OLGU SUNUMU CASE REPORT

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Treatment Strategy for Pathological Fractures of the Mandible: A Report of Nine Cases and a Review of the Literature

Mandibular Patolojik Kırıklarda Tedavi Stratejileri: Dokuz Vaka ve Literatür Derlemesi

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ABSTRACT Pathological fractures of mandible are uncommon and challenging due to several complicating factors. The aim of the study is to report our experience concerning treatment strategies and difficulties in handling mandible with pathological fracture. Data concerning this condition in the literature has been analysed. This study reviewed retrospectively the records of patients who presented with a pathological fracture of the mandible. The collected data included age, sex, mechanism of injury, aetiology, anatomic site of fracture, treatment and complications. There were 7 patients with pathological fracture (mean age of 60.1 years) and two patients with atrophic fracture. Aetiologic factors of pathological mandibular fractures were osteoradionecrosis, bisphophonates osteonecrosis and osteomyelitis. All cases were treated with surgical approach. Some complications such as plate expose and infection have developed. The most frequent primary treatment utilized was resection of diseased part of bone and fixation with reconstruction plates. Complications occur more often because of local and general conditions of patients.

Keywords: Mandible; pathological fracture; osteoradionecrosis; bisphophonates osteonecrosis of the jaw

ÖZET Mandibulada patolojik kırıklar sık görülmemekle birlikte komplike faktörler eşlik ettiğinden tedavileri zordur. Bu çalışmanın amacı, patolojik kırığın görüldüğü mandibulalarda karşılaşılan zorluklar ve ilgili tedavi yöntemleri konusunda edindiğimiz tecrübeleri rapor etmektir. Bu klinik durum ile ilgili literatürde analiz edilmiştir. Mandibulada patolojik kırık ile başvuran olguların kayıtları retrospektif olarak toplanmıştır. Yaş, cinsiyet, kırığa sebep olan travma, patolojik kırığın etiyolojisi, kırığın lokalizasyonu, tedavi ve komplikasyonlar ile ilgili veriler toplanmıştır. Patolojik kırığı olan 7 hasta (yaş ortalaması: 60,1) ve atrofik kırığı olan 2 hasta değerlendirilmiştir. Osteoradyonekroz, bifosfonat nekrozu ve osteomyelit patolojik mandibula kırıklarına neden olan etiyolojik faktörlerdir. Bütün olgularda cerrahi yaklaşım uygulanmıştır. Plak ekspozu ve enfeksiyon komplikasyonları geliştiği rapor edilmiştir. Nekroze kemiğin rezeksiyonu ve rekonstrüksiyon plağı ile segmentlerin stabilizasyonu en yaygın uygulanan tedavi şeklidir. Hastalarda genel durumun bozuk olması ve lokal faktörlerin de olumsuz olması patolojik mandibula kırıklarında komplikasyon oranının daha fazla olmasına neden olmaktadır.

Anahtar Kelimeler: Mandibula; patolojik kırık; osteoradyonekroz; çenenin bifosfonat ile ilişkili nekrozu

Pathological mandibular fractures are rare, accounting for fewer than 2% of all fractures of the mandible.[1] Definition of a pathological fracture is contraversial. Pathological fracture is a fracture that results from normal function or minimal trauma in a bone weakened by pathology. However this definition contradicts with pathological fractures due to atrophy in elder people, since atrophy may not be regarded as pathology. Carlsen and Marcussen grouped their patients' spontaneous pathological frac-

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tures and spontaneous non-pathological fractures that include fractures of atrophic mandibles or iatrogenic fractures to deal with the controversy in definition.[2]

The treatment strategy differs and depends upon causative bone pathology and the patient´s general health including co-morbid diseases.[1-4] There are few case series of pathological fractures of mandible that discuss treatment strategies and aetiologic factors.[3-5] Abir et al. have reported ten cases of pathological mandible fractures and treatment outcomes.[5] Carlsen and Marcussen reported 25 patients presented with 25 spontaneous fractures: 17 pathological and 8 non-pathological spontaneous fractures.[2]

The aim of this study is to evaluate the treatment protocols of pathological mandibular fractures based on etiology and patients' general status and management of complications. We have reviewed the literature about the pathological mandibular fractures.

