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Histiocytosis X: Review literature and a case report(โรคฮิสติโอไซโตซิส เอ็กซ์ : รายงานผู้ป่วยและบทความปริทัศน์)

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โรคฮิสติโอไซโตซิส เอ็กซ์ : รายงานผู้ป่วยและบทความปริทัศน์

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บทคัดย่อ

โรคในกลุ่มของฮิสติโอไซโตซิส เอ็กซ์ ประกอบด้วย 3 โรคคือ อีโอซิโนฟิลิค แกรนูโลม่า โรคแฮนด์ ชูลเลอร์ คริสเตียน และ โรคเลทเทอเรอร์ ไซวี โรคในกลุ่มนี้มักพบในผู้ป่วยที่มีอายุน้อยและมีลักษณะทางจุลพยาธิวิทยาคือมีการเพิ่มจำนวนของแลงเกอฮานเซลล์รวมถึงการรวมตัวของพวกอีโอซิโนฟิล ลักษณะของภาพถ่ายทางรังสีจะพบเงาดำวงเดียวหรือหลายวงที่มีขอบเขตชัดเจนหรือลักษณะของฟันลอยอยู่ในอากาศในกรณีที่มีความรุนแรงมาก เนื่องจากลักษณะอาการแสดงในช่องปากที่หลากหลายซึ่งอาจนำไปสู่ปัญหาในการวินิจฉัยโรค บทความนี้จึงเน้นเกี่ยวกับบทบาทของการตัดชิ้นเนื้อไปตรวจทางจุลพยาธิวิทยาโดยเฉพาะอย่างยิ่งการพบแลงเกอฮานเซลล์ การผ่าตัดเป็นการรักษาที่นิยมทำกันโดยเฉพาะรอยโรคในกระดูกขากรรไกรในขณะที่การใช้รังสีรักษาและเคมีบำบัดควรจะสงวนไว้ใช้ในกรณีที่รอยโรคมีเป็นจำนวนมากหรือไม่สามารถเข้าไปทำการผ่าตัดได้ บทความนี้เป็นการรายงานถึงผู้ป่วยชายอายุ 22 ปีซึ่งมีปัญหาเหงือกบวมและฟันโยก นอกจากนี้ยังพบต่อมน้ำเหลืองบริเวณใต้ขากรรไกรล่างมีขนาดใหญ่ขึ้น ภาพถ่ายทางรังสีพบรอยโรคในกระดูกหลายชิ้น ชิ้นเนื้อที่ตัดไปตรวจทางจุลพยาธิวิทยาแสดงให้เห็นถึงกลุ่มของแลงเกอฮานเซลล์ที่อยู่ปะปนกับอีโอซิโนฟิล แลงเกอฮานเซลล์ย้อมติดสีเมื่อใช้เทคนิคอิมมูโนฮิสโตเคมีสำหรับเอส-หนึ่งร้อย การรักษาในผู้ป่วยรายนี้กระทำโดยใช้การผ่าตัดร่วมกับรังสีรักษา

(ว.ทันต.จุฬาฯ. 2541;21:127-135)

Introduction

In 1953 Lichtenstein[1] defined the term histiocytosis X to encompass three clinical entities: eosinophilic granuloma, Hand-Schüller-Christian disease, and Letterer-Siwe disease. This grouping was based upon histologic similarities and on the possibility of transformation of one condition into another.[2,3] Despite histologic similarities, these three entities differred in clinical presentation and severity of involvement.[4,5] They were as follows:

1) Localized form of histiocytosis X (eosinophilic granuloma) presented with single or multiple skeletal lesions without extraskeletal involvement. It usually affected children and young adults. 2) Chronic disseminated form of histiocytosis X (Hand-Schüller-Christian disease) presented with both skeletal and

extraskeletal involvement. It followed a chronic clinical course and was characterized by a classic triad of diabetes insipidus, exophthalmos, and osteolytic skull defects. It usually affected children over the age of 3 years. 3) Acute disseminated form of histiocytosis X (Letterer-Siwe disease) presented with multiple organ involvement and followed a rapid, sometimes fatal, course. It usually affected infants and children less than 3 years of age.[5-8]. The terms histiocytosis X, idiopathic histiocytosis, Langerhans cell disease, Langerhans cell histiocytosis, and Langerhans granulomatosis were synonymous.[9,10]

