Review Article

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Current management of avascular necrosis of the metacarpal head: a comprehensive literature review

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Background: Avascular necrosis (AVN) of the metacarpal head is a rare disease that may lead to progressive destruction of the metacarpophalangeal joint and hand function. This study aimed to describe the epidemiology, possible risk factors, clinical presentation, diagnostic workup, and treatment of the rare condition of avascular necrosis of the metacarpal head.

Methods: Articles were searched using the subject words "Dieterich disease","Mauclaire's disease", and "avascular necrosis of metacarpal head" in the PubMed and Scopus databases. Studies were retained for review after meeting the inclusion criteria. Those outcomes relevant to diagnose and assessing AVN of the metacarpal head and those related to curative management were extracted.

Results: The literature search revealed 45 studies with 55 patients. Although the aetiology of osteonecrosis has not been clearly delineated, AVN of the metacarpal head most commonly arises from trauma but other risk factors may also be involved. Plain radiographs are often negative and therefore likely to be missed. Early-stage osteonecrosis of the metacarpal head was best assessed using MRI. Given the rarity of this condition, there is no clear consensus on the treatment.

Conclusions: Avascular necrosis of the metacarpal head should be considered in the differential diagnosis of painful metacarpophalangeal joints. An early understanding of this unusual disease will provide an optimal clinical outcome, restoring joint activity, and resolving pain. Nonoperative treatment cannot cure all patients. Surgical management is based on the patient and lesion characteristics.

Keywords: avascular necrosis (AVN), dieterich disease, metacarpal head, osteonecrosis

Introduction

AVN of the metacarpal head is a rare disease. Synonyms of AVN include osteonecrosis, ischaemic necrosis, subchondral avascular necrosis, and aseptic necrosis of the bone. The earliest description of the disease was described by Mauclaire 1927, but it is also known as Dieterich disease with an unknown aetiology[1–4]. There have been few reports in the literature on avascular necrosis of the metacarpal head[2,3,5–15]. Most of them were case reports, which lacked detailed and systematic descriptions.

HIGHLIGHTS

- Avascular necrosis (AVN) of the metacarpal head is a rare disease.
- There is no clear consensus on treatment of this avascular necrosis.
- Early understanding of this unusual disease will provide an optimal clinical outcome.
- We reviewed avascular necrosis of the metacarpal head and the theories regarding the aetiology and the treatment options for this rare disease.

Meanwhile, there is no clear consensus on the best treatment strategy due to the rarity of the condition. This study aimed to describe the epidemiology, possible risk factors, clinical presentation, diagnostic workup, and treatment of the rare condition of avascular necrosis of the metacarpal head. This may benefit enhancing the awareness and medical management of AVN of the metacarpal head.

Methods

This review was performed by collecting articles published in peer-reviewed scientific journals. PubMed and Scopus databases were searched to retrieve published studies and accompanying references, using keywords or combination of keywords. The "Dieterich disease" and "Mauclaire's disease" each were used as a query. And the combination of keywords used in PubMed was as follows: (metacarpal head) AND (necrosis or avascular

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necrosis or osteonecrosis). We further screened reference lists of all included studies to identify any other potentially eligible articles. The last literature search for this review was performed on 31 July 2021.

Studies published in 1990 or later, regarding cases of AVN affecting bones of metacarpal were included. We included observational studies (case reports, case series, prospective, or retrospective studies) that provided data concerning characters of avascular osteonecrosis of the metacarpal head. Comments, conference abstracts and letters to editors were excluded. In additional, articles of which the original text could not be obtained or in which AVN data could not be extracted were also excluded.

Duplicate articles were removed, and a preliminary review of the remaining studies was conducted according to the title and abstract. According to the above selection criteria, the full-text search of relevant literatures and their citations was carried out. To facilitate extraction of results and explore the possibility of further analysis, studies selected for review were documented (Supplemental Digital Content 1, http://links.lww.com/JS9/A324). Most of them were case reports, which also involved the potential mechanisms of the disease that result in osteonecrosis. Data on corticosteroid use, trauma, genetic factors, and other characteristics of osteonecrosis were reviewed. These associated clinical features, as well as advances in diagnostic techniques, were also compiled. Finally, the treatment of avascular osteonecrosis of the metacarpal head was studied.