METHODS

A retrospective review of cases treated in the Departments of Oral and Maxillofacial Surgery and Otorhinolarengology at the University of Selçuk from 2012 to 2015 was undertaken. Collected data included age, gender, aetiology, site, management and outcome. Nine cases (1 female, 8 male) age range between 46 and 77 years were evaluated. Seven out of nine cases were diagnosed with pathological fractures. Mean age was 60.1 for cases of pathological fracture. However 2 out of nine cases developed fractures secondary to severe atropy which is a debatable aetiologic factor for pathological fracture as mentioned in introduction section. Therefore these two cases were categorized as atrophic fractures instead of pathological fracture. This study is compliant with Declaration of Helsinki protocols and granted exemption of institutional review board due to retrospective nature of the study. Written informed consent was obtained from each patient.

A computerized literature search was conducted for published papers using Medline and MeSH term "pathological fracture" in combination with "mandible". Articles presenting cases and populations of patients with pathological mandibular fractures were identified. Only articles in English language were considered. Patients' data including: sex, age, aetiology, fracture site, treatment and the presence of postoperative complications were analysed and compared with previously published data.

CASE SERIES

Aetiologic factors of pathological mandibular fractures were osteoradionecrosis (ORN), bisphophonates related osteonecrosis of jaws (BRONJ) and osteomyelitis (OM). The most frequent treatment utilized was mandibular resection of diseased bone and fixation with reconstruction plates without further reconstructive surgeries such as grafting (Table 1). Tooth extraction had triggerred necrosis of jaws in 4 patients.

Mechanism of injury was minimal trauma such as mouth opening exercise, hit by child or normal function like chewing (Table 1). The patients with ORN had been sent to hyperbaric oxygen unit following diagnosis of ORN before the pathological fracture had developed.

Seven patients with ORN, BRONJ and OM where there was no expectation for normal union of fracture lines were treated with excision until normal bleeding bone. OM and osteonecrosis cases were confirmed histopathologically. The continuity defects of mandibles were bridged with reconstruction plates. Reconstruction plates were performed with minimal 3-4 bicortical screws placed on either side of the resection (Table 1). Surgical reconstructions were performed transservically in all cases under general anesthesia (Figure 1). Two patients with significant soft tissue deficits after resection of diseased bone had regional pediculed flap advancements and rotations (sternocleidomastoid flap) to close deficit simultaneously. During follow-up, the intra-oral fistulas disappeared. One patient who had fracture in condyle while chewing secondary to ORN was treated with condylectomy via preauricular insicion (Figure 2).

Two patients who had fracture caused by severe atropy were treated with open reduction and fixation with reconstruction plate without any complications (Figure 3). These two patients were classified as atrophic fractures rather than pathological fractures (Table 2).

TABLE 1: Demographic, clinical data, aetiology and treatment referring to the group of pathological fracture cases.

Patient number	Gender	Age	Region	Aetiology	Injury related with fracture	Treatment	Follow up	Complication
1	M	54	Corpus	ORN	Mouth opening exercise	Resection and reconstruction plate	-	Pneumothorax related with anesthesia and exitus
2	M	59	Condyle	ORN	Hit by child	condylectomy	2.5 years	-
3	M	46	Angulus	ORN	Unaware	Resection and reconstruction plate	2 years	Plate expose
4	F	77	Corpus	BON (Aledronic acid)	Chewing	Resection and reconstruction plate	4 years	Loosening of plate and plate expose
5	M	61	Corpus	BON (Zoledronic acid)	Unaware	Resection and reconstruction plate	3 years	-
6	M	63	Corpus	OM	Chewing	Resection and reconstruction plate	3 years	Hardware (miniplate) fracture and infection
7	M	61	Angulus	OM	Unaware	Resection and reconstruction plates	1 years	-

ORN: Osteoradionecrosis; BRONJ: Bisphophonates related osteonecrosis of jaws; OM: Osteomyelitis; BON: Bisphosphonate osteonecrosis.

FIGURE 1: A 77 years old female patient with pathological fracture at corpus region of mandible secondary to bisphophonates osteonecrosis. Axial and coronal computed tomography shows fracture line (a and b) unstable fracture line and nearly exposed labile reconstruction plate following resection operation (c) although unstable fracture line oral mucosal healing could beobtained with sternocleidomastoid muscle flap (d) unstable plate and soft tissue between plate and bone were removed and longer reconstruction plates bended according to shape of mandible (e and f) panoramic radiographs following secondary operation (g) extraoral soft tissue healing and stable mandible could be obtained (h).

