Introduction:
A fracture is complete or incomplete discontinuity of bone caused by direct or indirect force. A pathologic fracture is one that occurs even with a low impact trauma due to weakened bony architecture from a preexisting pathological lesion [5]. Pathologic fracture may be acute which can be diagnosed from various signs and symptoms. It may even be a chronic silent affair due to masking of signs from preexisting pathology. Mandibular fractures are most frequently occurring injuries with 2% of such fractures. Dentists should therefore have a thorough knowledge of its etiology, for immediate assessment and management.

Mandibular pathologic fractures besides causing severe pain and restriction in function, can lead to secondary infection and malunion if not adequately treated. Therefore, when a fracture is noticed, the first assessment should be to know if the bone in involved was normal, normal but inadequate or abnormal. Treatment of pathologic fractures is complicated because it involves reduction and stabilization of bone already compromised from underlying pathology. It is also difficult because when bone pathology is a generalized systemic condition, it may restrict the choice of treatments available.

Causes of bone weakness leading to mandibular pathologic fractures are:
1. Inadequate or abnormal bone formation
2. Resorption of internal bone mass weakening the bony architecture
3. Reduction of bone quality decreasing ability to bear the stress of function
4. Pathologic bone remodeling
5. Local bone destruction due to cysts and tumors (primary and metastatic) thus reducing the total amount of bone available to bear the load of mastication.

Signs and symptoms of pathologic mandibular fractures:
Any pathologic fracture will present with signs of fracture and of the underlying pathologic process. These include: pain, tenderness, clicking, deviation of jaw, numbness of lower lip and chin area, difficulty in chewing, exophytic lesion, buccolingual swelling and obliteration of vestibule, mobility of jaw fragments, open bite, malocclusion and ecchymosis.

Diagnosing pathologic fractures:
Investigations need to be first carried out to locate the fracture and its extent. Radiographs like orthopantomogram and postero-anterior view may show destructive radiolucent lesion on the mandible, with pathologic fracture through the inferior border. Overriding of the edges of the inferior border of the mandible may be noted. Computed tomography (CT) or Cone beam computed tomography (CBCT) scan to know the complete extent of fracture and obtain some idea of the underlying pathology may be carried out. Magnetic resonance imaging (MRI) is very sensitive to early marrow changes. It can help locate metastases prior to the appearance on radiographs and CT, but is not as helpful for bony anatomy.

Identifying the pathologic process is performed by incisional biopsy of suspicious lesion at the fractured site. On suspecting metastatic lesion, work-up including contrast enhanced CT of the head and neck with evaluation of lymph nodes of the neck is required. Chest X-ray, abdominal ultrasounds are also included in the investigations.

Laboratory investigation includes a complete blood count (CBC) when working up any suspected malignancy. Erythrocyte sedimentation rates (ESR) and C-reactive protein (CRP) levels that signal an inflammatory process involvement should be obtained. Various tumor marker levels will also need to be measured for specific malignancies. Thyroid panel to help eliminate the suspicion of thyroid malignancy is needed. Kidney function tests should also be performed. Total body radionuclide bone scan is useful in searching for other skeletal sites of tumor involvement.

There are a number of conditions that could make the mandible susceptible to fracture. These are listed in the table below (Table 1) [2].

Osteogenesis imperfecta
This is a dominantly or recessively inherited disorder of connective tissue characterized by bone fragility [7]. The osteoblasts form an abnormal matrix which fractures under minor mechanical loads. Patients with osteogenesis imperfecta frequently suffer fractures, either caused by relatively mild trauma or occurring
spontaneously. Fracture is noted in over 80% of the extremities as they face more mechanical loads [7]. Facial bone fracture occurs rarely but cases have been reported. Gallego et al reported mandibular fracture during atraumatic molar extraction in a patient with osteogenesis imperfecta [7]. Such fractures can be managed with open reduction with plates and maxillomandibular fixation. Physiotherapy, rehabilitation and orthopaedic surgery is important in osteogenesis imperfecta [7]. The healed bone may be of inferior quality so more care needs to be observed even after treatment.