Results

About 45 articles describing ~55 patients were included in our review. The flowchart illustrates the study selection process in our review (Fig. 1). The studies included in this review, as well as patient characteristics, are summarized (Tables 1 and 2, Supplemental Digital Content 1, http://links.lww.com/JS9/A324).

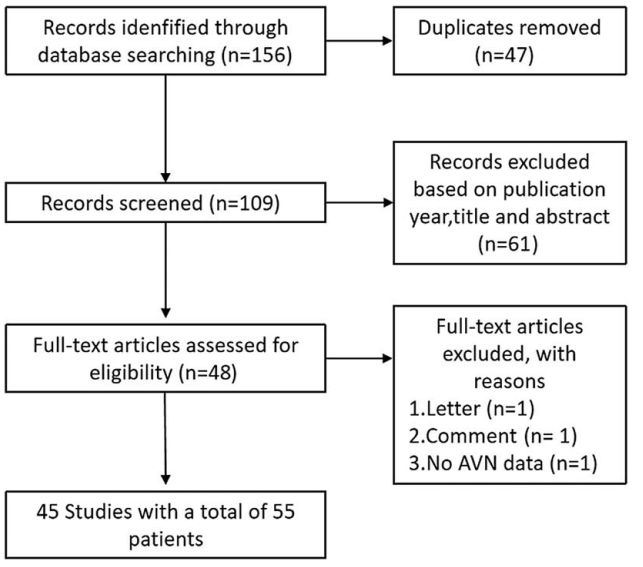

Figure 1. Flowchart presenting the study selection process of publications on avascular necrosis of the metacarpal head. AVN, avascular necrosis.

Table 1

Demographic epidemiology (total patients: 55).

Sex	38 (male)	15 (female)	2 (unknown)	
Age, (year)	34 (< 20)	12 (21–50)	8 (> 51)	1 (unknown)
Hands involved	41 (right)	7 (left)	4 (bilateral)	3 (unknown)
Fingers involved	47 (solely)	5 (double)	3 (multiple)	
thumb	1	0	0	
Index	7	0	4	
Middle	20	7	5	
Ring	18	3	4	
Little	1	0	4	
Susceptibility factor	27 (prior trauma)			
	21 (idiopathic or no reason)			
	Steroid use includes 2 (juvenile dermatomyositis), 1 (inflammatory lung disease), 1(nephrotic syndrome)			
	1(prothrombin 20210 mutation)			
	1 (Freiberg disease)			
	1 (SLE)			

SLE, systemic lupus erythematosus.

Epidemiology

AVN usually involves the distal end of the long bone, most commonly the femoral head, and rarely the metacarpal head. There were fewer than 30 cases of metacarpal head AVN described in the literature before 1990[1]. We reviewed the literature from 1990 to 2021 and identified 45 articles describing ~55 patients. The age of patients in the literature between 6 and 82 years is predominantly teenagers[16,17]. This was confirmed in our review. Approximately 61.8% of the reported patients were under the age of 20 years, especially in the second decade. According to Wright and Dell[1], men are slightly more affected than women with a male-to-female ratio of 3:2. Our statistics show that the ratio of males to females is ~7:3, and males tend to be more frequently affected. Dieterich is observed in all metacarpals, most commonly in the middle finger, and most rarely in the thumb[1,2]. It has also been reported to involve multiple metacarpal heads[18–21]. In the present review, more than two-thirds of the lesions were in the middle and ring finger. There were 85.5% of cases involving only one finger, four had two fingers, and the remaining four had multiple lesions. The right hand was affected by 78.8% of the 52 patients who reported the injured side of the hand. Only seven cases occurred in the left hand only, and the remaining four cases occurred bilaterally (Table 1). Our review indicated that AVN was more likely to develop the solitary lesion, in the middle and ring finger of the right hand, in males under the age of 20 years.

Pathogenesis of osteonecrosis

The aetiology of avascular necrosis of the skeleton is unclear. It is widely believed that the ultimate common pathway for osteonecrosis involves a lack of blood flow to the bone[22,23]. Reduced blood circulation of the bone due to a particular vascular configuration is also discussed in the AVN of the metacarpal head[1,23]. In a metacarpal vascular anatomy study, Wright and Dell noted the absence of large nutrient vessels in 35% of specimens, which may place distal metacarpal epiphyses at risk for

Table 2

Imaging examination and treatment (total patients: 55).