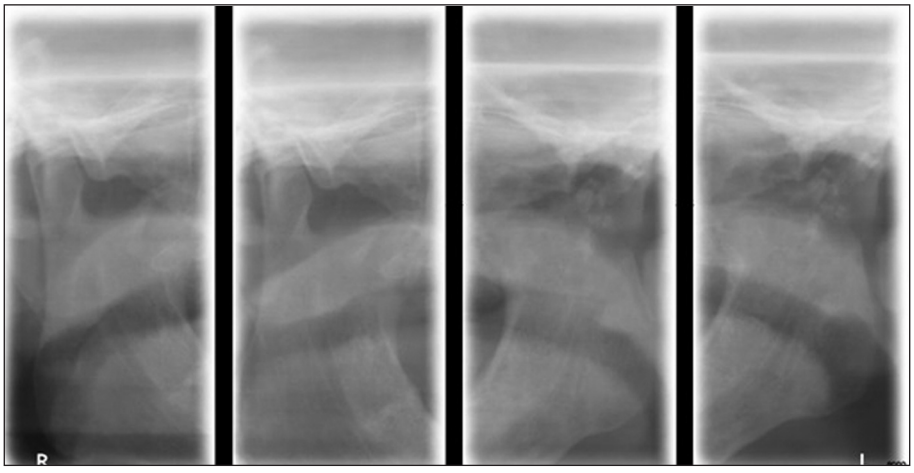

FIGURE 2: Radiograph of temporomandibular joint shows lytic apperance of left condyle one month after fracture.

Plate related complications were experienced for three patients with ORN, BRONJ and OM. Plate expose through scarred soft tissues was observed in a patient who had received radiation therapy (Figure 4). Tongue flap was used to cover oral soft tissue deficit and free fibular flap was performed secondarily for the patient who had pathological fracture caused by ORN. However partial flap loss has been developed.

Loosening of hardware (material used for osteosynthesis) happened in a patient with BRONJ in postoperative six months period. Loosened hardware had almost perforated the skin and caused pain while chewing due to mobility of segments. Reconstruction plate with more holes that cover till contralateral parasymphisial region was exchanged for the patient with BRONJ related pathological fracture (Figure 1). Instability related with miniplate fracture and purulent infection was seen in a patient with pathological fracture caused by OM, even though antibiotic therapy against causative microorganisms had been prescribed for three weeks before surgical treatment. Curettage and sequestretomy were applied to affected bone with two points miniplates fixation at first operation. Following plate fracture, infected bone was resected more widely, reconstruction plate was applied and long term antibiotic regimen was pre-

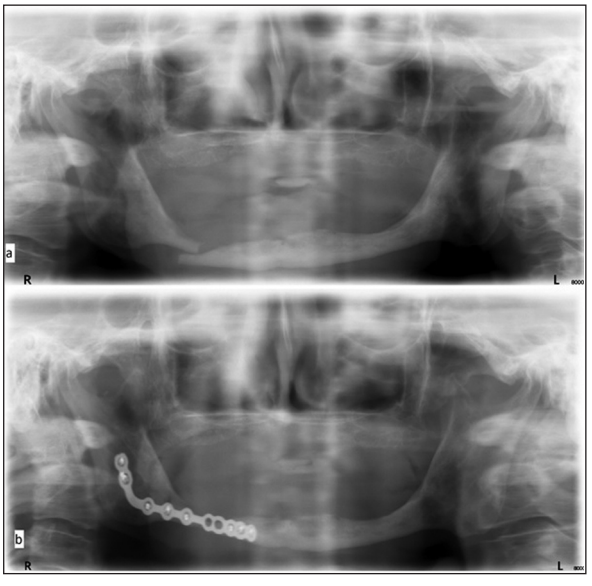

FIGURE 3: Preoperative (a) and postoperative (b) orthopantomogram of atrophic fracture of mandible.

TABLE 2: Demographic, clinical data, aetiology and treatment referring to the group of atrophic fracture cases.