**Mandibular atrophy:**

With increasing mandibular atrophy, the physical size of the mandible decreases (Figure 1). According to Luhr’s classification, a fracture in the mandible with a width of less than 15-20 mm is considered to be an atrophic mandibular fracture [13]. A severely atrophic mandible may fracture due to minor trauma or even during mastication. These fractures constitute about less than 1% of the total facial fractures and often occur bilaterally involving the mandibular body [15]. The patient may exhibit extraoral and intraoral ecchymosis associated with fracture with pain and mobility of anterior mandible.

Treating atrophic edentulous mandibular fracture is challenging due to a number of reasons. These fractures usually occur in the geriatric population who may be medically compromised to undergo surgical procedures. Also the bone regenerating process is decreased in the aged population. Due to severe atrophy there is insufficient and poor quality bone for fracture healing. The blood supply to this bone will be more periosteal and less of endosteal thus causing further healing delay and also there are no teeth present for good reduction of fractures.

Treatment of these fractures is planned based on patients health and blood supply. If health is severely compromised, closed reduction and liquid diet would be an option. But open reduction and rigid internal fixation is able to deliver good results with possibility for immediate functional rehabilitation. When open reduction is performed, intraoral approach will eliminate chances of scarring and facial nerve injury. Extraoral approach if taken will provide better visualization of fragments, less periosteal stripping and less chances of infection from intraoral environment. Locking bone plate system can be used which are believed to prevent disruption of underlying periosteal vascular supply. Also in the locking plate system it is not necessary for the plate to intimately contact bone at all sites. They can be applied on the inferior border of mandible thus avoiding need for removal in denture construction. Less rigid options like miniplates may not be a very good option in atrophic fractures as in spite of low masticatory loads in the edentulous mandible since it is still subjected to forces from other directions [15].

**Impacted third molar:**

Pathologic mandibular fracture from impacted third molar removal is a rare complication [23]. Deeply impacted molars usually require massive bone removal that might weaken the mandible and predispose to fracture. Bone at the site of impaction may also be weakened due to inflammatory process like pericoronitis or abscess.

Pathological mandibular fracture associated with the removal of teeth can occur either during the procedure or in the immediate postoperative weeks. Several risk factors can be associated with this type of pathological fracture, such as age, gender, types of impaction, existing infection and surgical technique. Ankylosis of the impacted tooth among older patients may also complicate removal, as more extensive bone removal may be needed [4]. Sectioning of the tooth is highly recommended to reduce the amount of bone removed. Patient should also observe extreme care following third molar extraction to not bite on hard foodstuff post extraction. If fracture occurs, treatment includes closed or open reduction or no treatment with soft diet depending upon amount of bone displacement.

**Osteoporosis:**

Osteoporosis is an inherited disease of bone in which there is failure of normal osteoclastic resorption. Although osteoclasts fail to resorb bone, osteoblasts exhibit normal function. The imbalance between osteoblastic apposition of bone and osteoclastic resorption leads to increasing bone density throughout the skeleton [1].

The affected bone fractures more easily, probably because trabeculae are not properly aligned along the planes that buttress the bone against stress. Another complication in osteoporosis is osteomyelitis which can further weaken the bone and make it susceptible to fracture. Osteomyelitis in such patients may not heal well therefore patients with osteoporosis should maintain good oral hygiene [1].

**Cysts and Benign Tumors:**

Many cystic lesions when extensive can lead to pathological mandibular fractures such as aneurismal bone cysts, follicular cysts, radicular cysts, and odontogenic keratocyst [11]. Mandibular angle and body are the most frequently observed locations of fractures associated with benign cysts. Cystic lesions or tumors grow in size causing expansion thinning and resorption of surrounding bone thus weakening it and making it more prone to fracture. Management will involve curettage of the cysts or resection of tumor and fixation of the fractured segments. In case of insufficient healthy bone after surgery, secondary reconstruction is required [10].

**Primary and secondary malignant lesions:**

Primary and metastatic tumors seen in the mandible can also cause fracture. These include lesions like angiosarcoma, metastatic lesions of follicular carcinoma of thyroid and oesophageal carcinoma [2, 12,18]. Metastatic lesions in the jaw may present with pain, swelling, mobility of teeth, pathologic fractures and paresthesia. Radiographically, metastatic lesions are most often ill defined and are usually radiolucent but may be radiopaque mixed lesions [2, 18]. Figure 2 shows evidence of pathologic fracture in an edentulous mandible of a female affected by squamous cell carcinoma.