Imaging	52 (X-ray)
	29 (MRI)
	10 (bone scanning / bone scintigraphy)
	5 (CT)
Therapy	18 (nonoperative)
	28 (operative)
	8 (unknown)
	1 (no therapy)
Nonoperative[18]	10 (success)
	4 (nonoperative with no outcome)
	4 (failed or refuse surgery)
Operative[28]	13 (curettage and cancellous grafts)
	5 (osteochondral autograft transplantation surgery)
	2 (wedge flexion osteotomy)
	2 (costal osteochondral graft)
	2 (transplantation of a metatarsal head)
	1 (total joint replacement)
	1 (hemiarthroplasty)
	1 (arthrodesis)
	1 (denervation)

CT, computed tomography.

AVN development[1]. Because of the lack of significant intraosseous vascularization, the metacarpal heads relied solely on small pericapsular circumferential arterioles. The incidence of small pericapsular arteries alone in the long finger was 60%. Any compromise to these small pericapsular vessels might jeopardize the nutrition of the metacarpal head, leading to avascular necrosis of the metacarpal head.

AVN of the metacarpal head may occur without any obvious specific possible risk factors and is therefore named idiopathic[6,10,24,25]. Its association with trauma and anatomic variations in the blood supply to the metacarpal head has also been recognized[2,3,7]. As with other aseptic bone necrosis, a connection to steroid intake has been discussed in Dietrich's disease[1,26]. Comorbidities such as systemic lupus erythematosus (SLE), Freiberg disease, and juvenile dermatomyositis have been described[21,27,28]. In this study, we summarize the possible risk factors for its development (Table 1). Our review indicated that the most cases were trauma related, with only a few sporadic cases possibly related to steroid use or other diseases. Therefore, trauma may be an important cause of AVN of the metacarpal head.

Trauma

AVN of the metacarpal head is believed to be either the result of an occult fracture or secondary to a traumatic effusion[2,5,14,15]. The metacarpophalangeal (MCP) joint is usually prominent in the hand and is thus vulnerable to damage. The higher rate of involvement of the third metacarpal head is thought to be the result of its relative prominence and susceptibility to trivial trauma[1]. Traumatic effusion of the MCP joint may jeopardize the nutrition of the metacarpal head[29]. In the face of traumatic effusion, the tamponade effect of these vessels may lead to avascularity of the distal epiphyses, accompanied by bone and cartilage necrosis and collapse. Angiography showed venous pooling, suggesting that blunt trauma or occult microfracture may compress the periosteal blood vessels and delay venous return[25]. This indicated that delayed venous return might result in hypoxia and increased intramedullary pressure, which eventually leads to necrosis and collapse. Sagar *et al.*[7] reported two paediatric cases of AVN of the fourth metacarpal head secondary to indirect trauma. One patient also had a fifth metacarpal neck fracture, and the other had an occult fracture of the fourth metacarpal before AVN. Moreover, the metacarpal fracture itself may lead to avascular necrosis of the metacarpal head. McElfresh *et al.*[30] found that transverse metacarpal head fractures presented with a high incidence of ischaemic necrosis, especially when the fracture was displaced.

In addition, Gannon *et al.*[14] believe that chronic, repetitive loading of the metacarpal heads may have led to avascular changes. Multiple metacarpal head involvement has been reported in a 14-year-old gymnast, possibly secondary to repetitive trauma[20]. Similarly, a case of bilateral multiple metacarpal head necrosis was reported in a 14-year-old patient with a history of rock climbing[19]. These reports of AVN after direct or indirect trauma suggest that disruption of the vascular supply to the metacarpal head may be the most important risk factor.

Corticosteroids use

The effects of glucocorticoids on the human system are numerous and extremely complex[31]. Systemic corticosteroids, which are associated with osteoporosis and pathological fractures, may also cause chylomicrons to increase, thus blocking the terminal artery, leading to bone necrosis[1]. Altered lipid metabolism induced by corticosteroids via increased adipogenesis, fat hypertrophy, and fat emboli. It may cause ischaemic osteonecrosis through elevation of intraosseous pressure[31]. It is also believed that steroid-induced bone disease arises from changes in the number of bone cells. The accumulation of apoptotic osteocytes may contribute to osteonecrosis[22]. There have been reported cases of AVN of the metacarpal head coexisting with talus due to the consumption of high-dose cortisone disease for severe inflammatory lung disease[13].