Oral involvement of histiocytosis X has been reported to range from 10% to 77%.[2,6,11] Lesions found in the oral cavity may be the initial signs, and in many cases, they were the only manifestation of the disease.[11-13] The age of the patients afflicted with histiocytosis X varied, ranging from infants, children, to young adults.[6,14-17] Males were afflicted more often than females.[6,12,18] Skeletal lesions of histiocytosis X may occur in any bone; however, they were more common in the skull, facial bones, vertebrae, pelvis, ribs, and long bones.[18,19] Within the jaws, the posterior parts of the mandible were most commonly affected.[6,16,19] Extraskeletal lesions were reported in skin, lymph nodes, liver, spleen, lungs, gingiva, and hypothalamus.[4,20,21] Clinically, the lesion may present without physical sign and symptom and may be found upon a roentgenographic examination of bones of the head or other areas. On the other hand, the patient may present with local pain, swelling, tenderness, gingivitis, loosening of the teeth, oral ulceration, poor healing of wounds, foul breath, abnormal taste or gingival hemorrhage.[5,7,14,18,21,22]

According to the study of Dagenais et al[23], it was found that the following radiographic characteristics were useful in the diagnosis of histiocytosis X of the jaws: 1) the appearance of round or oval solitary intraosseous lesions associated with periosteal new bone formation 2) the presence of multiple lesions of the alveolar process that had a well-defined periphery 3) a scooped-out effect in which a portion of the superior aspect of the crest of the alveolar bone was maintained at the mesial and distal margins of the area of destruction 4) sclerosis of bone and 5) mild root resorption. The periphery of the lesion of histiocytosis X in the jaws was considered to be well-defined but not corticated.[3,6,18,22,24] The entire lamina dura or portion of it was absent in case that affected the alveolar process. The amount of alveolar bone loss was variable, but frequently the destruction of the supporting alveolar bone was so severe enough to displace the teeth and, in some cases, made the teeth appear to be "floating in the air".[6,10,22,23,25]

Histologically, the features of the oral lesions were essentially the same as in lesions found elsewhere in the skeleton and in soft tissue.[6] Histiocytosis X was characterized by the proliferation of Langerhans cells. These Langerhans cells are large cells with abundant cytoplasm, indistinct cell borders, and oval to reniform nuclei which may contain conspicuous nucleoli. These Langerhans cells were most often arranged in sheets admixed with varying numbers of eosinophils and other inflammatory cells.[6,8,10,16] The ratio of Langerhans cell to inflammatory cell may vary from field to field within a single lesion, as well as from case to case. The demonstration of Langerhans cell was very crucial for the diagnosis of the diseases in the group of histiocytosis X.[9] When the lesion matured, fibrosis occurred and eosinophils became less numerous or may even disappeared.[14] Inflammation at the site, when severe enough, may mask the histologic features which were essential for the diagnosis, particularly in small intraosseous lesions or superficial gingival lesions.[6] Early lesions, when biopsied, may not be diagnostic. They may show only inflammatory granulation tissue suggestive of a bacterial infection or osteomyelitis.[5]