Patient number	Gender	Age	Region	Aetiology	Injury related with fracture	Treatment	Follow up	Complication
1	M	69	Corpus	Atropy	While sleeping	Reconstruction plate	1 year	-
2	M	67	Corpus	Atrophy	chewing	Reconstruction plate	1.5 year	-

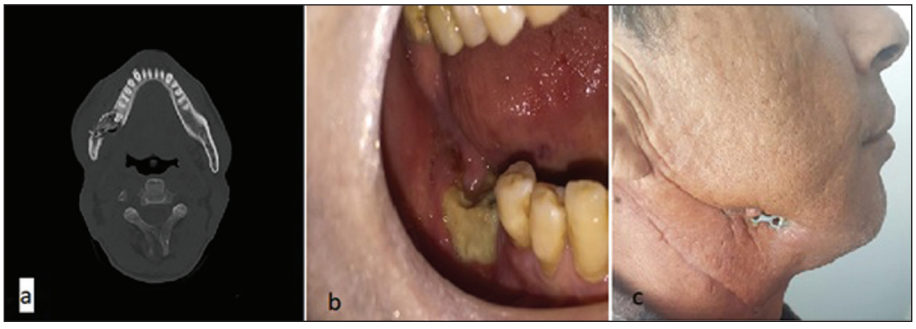

FIGURE 4: A 54 years old male patient with pathological fracture at angulus region of mandible secondary to osteoradionecrosis. Axial computed tomography shows irregular bone distruction and fracture line (a) İntraoral photo of necrotic bone (b) A complication of plate exposure following operation (c).

scribed. Complications related with OM and BRONJ were treated successfully however the patient with ORN failed to heal even after secondary treatment with microvascular surgery.

A patient with ORN related pathological fracture deceased just after the operation due to respiratory arrest related with pneumothorax.

DISCUSSION

Pathological fractures are complex to treat due to their diverse aetiologies. Colletti and Ord reported 43 patients with pathological fractures with wide variety of etiologies such as ORN, impacted teeth extractions, BRONJ, endoosseous implants, OM and malignities. ORN is reported as the most predominant aetiologic factor for pathological fractures. OM and BRONJ are the most common second and third causative factors of pathological fractures.[3] Cancer metastasis, histiocytosis multiple myeloma, odontogenic cysts, actinomicosis infection and giant cell reperative granuloma are other reported reasons of pathological mandible fractures.[6-11] Abir et al. reported ten cases of pathological fractures due to malignities, ORN, cysts and atrophy.[5] In our case series, we evaluated seven cases who have inflammatory causes such as ORN, BRONJ or OM and two cases who have fractures without any pathological condition rather than severe atrophy. Since atrophy is a natural process, it is contraversial to classify this type of fracture as pathological fracture.

As atrophic fractures caused a contraversy in definition of pathological fractures, a new classification named with atrophic mandibular fractures have been arisen. Atrophic mandibular fracture is defined as a fracture that occurs predictably at the body (often bilateral) due to mechanical weakening because of atrophy.[12] Although some studies presented atrophic mandibular fractures under classification of pathological fractures, we considered atrophic fractures seperately and highlighted new terminology of atrophic fractures.[4,5] Castro-Núñez et al. reviewed the treatment modalities for the management of atrophic mandibular fractures.[13] Brucoli et al. assessed the demographic variables, causes, and patterns of edentulous atrophic fractures of the mandible and reported female predominance with an avarage age of 75. The most frequent causes of injury was fall and assaults for atrophic fractures.[14.]However two cases of atrophic fractures occured spontaneously while chewing and sleeping in our case series. Surgical approach is predominant modality of treatment for atrophic mandibular fractures.[13,14] Two male patients with mean age of 68 who had atrophic fractures were treated with open reduction and internal fixation with reconstruction plates in our case series.

Male predominance with an average age of 60.6 years was reported for 25 spontaneous fractures.[2] Male patients were predominant and mean age was reported as 59 years by Coletti and Ord in the cohort study of pathological fractures.[3] The incidence of pathological mandibular fractures is reported higher in male patients unlikely atrophic fractures.[5,14] The avarage ages of pathological fractures were high.[2-4] This could be explained by the fact that older patients are more prone to have malignancies, atrophic jaws or osteoporosis. In our study, 6 male and 1 female

have had pathological fractures with mean age of 60.1 years.