Juvenile dermatomyositis (JDM)

Juvenile dermatomyositis is the most common chronic inflammatory myopathy in children. Current treatments include glucocorticoids and methotrexate, with a long usage period. Although rare in JDM, Robinson *et al.*[21] reported two cases of metacarpal head necrosis. In these two juvenile patients, osteonecrosis involved multiple metacarpal heads and carpal bones. It is speculated that metacarpal AVN may be a complication of JDM treated with corticosteroids.

Renal transplant

Renal transplant recipients usually have years of systemic chronic renal failure and renal osteodystrophy. The direct effects of toxins in the body, such as uraemia, combined with the effects of long-term systemic steroid therapy, lead to a high rate of osteonecrosis[22]. Furthermore, osteonecrosis and spontaneous fractures occur in the trabecular area after renal transplantation[32]. Femoral head necrosis is the most common lesion, and multiple metacarpal head necrosis is also involved[26]. However, they suggest that avascular osteonecrosis is a complication of long-term systemic steroid therapy in renal transplant recipients[26].

Nephrotic syndrome

The youngest case of metacarpal head necrosis reported in the current literature was a 6-year-old boy with nephrotic syndrome[16]. He had received glucocorticoid therapy for eight months. The patient had no symptoms during the entire clinical course and received no treatment, but the third metacarpal was eventually completely regenerated.

Systemic lupus erythematosus

SLE is a multisystem autoimmune disease. Steroids are an essential part of treatment programs. Moreover, osteonecrosis in patients with SLE generally involves multiple metacarpal heads[18,28]. Although most reported cases of osteonecrosis were associated with steroid use, it may not be the only risk factor in patients with SLE. Vasculitis might result from inflammation of the small end-arterioles causing obliterative arteriopathy with luminal narrowing, resulting in ischaemic injury[7]. Arteriolar vasculitis may act alone or in conjunction with fat embolism induced by steroid therapy, resulting in arteriolar occlusion and ischaemic necrosis of the bone[18,29]. The presence of antiphospholipid antibodies, known to cause intravascular coagulation and vasculitis, has also been suggested as a potential cause of osteonecrosis. Patients with antiphospholipid antibodies may benefit from anticoagulant therapy for the risk of osteonecrosis[22]. Therefore, SLE may be an independent risk factor.

Hypercoagulability

An increased tendency for intravascular coagulation and thrombosis appears to be a potential risk factor for osteonecrosis. Coagulation disorders, including factor V Leiden-mediated thrombosis and protein C, S, or antithrombin III deficiency, may occur in patients with osteonecrosis[22]. Resistance to activated protein C is the most common genetic defect observed in patients with thrombosis. activated protein C -resistant genotypes have been shown to be based on a mutation in factor V (factor V Leiden). In the case of metacarpal head necrosis, analysis of mutations in the factor V Leiden and prothrombin 20210A genes showed positive results[33]. Therefore, occlusion of the microcirculation may be a risk factor for metacarpal osteonecrosis.

Freiberg's disease

Freiberg's disease is thought to be osteochondrosis, and most often occurs in the second metatarsal bone. Its prominent position and relative immobility may cause repeated microdamage increase loads during weight-bearing, leading to synovitis and damage to the blood supply[27,34]. The AVN of the metacarpal bone is similar to Freiberg's disease clinically and radiologically. Cases of AVN of the metacarpal head with concomitant Freiberg's disease have been described, indicating an association between them[17,34].

Alcoholism

Alcohol abuse is a possible aetiologic factor for femoral head osteonecrosis. Their associations have been discussed. Alcohol appears to impair blood flow through fat embolization or fat hypertrophy, leading to cell death due to its direct toxic effect on osteocytes and other marrow elements[22]. However, there have been no studies on the correlation between alcohol consumption and metacarpal head osteonecrosis. Only one article mentioned that alcohol consumption might be an underlying risk factor for metacarpal head necrosis[29].

Although the aetiology of avascular necrosis of the metacarpal head may be multifactorial. Our review indicated that most cases are trauma related. Moreover, it was found in the anatomical study that the blood supply of the metacarpal head was susceptible to injury. Therefore, trauma may be an important cause of AVN of the metacarpal head. The few cases of osteonecrosis are not sufficient to prove that it is due to SLE, Freiberg's disease, JDM itself or steroid use. It remains unclear whether it is the steroid or these diseases themself that causes AVN of the metacarpal head. However, osteonecrosis should also be suspected in adolescent patients with these diseases if they experience hand pain. Other potential risk factors need to be explored further.