By histochemical method, Langerhans cell showed the presence of enzymatic activity of adenosine triphosphatase, non-specific esterase and acid phosphatase and the absence of enzymatic activity of lysozyme and α_1-antichymotrypsin.[4,26,27] Staining with antibody to S-100 protein was widely employed as an aid in differential diagnosis of histiocytosis X. The S-100 protein was readily identified by immunohistochemical means in Langerhans cell.[5,26-28] Rabkin et al[28] and Ree and Kadin[29] reported that staining of Langerhans cells with peanut agglutinin and with antibody to S-100 protein were of comparable sensitivity in the diagnosis of histiocytosis X in formaldehyde-fixed, paraffin-embedded tissue sections. Electron microscopic study showed the presence of peculiar, rod-like, lamellated structure with central periodic striation in the cytoplasm. This cytoplasmic inclusion ; the Birbeck granule, was the ultrastructural hallmark of Langerhans cell.[10,26,28]

Pathologic conditions exhibiting clinical and radiographic features similar to histiocytosis X were hyperparathyroidism, fibro-osseous lesions, benign odontogenic tumors, multiple myeloma, primary malignancies and metastatic lesions.[5,7,24,25]

Various treatment modalities were advocated for

the treatment of histiocytosis X. Surgery, radiotherapy, chemotherapy and local steroid injection were used alone or in combination.[15,16,20,22,24,30] The modality chosen depended on the location and extent of the disease and ranged from no treatment to treatment with one or the combination of methods.[3,17] To be beneficial, the treatment should either speed healing or reduce complications and its sequelae should not be more devastating than the disease itself.[3] Surgical curettage was recommended for the treatment of most accessible lesions.[5,6,19,25] Curettage should be as thorough as possible, but healing may occur even without complete removal of the lesion, as was evidenced by examples of spontaneous regression and response to biopsy in the literature.[3,4,30] Most authors did not recommend curettage for the treatment of large lesions in weight-bearing bones because of the risk of pathologic fracture.[15,19] Radiation therapy was useful in surgically inaccessible areas, in weight-bearing areas, and in areas where surgery would cause dysfunction or disfigurement.[19] Low-dosed radiation of 1500 rad or less was recommended.[5,6,25] Despite of low dose of radiation, there were still dangers, particularly in young patients, of growth alteration, injury to developing teeth, and the remote possibility of post-irradiation neoplasia. Treatment must be weighed against the benefits of this form of therapy.[2,6,15,25] Vinblastine sulfate was the chemotherapeutic drug of choice in the treatment of Letterer-Siwe disease and Hand-Schüller-Christian disease, however, chemotherapy was rarely used in the treatment of localized eosinophilic granuloma.[19] Vinblastine sulfate used alone or in combination with prednisone yielded good result.[20] Intralesional injection of steroid was reported with favorable result.[3]

A case report

A 22-year-old Thai male who presented with the chief complaint of gingival swelling and teeth loosening for 3 months was referred to the Oral and Maxillofacial Surgery Division, Dental Department, Khon Kaen Hospital. He first noticed this problem one year prior to this presentation and it was on and off. His weight was decreased during the last three months. He was evaluated and the incisional biopsy of the gingiva from anterior region of the mandible was performed by a local oral & maxillofacial surgeon. Chronic non-specific inflammation was reported and he was referred to Khon Kaen Hospital for further investigation and management. The patient had a history of a motorcycle accident and sustained a right tibial fracture and a head injury 4 years ago. He also noticed a high water intake and an increase in the volume and frequency of urination. On clinical examination, he generally looked weak. Facial asymmetry, hypoplasia of the right zygoma, flatness of the right infraorbital rim, and chin deviation to the left side were also observed (figure 1). No paresthesia of infraorbital region, upper and lower lips were noted. Bilateral submandibular lymph nodes were enlarged, palpable, movable, but not tender. All remaining teeth of the lower arch (#33, 34, 37, 38, 42, 43, 44, 47, 48) were floating in soft tissue (figure 2). Teeth #17 had third degree mobility. There was a pathological fracture at left body of the mandible. Orthopantomogram showed irregular radiolucent areas with well demarcated borders involving from right to left ramus and all remaining mandibular teeth were floating in these areas. Moreover, periapical radiolucent areas of #17, 18 and #22, 23, 24 were also observed (figure 3). CT scan and 3D CT showed that the remaining alveolar process of the mandible, the right alveolar process of the maxilla, the right zygoma, and the right middle cranial fossa were destroyed (figure 4). Upon clinical examination and radiographic appearance, the differential diagnoses were histiocytosis X and malignant tumor. Rebiopsy of soft tissue from anterior region of the mandible was performed and it was reported as histiocytosis X. The total bone scan showed abnormal uptake at mandible, maxilla, and right zygoma (figure 5). The laboratory investigation of complete blood count, urinalysis, electrolyte, and chest X-ray were all within normal limits. He was taken to the operating room for enucleation of the lesions from the mandible and right maxilla as well as extraction of teeth #17, 18, 33, 34, 37, 38, 42, 43, 44 under general anesthesia. The soft tissue specimen was submitted for histopathological examination. Microscopically, the lesion showed pieces of connective tissue covered discontinuously with parakeratinized stratified squamous epithelium that exhibited focal hyperplasia. The underlying connective tissue showed several aggregates of Langerhans cells mixed with eosinophils and other inflammatory cells (figure 6). Immunologically, these putative Langerhans cells were stained positively for S-100 protein (figure 7). One month after the surgery, he was referred to Srinakarin Hospital for post-operative radiation and internal medicine consultation and management of diabetes insipidus. He received 1,000 rads of radiation. In the two-month follow-up visit, the mandibular fracture was healed and the symptom of diabetes insipidus was improving. Six months post-operatively, there was no sign of recurrence.