The treatment of pathological mandibular fractures associated with metastatic lesions should be to tackle the metastatic process on priority. Locating the primary malignancy is of utmost importance. Resection of the lesion and immobilization of the jaw should be carried out. Treatment is often limited by the patients’ general health, as a pathological fracture may represent an advanced stage of neoplastic disease. Radical surgery for resectable tumors with mandibular reconstruction should be
carried out. This may be followed by postoperative radiation or chemotherapy and radiation therapy depending on the final pathology.

**Multiple Myeloma:**
This is a plasma cell neoplasm characterized by plasmacytosis within the bone marrow leading to osseous destruction, renal insufficiency and anemia. Bone loss is due to diffuse osteopenia, focal osteolytic lesions, pathologic fractures and hypercalcemia. Fractures occur in approximately 30 - 40% of patients with multiple myeloma. These fractures may result from the direct deposits of myeloma cells within the bone or from bone resorption due to tumor releasing factors like osteoclast activation factors and factors causing osteoblast suppression. Malignant plasma cells are also able to induce osteoblast apoptosis. [21]

The most common site of fractures is in the spine (55%–70%) especially in the lower thoracic or lumber vertabral bodies. Vogel et al have studied pathologic fractures in patients with multiple myeloma who were on bisphosphonate therapy and found that they occur independently of myeloma activity. These fractures therefore should not be considered a sign of disease progression [22].

**Leukaemia**
Leukemia is proliferation of clone of abnormal hematopoetic cells. The leukemic cells multiply at the expense of normal hematopoetic cell lines. Bones involved in leukaemia may be femur, humerus, pelvis, skull, metacarpals, ulna, and the vertebral.

Radiographic appearance of leukaemic bone involvement has been described as a ‘moth eaten’. The presence of a pathological fracture though rare may be the first sign of an ongoing disease process or indeed may be the initial presenting sign which may be confirmed by haematological investigation [25]. The treatment of fracture in leukemia is complicated by the presence of the ongoing disease and involvement of other facial bones as well as long bones.

**Osteomyelitis:**
Osteomyelitis of the mandible may develop if a primary infection is not eliminated by proper treatment or if concurrent immunodeficiencies are present. Several predisposing diseases may be associated with osteomyelitis, such as diabetes, osteogenesis imperfecta, squamous cell carcinoma and pyknodysostosis. Osteomyelitis of mandible may be so widespread that it leads to fracture of mandible. Figure 3 shows clinical image and orthopantomograph of a male patient with osteomyelitis after extraction of multiple posterior teeth from right mandible which followed with pathologic fracture.

Management of osteomyelitic fracture involves intravenous antibiotics followed by procedures like sequestrectomy. Once the infection and infective focus is eliminated, the amount of remaining healthy bone has to be evaluated. Closed reduction with intermaxillary fixation should be considered as the ideal treatment in pathological fractures associated with osteomyelitis to avoid further ischemic necrosis by plate placement [1,16].

**Osteoradionecrosis**
Radiation is a double edged sword that can both heal and harm tissues. Post radiation osteoradionecrosis (PRON) has been defined as necrosis (devitalization and devascularization) of bone that may occur in association with radiotherapy for cancer in the absence of recurrent or metastatic disease [6].

Osteoradionecrosis will destroy the bony framework at the site thus making it susceptible to injury which are difficult to heal due to compromised vasculature.

Radiotherapy to primary tumors of tongue and oropharynx will lead to greater radiation exposure to bone in relation that is mandibular jaw. Patients may not present with actual signs of osteoradionecrosis until the fracture occurs as noted in Figure 4.

Prevention of osteoradionecrosis by maintaining good oral hygiene before during and after radiation therapy is a must. If extraction is required after 4 months or radiation treatment then hyperbaric oxygen (HBO) therapy should be considered. If necrosis sets in then treatment with HBO, sequestrectomy, free bone replacement, antibiotics and antioxidants should be considered. Osteoradionecrosis when associated with pathologic fracture is more complicated to treat. These patients may present with comorbidities and nutritional problems because of previous surgery and/or radiotherapy. First of all, surgeons should deal with the patient’s systemic problems, ruling out recurrent cancer and nutritional issues before managing the fracture [5]. Surgical margins are normally determined by resecting pathologic appearing bone until vascularized bone that bleeds readily is obtained. After resection of all diseased bone, the patient should undergo 20 HBO dives. Free tissue reconstruction should then be performed, followed by a further 10 HBO dives [19].