Diagnosis and assessment (Table 2)

Clinical symptoms and imaging examinations are the main tools for the diagnosis of osteonecrosis. A high degree of suspicion is required, especially if the patient has a history of trauma, steroid use, or other predisposing risk factors. The introduction of new imaging techniques, such as MRI, can help in the early diagnosis and improve treatment outcomes. As for the stage of metacarpal necrosis, no relevant literature has been reported to date. We can refer to the stage of femoral head necrosis or Freiberg's disease[22,27]. With early detection of this disease, appropriate protective measures can be instituted to minimize further joint damage.

Clinical presentation

Early clinical manifestations of AVN often lack specificity. There may not be any symptoms in the early stages of the disease[35–37]. Therefore, they were likely to miss a diagnosis. Clinical symptoms vary in severity, ranging from asymptomatic to pain, swelling, and limited movement of the MCP joint. The leading symptom is local pain over the affected MCP joints, and more specifically, the metacarpal heads[7,11,38]. Other symptoms may include crepitus and reduced grip strength[3,12]. Physical examination revealed tenderness over the metacarpal heads, with or without erythema, swelling, synovitis, or limited range of motion[8,10,29]. Subtle crepitation and subluxation may involve the affected MCP joint[3,27].

Imaging

Conventional X-ray films have been widely used foe the assessment of osteonecrosis. The X-ray shows characteristic changes with disturbance of the trabecular pattern, flattening, and sclerosis (Fig. 2)[8,10,12,39]. It sometimes has a step-off of the articular surface and collapse of the metacarpal head[29]. Initially, the X-rays were negative for fractures or other pathologies. During follow-up, X-rays may show subtle subchondral lucency and relative osteopenia in the affected metacarpal head. It is followed by fragmentation, sclerosis, and degenerative osteoarthritis of the MCP joint in later stages[9,40]. These include asymmetric joint space narrowing and osteophyte formation.

Computed tomography is rarely used to diagnose osteonecrosis but can show detailed osteonecrotic lesions. Collapse with a subchondral fracture, cystic changes, and osteochondral defect, including a crescent sign with cortical impaction of the

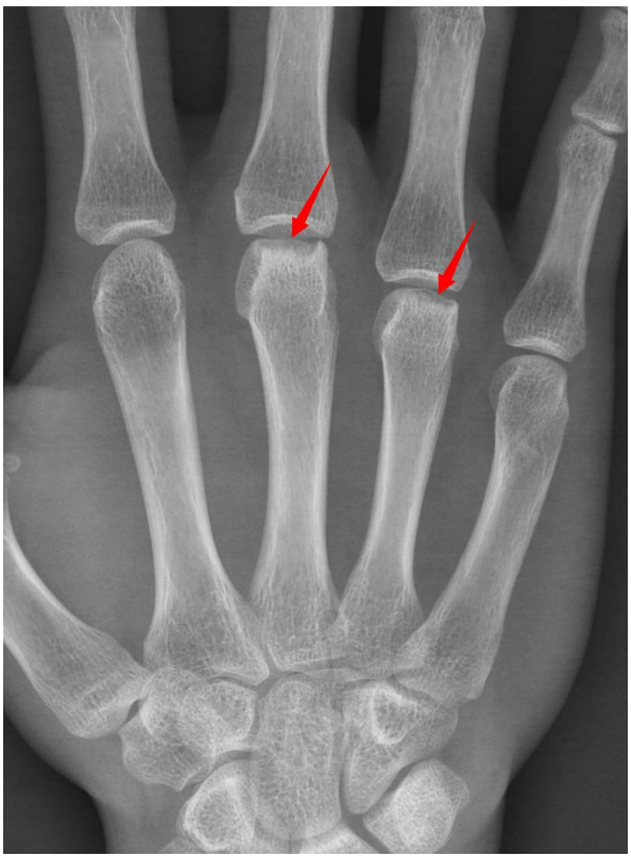

Figure 2. Anteroposterior radiograph shows subchondral collapse and sclerotic changes in the long finger and ring finger metacarpal head.

metacarpal head at the time of initial diagnosis, would be observed on computed tomography examination[8,19,41]. The crescent sign is a characteristic early finding of avascular necrosis, indicating disruption between the articular cartilage and the subchondral bone (Fig. 3)[42].