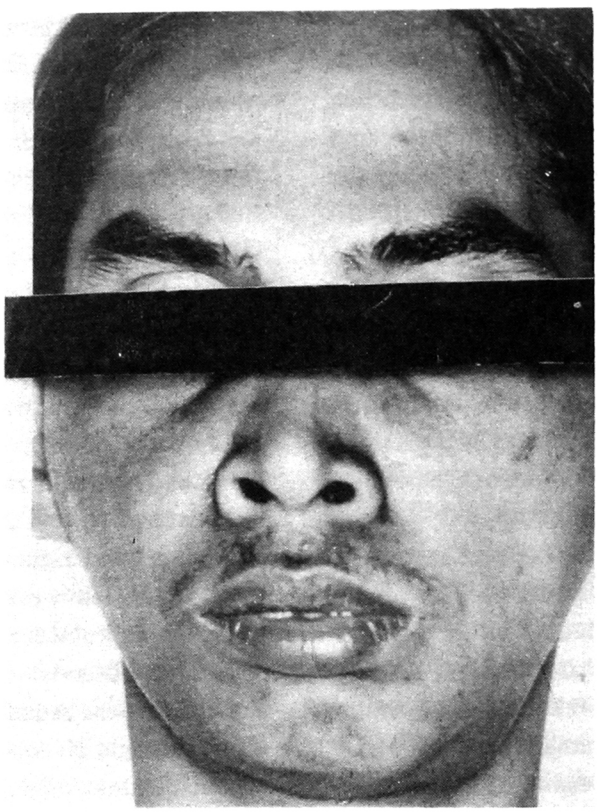

Figure 1 Photograph showing facial asymmetry and chin deviation to the left side.

