding of the mechanism for deficient growth of the mandible in this region secondary to internal derangement causing mandibular asymmetry.

Scintigraphy or nuclear medicine imaging has been used to assess mandibular asymmetry (24–26). These earlier studies suggested a value of scintigraphy to predict when mandibular uptake reached an adult norm (24) and also to be of value in treatment planning. In our experience with a mixed patient population in which some had condyle hyperplasia and others had internal derangement and degenerative joint disease, the
The value of scintigraphy seemed more limited because increased activity was seen in both the long and short sides of the mandible (Table 1). This suggests that scintigraphy alone would not be sufficient for assessment in a mixed patient population.

The discrepancy between the two sides of the mandible usually becomes apparent during the second decade of life when, for some unknown reason, one condyle becomes more active than the other (27). In some cases trauma has been suggested as the cause (28). Because the histo-
logic picture is relatively normal and the condition is self-limiting it cannot be considered a true neoplastic process. The treatment of unilateral condyle hyperplasia depends on whether the condyle is still growing (29). This is difficult to determine, and attempts have been made to use scintigraphy, but probably serial measurements on cephalometric radiographs taken at about 6-month intervals would be equally or more reliable. Resection of the hyperplastic condyle, radiation therapy, sagittal osteotomy, functional orthodontic appliances, or simple observation have been suggested as treatment. Today surgical treatment with resection or osteotomy probably would be the most frequently used treatment. When a large shift of the mandible is present, it may be necessary to reestablish the proper upper and lower jaw relationship by a combination of a subcondylar osteotomy and a LeFort I osteotomy of the maxilla.

Until recently there has been no consideration in treatment planning that mandibular asymmetry can be caused by degeneration of the short side and that this degeneration and/or growth deficit in the growing child or adolescent (21, 23, 30) could be related to internal derangement secondary to long-standing disk displacement. This clinical and radiographic study, however, suggests the possibility that internal derangement of the temporomandibular joint may be a cause of mandibular asymmetry. This information should have a significant impact on treatment selection.

Based on this investigation, we recommend bilateral tomography of both temporomandibular joints and rami of the mandible, to assess condyle hyperplasia versus condyle hypoplasia and/or degenerative joint disease, as well as bilateral sagittal and coronal MR imaging, for patients presenting with mandibular asymmetry (Table 2). Arthrography is an alternative if MR is not available. It is our belief that the status of soft-tissue structures of the temporomandibular joint might represent a significant component of the asym-

Fig. 5. Causes of mandibular asymmetry.

TABLE 2: Imaging strategy for patients with mandibular asymmetry

| 1. Lateral and anterior posterior cephalograms |
| 2. Lateral tomograms of temporomandibular joints, including entire mandibular ramus |
| 3. MR or arthrography of both left and right temporomandibular joints |
metry and that knowledge of this information is important for understanding the disease and effective treatment planning.

Acknowledgments

Dr Laurie C. Carter, State University of New York at Buffalo, is acknowledged for histologic preparation and analysis of the specimen shown in Figure 1D.

References