Carlsen and Mercussen reported 25 patients with spontaneous fractures. Regardless of the cause, body of the mandible is the most predominant region to have pathological fractures.[2] Although condylar region constitute the majority of the traumatic fractures, it has rarely been fractured due to pathological reason.[2,4,15] In our case series, corpus mandible is the most predominant region to encounter with pathological fractures as in the previous literatures. One of our cases have had condylar fracture secondary to ORN. Linderup et al. reported a patient with a head and neck infection involving mandibular condyle and resulting with pathological fracture of condyle which was managed non-surgically.[16] A case of pathological condyle fracture due to BRONJ was treated non surgically by Carlsen and Marcussen.[2] Jowett et al. reported a case with coronoid process fracture secondary to BRONJ in which removal of the fractured coronoid process and debridement of necrotic bone were performed as treatment.[17] Cancer metastasis ORN, giant cell tumour are reported reasons of pathological condyle fractures.[4,18,19] We have treated our patient with condylectomy without reconstruction with the aim of pain relief.

Pathological fractures in conjuction with ORN is more complex to treat as pathological fracture presents with advanced surrounding soft tissue fibrosis that may be a primary cause for increased complication rate.[20-22] Marx claimed that ORN occurred when radiation-induced endoarteritis led to areas of poor vascularity and hypoxia, in turn causing chronic inflammation and eventually wound breakdown. In cases of ORN, difficulty in swallowing, nutritional problems, limited mouth opening caused by previous surgeries or radiotherapy are superimposed to decreased blood supply of soft tissues.[23] One review of 1,000 patients having head and neck radiation found 2.6% developed ORN and 23% of these progressed to pathological fractures.[24] Localized factors such as absence of cervical vessels and systemic status of patients may compromise microvascular flap reconstruction.[3] Sawhney and Ducic reported 36 patients with pathological fracture due to ORN whom the resections were performed and reconstruction plates were applied. Twenty six out 36 patients reconstructed with microvascular surgery, secondarily. However 24% complication rate was reported in that cohort study.[25] Non-vascularized bone grafts are not recommended for continuity defects of irradiated patients.[26]

BRONJ cases are seen more frequently therefore the challenge of managing BRONJ is becoming a mainstay of interest in oral and maxillofacial surgery. Pathological fractures secondary to BRONJ may develop following curettage of BRONJ lesion.[27] Although soft tissue is not damaged unlikely the cases of ORN, these patients usually suffers from malignities. Luckily our cases were prescribed bisphosphonates for osteoporosis. Wongchuensoontorn et al. reported three cases of pathological fractures that had developed secondary to BRONJ. The cases were treated with resection of necrotic bone and reconstruction plates were used to bridge the gap without any bone graft.[28] In our cases we have performed a segmental resection of necrotic part of mandible. Primary reconstruction has not been planned. Reconstruction plates were used to stabilize the segments in resected mandibles. In the literature, there is still an ongoing debate on the treatment of pathological fractures due to BRONJ. Some studies suggest a conservative treatment for as long as possible, whereas other studies suggest an aggressive surgical treatment.[29-32] Engrroff and Kim supported primary reconstruction with vascularized bone.[31] However, some patients with pathological fractures are not suitable candidates for immediate reconstruction due to their systemic conditions such as underlying cancers, advanced systemic diseases.[28] Three of our cases who had condylar fracture due to ORN and fractures related with severe atrophy did not have indication of bone reconstructions. Remaining cases who were treated with resections of necrotic bone segments caused by ORN, BRONJ or OM could be treated with microsurgical reconstructive procedures. However, considering the risk of perioperative complications and unsuitable systemic conditions of patients, surgical resections without further surgeries for bone grafting were preferred in our cases. We aimed palliation of pain and maintainance of oral function to obtain a better quality of life.

A mandibular pathological fracture caused by OM is managed stepwise. Antibiotic therapy is prescribed against the causative microorganisms and then treatment of fracture is planned depending on the amount of viable bone following resection or sequestrectomy.[2,3] OM can be associated with implants or third molar extractions. Furthermore, underlying conditions such as diabetes predispose to OM.[3,33] Ogasavara et al. reported a case of pathological fracture of the mandible secondary to OM who was treated with intermaxillary fixation.[34] The cases of pathological fractures secondary to OM were treated with antibiotic therapy at first and then fixated surgically with miniplates or reconstruction plate. However, infection and hardware fracture have been noted in our patient who was fixated with miniplates. Antibiotic treatment was represcribed and resection was performed for infected bone and reconstruction plate was used for fixation following removal of miniplates. Second OM patient of us who was treated with resection and fixation with reconstruction plate reported no complication.