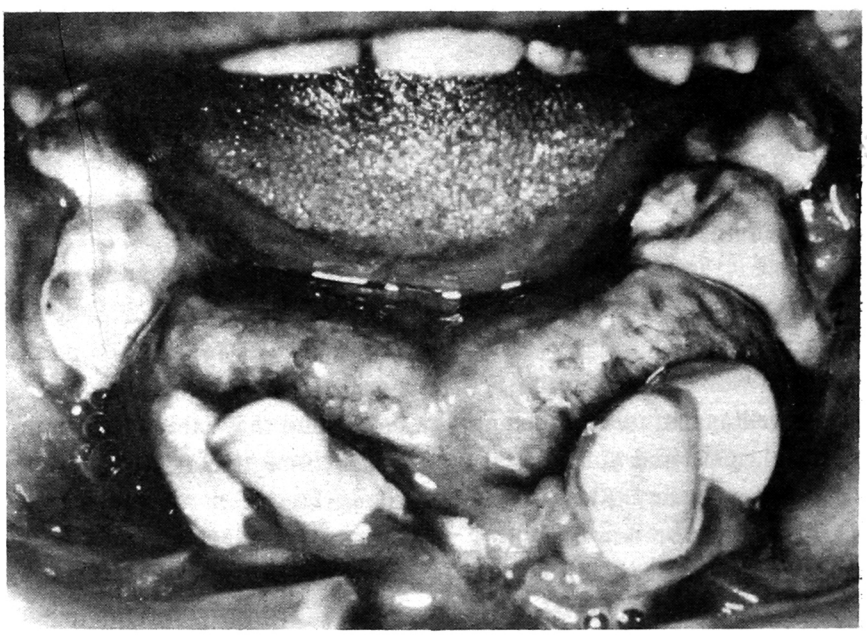

Figure 2 Photograph showing intraoral appearance of generalized gingival inflammation with heavy calculus deposit and all the teeth in the lower arch seemed to be floating in soft tissue.

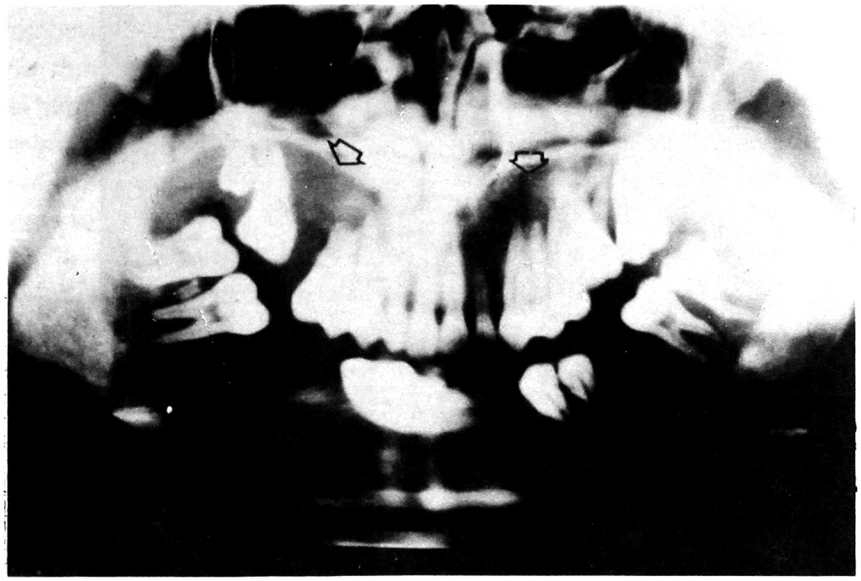

Figure 3 Orthopantomogram showing bone destruction extending from left to right body of the mandible, maxilla area#16 to right tuberosity and periapical area # 23-25 (arrows).