MRI is the most sensitive tool for the diagnosis of bone AVN. Plain radiographs may be normal in the early stages of AVN. MRI shows that these early changes in AVN are usually localized to the superior or subchondral regions of the bone[12,43]. The characteristic findings of AVN on MRI include T1-weighted images showing diminished signal intensity within the background of the bright signal of fatty marrow, and T2-weighted sequences displaying a distinctive pattern designated the double-line sign[11,14]. MRI can also assess the extent of the necrotic area and evaluate the overlying cartilage. In the case of a preceding history of trauma, MRI confirmed a subchondral fracture in the metacarpal head and joint effusion in the MCP joint[7]. This information can be helpful for surgical planning and management.

Bone scan (or bone scintigraphy) can be helpful in discovering increased uptake at the MCP joint in the early stages[44]. In addition, it can screen for other bone lesions, particularly in patients receiving systemic steroid treatment, who are at a high risk for AVN. Bone scanning has high sensitivity but low specificity. Bone scintigraphy can diagnose osteonecrosis prior to radiographic changes[18]. Chronic inflammation and avascular necrosis are difficult to distinguish when bone scans show increased uptake.

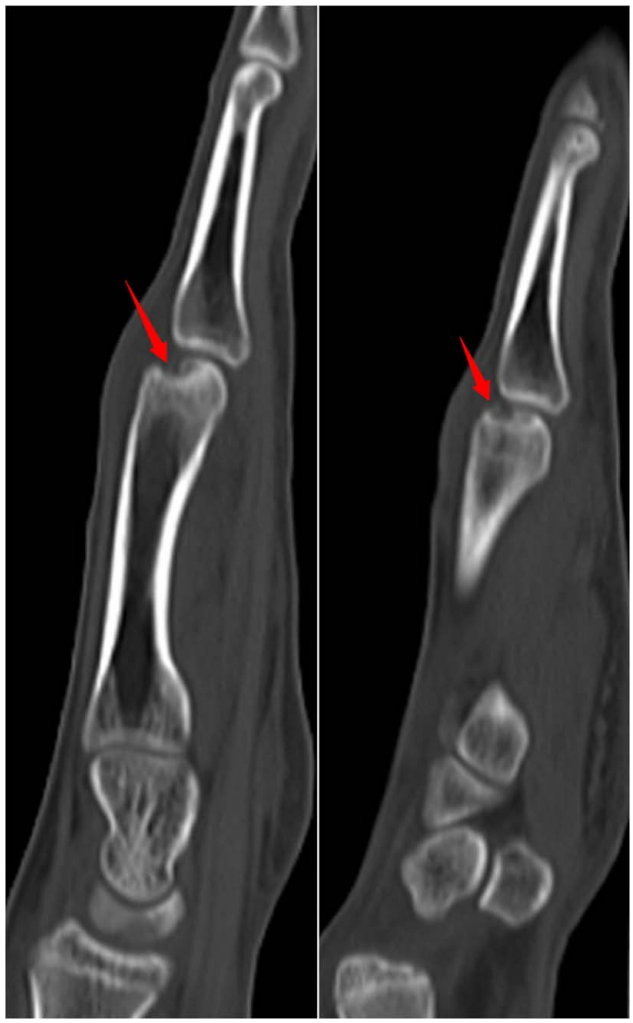

Figure 3. Sagittal images of computed tomography demonstrate cystic, osteochondral defect, and sclerotic changes in the metacarpal head.

Less common examination

Histologic examination is usually performed postoperatively, most of which show osteonecrosis[10,11,33,45]. Digital subtraction angiography can examine the blood circulation supplying the metacarpal head and check whether there is a lack of blood flow to the bone[25]. Arthroscopy is not used as a routine examination and can be used as a therapeutic examination to determine the ultimate surgical treatment[12].

Treatment

Given the rarity of this condition, there is no clear consensus on the treatment. The literature contains descriptions of both nonoperative and surgical therapies, both with different successes. The treatment ranges from rest to prosthetic arthroplasty or arthrodesis (Table 2). In a 6-year-old patient with steroid-treated nephrotic syndrome, the metacarpal head completely regenerated after avascular necrosis, although no treatment was administered[16]. Successful nonoperative treatment has also been reported, but many patients eventually undergo surgery.