**Renal osteodystrophy**
Renal osteodystrophy is the term applied to all pathologic features of bone in patients with renal failure. The abnormal kidney function results in hyperphosphatemia, and hypocalcemia, resulting in secondary hyperparathyroidism.

The findings of renal osteodystrophy diagnosed with conventional radiography include osseous resorption, soft-tissue calcification, osteopenia, amyloid deposition, and fracture. The most common complication of renal osteodystrophy is fracture, which may be insufficiency fractures through osteomalacic bone or pathologic fractures through brown tumors or amyloid deposits [14].

**Paget disease**
In pagets disease osteoclastic resorption of normal bone is accelerated initially. New abnormal bone is formed by increased osteoblastic activity. Later on osteoclastic resorption wanes and osteoblastic apposition predominates. It is a chronic disease which in its worst form causes severe pain [24]. Weight bearing bones break easily or may slowly bend under pressure to produce crippling, bowing deformities and compression of spinal nevus. If the base of the skull is affected, narrowing of cranial foramina may cause deafness and blindness. Facial bones, jaws, ribs and those bones distal to the elbow and knee are far less often involved. Fracture in Pagets disease is managed by first taking care of pain. Reduction of fractured segment is then performed. Subcutaneous injections of calcitonin reduces the rate of osteoclastic resorption.

**Implant placement**
If the technique and planning is correct then placement of implant at smaller edentulous areas will not cause fracture. If the edentulous span is long then there may be severe alveolar bone resorption thus making the jaw weak. Fracture during implant placement is most likely to occur in jaws with anterior mandibular bone lower than 12 mm. This occurs due to unfavourable mechanics.

Fracture may also occur due to implant failure and osteomyelitis[16]. Marginal bone loss around a dental implant may be an area of weakness. Therefore assessing height and width of available bone prior to implant placement and using bone graft whenever necessary can help avoid this mishap [5].
In case of fracture due to implant failure removal of the failed implant, antibiotic therapy, debridement and fracture reduction is the favored procedure [16].

Gorham’s disease (vanishing bone disease)
Massive osteolysis is a condition characterized by spontaneous, resorption of bones. It is an idiopathic, nonmalignant, destructive, proliferation of angiomatous tissue with replacement by fibrous tissue. Massive osteolysis usually appears in children or young adults and the mandible is the second most common location after the arm. Usually no palpable mass or tenderness is present in the area, but depression may be seen. Radiographically, small radiolucencies resembling osteoporosis may be noted initially and then followed by disappearance of a portion of the bone or a regional group of bones [9].

Cases in the past have been managed with surgery and radiotherapy followed by reconstruction. If significant amount of bone mandible destruction has occurred, erich arch bar across the dentition is placed to stabilize the mandible. Any progression in disease can yield loose hardware and likely infection. Conservative treatment provides reasonable outcomes for fractures related to this disease [17].

Kinky Hair Syndrome:
Timothy et al have reported mandibular fracture in a toddler with kinky hair syndrome. This syndrome is a rare X-linked disorder which affects copper absorption and metabolism. This is a neurodegenerative disorder which is also characterized by generalized osteoporosis and kinky hair due to connective tissue abnormalities. In case of fractures in this condition, healing is poor. Therapy with copper replacement provides some relief [3].

CONCLUSION:
Management of pathologic fracture involves two aspects, management of pathology and management of fracture. If the underlying pathology is a generalized systemic condition then fracture reduction will be done prior to complete treatment of the systemic cause. But if the pathology is a localized bone disease then elimination of the underlying pathology and fracture management will be performed simultaneously. A priority has to be given to infection control. Regular follow ups to evaluate healing process at the fracture reduction site and recurrence is a must.

Figure 1: Radiographic views of edentulous mandibular jaws showing complete alveolar ridge resorption

Figure 2: Intraoral view and orthopantomograph showing pathologic fracture in a mandible weakened with underlying carcinoma.

Figure 3: Clinical images and radiographs showing pathologic fracture in mandible affected by osteomyelitis post extraction.
**Table:**

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