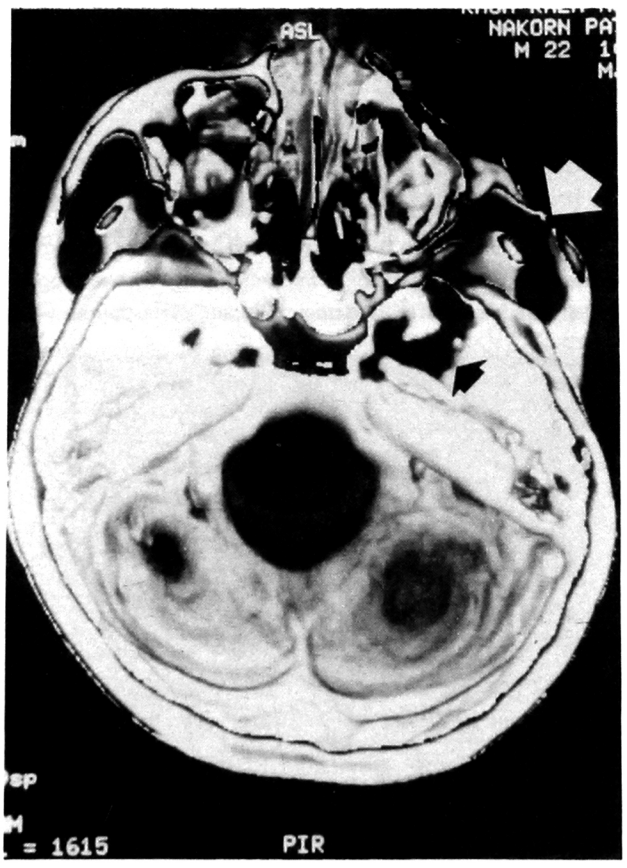

Figure 4 3D CT demonstrating the destruction of right middle cranial fossa (black arrow) and right zygoma (white arrow).

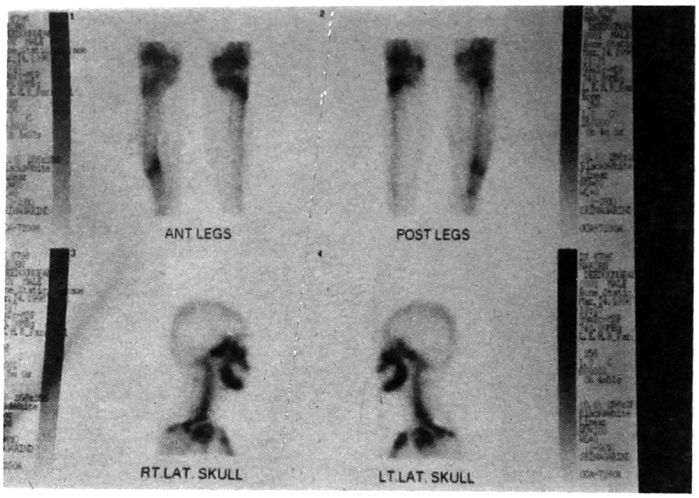

Figure 5 Bone scan demonstrating increased uptake at mandible, maxilla, and middle cranial fossa.

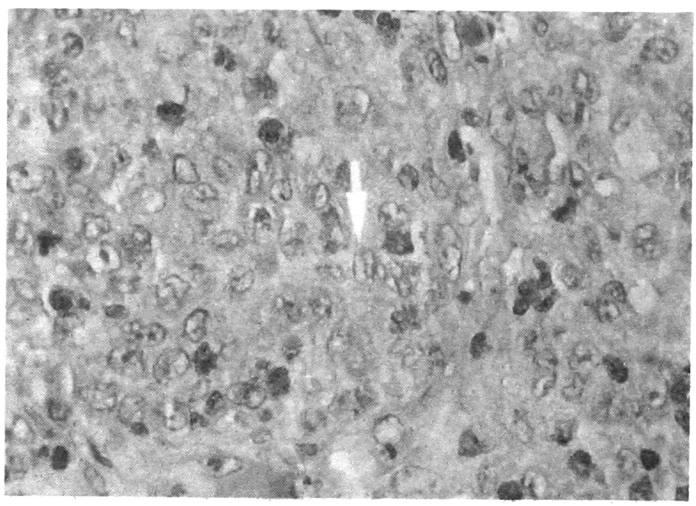

Figure 6 Photomicrograph showing sheet of Langerhans cells (arrow) with pale oval nuclei and prominent nucleoli admixed with eosinophils and other inflammatory cells. Hematoxylin and eosin stain. x 642.