Nonsurgical management

Metacarpophalangeal joints are non-weight-bearing joints, and nonoperative treatment is usually the first option. The simplest treatment is joint immobilization with splinting or a short-arm cast[2,7,15]. Limitation of activities and immobilization of the MCP joints involved is recommended over the first few weeks. The duration of joint immobilization ranged from two to six weeks[7]. Non-steroidal anti-inflammatory drugs and physiotherapy, such as electrical stimulation[21], can be used in patients with persistent symptoms. Extracorporeal shock waves and pulsed electromagnetic fields have been used to treat osteonecrosis[46]. The rest, immobilization, and use of non-steroidal anti-inflammatory drugs may be sufficient to control symptoms, and nonoperative treatment has been reported to be successful[2,3,47]. In one case, intra-articular steroid infiltration was used, and the symptoms were not controlled, but no detailed instructions were given[29]. Other pharmacological agents, such as diphosphonates[21], can also be used for treatment, but their clinical effect is unclear.

However, the duration of the course of nonsurgical management remains ambiguous. Nonsurgical management was followed for 3 months in some cases[3,11]. Similarly, nonsurgical management was offered to all patients (rest, placement of an orthosis, and non-steroidal anti-inflammatory drugs) for 3–6 months in another study; two patients had full active range of motion and pain symptoms improved[2]. The duration of nonoperative treatment should be longer for adolescents, especially for those with multiple lesions. Most cases of successful nonoperative treatment were juveniles, which might be related to the regenerative potential of the growing bone[3,15,24].

Surgical treatment

Nonoperative treatment is generally believed to be at an increased risk of early arthritic involvement of the MCP joint in the future[11,12,14,41]. Surgery may be considered when nonoperative treatment fails. Many studies believe that persistent pain with subchondral collapse of the metacarpal head should undergo surgical therapy[2]. Bone curettage and cancellous bone grafting, flexion osteotomy, metatarsal transplantation, costal osteochondral autograft, osteochondral autograft transplantation surgery, arthroplasty, MCP joint denervation, and arthrodesis have been performed in individual cases (Table 2)[2,11].

In the studies examined in this review, there is no uniform standard for surgical indications. Generally, the type of surgery depends on the damaged condition of the cartilage layer. If the cartilage is still good, subchondral curettage and cancellous bone grafting yield excellent results[29]. If the cartilage is destroyed, other joints-preserving surgery techniques may be necessary. Arthrodesis or joint replacement should be considered as salvage operations when cartilage erosion is severe or joint surfaces collapse with persistent symptoms.

Decompression

Surgical decompression has been reported to provide pain relief and functional improvement. Core decompression is to reduce the intramedullary pressure and block the procedure of worsening ischaemia. Treatment in the early stages of the disease leads to better clinical outcomes[22]. Myerthall and Graham reported a case of avascular necrosis at the base of the second metacarpal. A 1.6 mm K-wire was used to drill holes into the metaphyseal area to decompress the medullary canal. After a transient improvement in the symptoms following this procedure, the patient underwent a second decompression through a cortical window and a cancellous bone graft three months later[48]. There have been no case reports of metacarpal head core decompression alone, and most of the cases were performed with both open window decompression and cancellous bone graft[2,11]. Occasionally, arthrotomy and articular debridement have been performed to reduce the pressure in the articular cavity[10,29]. The long-term prognosis of the metacarpal head and metacarpophalangeal joint after surgical decompression is unknown.

Curettage with or without cancellous bone graft

Curettage and cancellous bone grafting are among the most widely used surgical methods. Necrotic bone fragments were removed through a small hole in the cartilage-bone junction, and cancellous bone was transplanted from the distal radial epiphyseal to fill in the subchondral defect[2,10,11,29]. This method preserves the intact cartilage layer and provides it with a solid underlying bony base. Hu *et al.*[10] reported a case treated with curettage of necrotic bone, debridement of inflammatory synovium, and multiple drilling at the bare bone of the articular surface with 0.16 cm Kirschner wires. However, Karlakki and Bindra concluded that curettage and bone grafting were not revascularization procedures. It could not stop the progression of degenerative deformities of the metacarpal head[11]. It has been reported that a vascularized bone graft from the radial side of the adjacent metacarpal bone was placed on the cancellous bone graft in the necrotic metacarpal[33]. In many cases, curettage and cancellous bone grafting have reported excellent results; there have been no reports of metacarpal head degeneration during long-term follow-up.