The management of atrophic mandible is controversial since limited buttress of bone and elder age of patients. General health status may contraindicate general anesthesia in aforementioned cases. Therefore conservative treatment had to be choice of treatment.[2] Atrophic mandible is defined as having a vertical height less than 20 mm.[35] Fixation with a rigid plate such as reconstruction plate via an extraoral approach is preferred as closed reduction techniques showed higher complication rate.[36,37] In vitro study of Sikes et al. proved reconstruction plates are more stable than miniplates.[38] We have treated fractures of atrophic mandibles with reconstruction plates via submandibular approach. There were no complications on follow up, bony unions could be obtained and the patients recovered easily. Treatment results of atrophic mandibles were better than other pathological fractures with bone pathology.

Two main reasons are presented which challenges treatment of pathological fractures. First one is systemically compromised patients are avoided extensive surgeries, the second one is bone quality which is probably non-viable.[3] Pathological fractures of the mandible most often have to be treated by eliminating the underlying condition while immobilizing the fragments either with osteosynthesis or intermaxillary fixation.[4] Coletti et al. have based their management strategies as an algorithm. The patients with bone diseases which restrict normal bone healing were suggested to treat with bone resection and reconstruction plates application with or without primary or secondary reconstruction. This method is proposed for cases with ORN, BRONJ and OM.[3] Resection till healthy bone is reported to be important for success of treatment. Ionnides et al. recommend resection past 1 cm normal appearing bone which is recognisable by normal bleeding.[39] We treated our patients with reconstruction plates due to bone loss caused by destruction or resection in the fractured area. Coletti and Ord treated majority of their patients with resection and fixation with reconstruction plates. Complication rate was reported as 40% following treatment including plate expose, hardware failure, infection, oral or cutaneous fistulas, malocclusion and graft failure, respectively, 59% of these complications were associated with patients who received radiation therapy.[3] Zanicotti et al. reported plate expose in a case of pathological fracture secondary to ORN.[40] In our case series, we have inspected fractures caused by ORN are more prone to complications. Furthermore, it is hard to deal with these complications in irradiated patients as reported by Coletti and Ord.[3] One of our patients who received radiation therapy died of anesthesia complication related with ventilation problem due to restricted mouth opening. Plate expose due to scarred soft tissue with poor healing potential patient was observed in an irradiated case. Although free flap reconstruction was performed to deal with that complication, the case did not respond well and partial flap loss has occured. One of our patients had failure of reconstruction plate which lost its stability and almost perforated the skin in six months. The patient was reoperated and reconstruction plate was exchanged successfully with longer one which extended to contralateral side of mandible. Plate expose could be hindered by means of early intervention before perforation of skin and good quality of soft tissue unlikely post radiation therapy.

To summarize, pathological fractures have a diverse aetiology and the presence of systemic diseases and localized bony diseases makes these patients dif-

ficult to treat. Atrophic fractures of mandibles should be handled separately as there is no major qualitative disorder in bone tissue that could challenge the treatment. We conclude that the treatment of mandibular fractures due to BRONJ gives more stable results comparing with ORN and OM by reason of better condition of surrounding tissues. We suggest to take some points into account to obtain a successful result. Removal of necrotic segment till the bleeding bone margins is key point to accelerate healing process and to avoid further induction of necrosis. Local and radical resections lead to defective fractures that indicated to treat with load bearing plates instead of load sharing plates. Patients did not demand secondary reconstruction in follow up period except one patient with partial flap loss. To conclude treatment strategy must be adapted to each patient individually.

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Conflict of Interest

No conflicts of interest between the authors and / or family members of the scientific and medical committee members or members of the potential conflicts of interest, counseling, expertise, working conditions, share holding and similar situations in any firm.

Authorship Contributions

Idea/Concept: *Esin Demir;* ***Design:*** *Esin Demir, Çağdaş Elsürer;* ***Control/Supervision:*** *Mete Kaan Bozkurt;* ***Data Collection and/or Processing:*** *Esin Demir, Ömer Erdur;* ***Analysis and/or Interpretation:*** *Esin Demir;* ***Literature Review:*** *Esin Demir;* ***Writing the Article:*** *Esin Demir;* ***Critical Review:*** *Ömer Erdur;* ***References and Fundings:*** *Çağdaş Elsürer;* ***Materials:*** *Mete Kaan Bozkurt.*

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