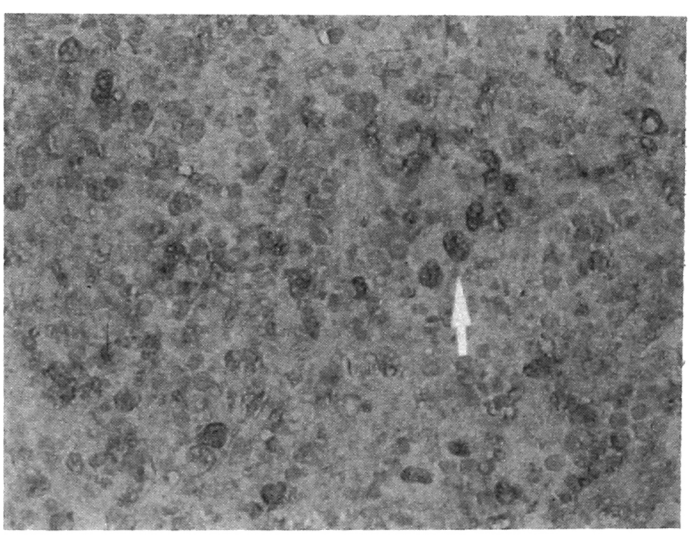

Figure 7 Photomicrograph showing positive S-100 staining of Langerhans cells (arrow). Immunoperoxidase stain for S-100 protein. x 642.

Discussion

The etiology of histiocytosis X remained unknown. Histiocytosis X was once thought to be a lipid metabolic disorder, but lipid-laden histiocytes were not an invariable finding in tissue examined. Furthermore, when lipid was present, it appeared as normal cholesterol.[25] Currently, most investigators agreed that this disease was a reactive process rather than a neoplasm.[4] DNA flow cytometric studies provided no evidence of aneuploidy.[31] An inflammatory etiology was favored by some investigators because of microscopic features, the clinical course, and the response of the disease to conservative therapy in most instances.[6] A bacteriologic origin was suggested, but the possibility seemed unlikely, since numerous attempts to isolate and culture specific microorganism were not successful.[6,18] Some authors suggested that this disease may result from an exuberant reaction to an unknown antigenic challenge.[10] Some patients exhibited deficits in certain aspects of the cell-mediated arm of the immune system. A deficiency of suppressor T lymphocytes, the low level of serum thymic factor, the response to thymic extract, and the abnormalities in the thymus biopsies suggested the thymus abnormality in this disease. These immunologic defects may affect normal regulatory mechanism with resultant Langerhans cell proliferation.[10,32] All of these factors would suggest an immunologic basis for the disease. It was also interesting to speculate that the broad spectrum of disease of histiocytosis X was due to the degree of the immunologic defect and quantity and location of antigen.[33]

When the lesions involved the jaws, the differential diagnosis should include lesions commonly observed in the jaws such as odontogenic cyst, periapical infection, and periodontal disease.[21,22] The clinical features of the cyst were usually diagnostic and could be confirmed by biopsy. The fact that the teeth in the involved areas responsed to vitality test should help to eliminate periapical lesions arising from pulpal necrosis such as periapical granuloma or cyst. The extent of the lesions of histiocytosis X was usually more widespread than in cases of common marginal periodontitis and the periodontal disease was rare among young-aged group unless associated with systemic conditions such as diabetes mellitus, hyperparathyroidism or scleroderma.[5] The other point was that the scoop-out form of bone destruction was not observed in periodontal disease.[23] All of these may be useful in the differential diagnosis of histiocytosis X from periodontal disease. Primary malignant tumors of the jaws were rare and some had certain characteristis to differentiate them from histiocytosis X. For example, multiple myeloma usually occurred in the middle-aged group or above and demonstrated the presence of Bence-Jones protein. Metastatic neoplasms could be eliminated in the absence of pain or paresthesia and in the radiographic evidence of periosteal new bone formation.[22] Important features which should suggest the possibility of histiocytosis X were 1) delayed healing of previous extraction site 2) severe localized periodontal involvement in an otherwise healthy dentition 3) recurrent mandibular swelling associated with halitosis, loose teeth, and dental infection 4) premature loss of deciduous teeth 5) early eruption of the permanent dentition, often in a position of malocclusion and 6) young-aged group.[25] The clinical data in age, gender and location of the lesions in this case correlated quite well with the previously reported ones. The patient in this case was male as in the majority of the previously reported cases. The lesions involved the jaw bones and the skull which were the common sites of histiocytosis X. The roentgenogram in this case demonstrated multiple bone involvements including the severe generalized bone loss with teeth appearing to be "floating in the air" in a young adult. This type of roentgenogram should alert the clinician to think of diseases other than periodontitis. Surgical exploration and pathological examination were essential in establishing the definite diagnosis.[22] In our case, the lesions showed the proliferation of Langerhans cells interspersed with eosinophils and other inflammatory cells. The presence of Langerhans cells was confirmed by the use of immunohistochemical staining with antibody to S-100 protein. The overall histologic features of this case was consistent with the diagnosis of the diseases in the group of histiocytosis X. When the multiple bony involvements including the skull lesion and the symptom of diabetes insipidus were taken into account, it was likely that the diagnosis of histiocytosis X of the Hand-Scüller-Christian disease subtype was made. If the diagnosis of histiocytosis X is rendered, it is essential that the whole body skeletal scan be performed to search for additional asymptomatic lesions which may be present in other bones. There were a number of treatment modalities to choose from. Most jaw lesions were treated by surgery[6] as we did with our case. In addition, the inaccessible areas were treated by low dose of radiation. The combined surgery and radiation therapy gave satisfactory result in this case. The patient would be followed closely to determine

whether the radiation therapy was effective in controlling the diabetes insipidus. Not all the teeth involved by the disease needed to be sacrified.[6] However, we had to extract numerous teeth because of lack of bone support and periapical pathoses.

Conclusion

Histiocytosis X is a group of diseases with varied clinical manifestations. Oral lesions may be the initial sign or, in many cases, the only manifestation of the disease, but oral lesions of histiocytosis X may mimic the manifestation of other disease. Only through thorough medical history, clinical examination, radiographic examination and, above all, the biopsy of the lesion that the accurate diagnosis could be made. The sooner the correct diagnosis is reached and the appropriate treatment instituted, the better the prognosis will be.

A case of histiocytosis X affecting the mandible, maxilla, zygoma and middle cranial fossa in a 22-year-old man was presented. The lesion showed the eosinophils scattered among the sheet of Langerhans cells which were considered the histological hallmark of the diseases in the group of histiocytosis X. The patient was treated by enucleation and extraction of the teeth followed by low dose of radiation and the result of the treatment was quite satisfactory.

Histiocytosis X: Review literature and a case report

Abstract

Histiocytosis X encompasses three clinical entities: eosinophilic granuloma, Hand-Schüller-Christian disease, and Letterer-Siwe disease. It usually occurs in young-aged group and is characterized by the proliferation of Langerhans cells and a collection of eosinophils Radiographically, it appears as single or multiple areas of well-defined radiolucencies or the appearance of teeth floating in the air in severe cases. Because of its varied oral manifestations which can result in potential diagnostic problem, the importance of biopsy, especially the demonstration of Langerhans cells, is stressed. Surgery is the preferred treatment for most of the jaws lesions while radiotherapy and chemotherapy should be reserved for lesions which are either too numerous or inaccessible to surgery. A case of histiocytosis X in a 22-year-old man was presented. The patient had a chief complaint of gingival swelling and teeth loosening. Bilateral submandibular lymph nodes were enlarged. Radiographs showed multiple bone involvement. The biopsy revealed aggregates of Langerhans cells mixed with eosinophils. Langerhans cells were stained positively for S-100 protein. The treatment of this case was accomplished by the combined surgery and radiotherapy.

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Keywords: histiocytosis-X, Langerhans cell, biopsy

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