Osteotomy

Wada *et al.*[12] reported a surgical approach by performing flexion osteotomy of the metacarpal neck in a teenager who had AVN of the third metacarpal head. The purpose of metacarpal neck flexion osteotomy is to transfer the mechanical load to the dorsal articular surface. The rotation of the metacarpal head can reduce the tension of the collateral ligaments, thus reducing the contact stress on the joint surface[6]. However, the premise is that the articular cartilage on the dorsal side of the metacarpal should be healthy. The osteotomy was performed in the metaphyseal region through the neck or proximal to the growth plate and gently opened from the dorsal side of the metacarpal head. A wedge-shaped bone graft harvested from the distal aspect of the radius was inserted and stabilized using Kirschner wires[6,12]. Postoperative immobilization was usually performed for six weeks, and Kirschner wire removal after fracture healing was confirmed. However, flexion osteotomy may reduce the range of extension.

Osteochondral autograft transplantation surgery

Osteochondral autograft transplantation surgery has been used for a variety of focal defects and necrosis of the knee, ankle, elbow, and wrist. Some require the use of an internal fixator to secure the graft[5]. It can also be used in metacarpal avascular necrosis with focal or partial osteochondral defects. Large,

completely defective osteochondral in the metacarpal or severe arthrosis in the proximal phalanx is a contraindication to this treatment. The articular surface of the proximal phalanx base must be well preserved[5,49].

Osteochondral plug harvesting from the non-weight-bearing articular surface of the knee, transferring, and pressing-fit to resurface focal metacarpal head lesions is an effective method[5,8]. An osteochondral harvester is typically required. The donor osteochondral plug can be obtained from the non-load-bearing portion of the lateral femoral condyle, between the sulcus terminalis and the physis[5]. In general, the cylindrical osteochondral plug should be carefully removed to avoid damage to the growth plate of the femoral condyle. On the other hand, the radius of curvature of the donor osteochondral plug should match the recipient's metacarpal head as much as possible[5,49]. The void of the femur can be filled with osteochondral allograft[49], substitutes, just left behind[5,50]. Filling the donor site defect can ensure faster healing and lower donor site morbidity[49]. Potential complications of this procedure include cortical penetration at the donor and/or recipient site, failure of plug healing, and incongruence of the articular surface.

Transplantation of metatarsal head

Erne and colleagues first reported on the treatment of two young patients with Dieterich's disease by transplantation of the calotte of the head of the second metatarsal. The prerequisite is the integrity of the base of the proximal phalanx[51]. During the operation, the necrosis calotte of the metacarpal head was resected flat, and the collateral ligament origins were preserved. A graft calotte of the metatarsal head is harvested at the second metatarsophalangeal joint. To achieve greater flexion motion of the metacarpophalangeal joint, the acquired metatarsal head was rotated around 180° so that its plantar portion was placed on the back of hand there. Fixation was performed with an absorbable screw. At the base joint of the second toe, the plantar plate is sutured with the periosteum between the distal metatarsal bone and the phalanx. Two patients had no chief complaints in daily life and had free finger function of their fingers. There was no discomfort in the resection area of the second metatarsal[51]. This method restores the shape and articular surface of the metacarpal head, which restores the range of motion of the MCP joint. While feasible, this approach represents non-vascularized grafts at the expense of the second metatarsal head.

Costal osteochondral graft

Costal osteochondral graft for total metacarpal head replacement is also a rare surgical procedure. Thomsen *et al.*[41] reported a case of extensive osteochondral injury of the metacarpal head treated with a costal osteochondral graft. A box-shaped slot was made throughout the metacarpal head to resect the osteochondral lesions. The collateral ligament origins and ~1.5 mm of the condyles on each side were preserved, and the grafted bone was firmly fixed with screws. A graft is harvested at the osteochondral junction of the sixth rib and shaped to match the defect. It is recommended that cartilage should be as thin as possible because revascularization is mediated primarily by the surrounding soft tissue[41]. As we age, the cartilaginous ribs develop scattered calcification[25,41]. In another report, the collapsed metacarpal head was resected. An osteochondral autograft from the 8th rib was trimmed into the joint cartilage surface and bony stem. The bone graft was directly inserted into the medullary cavity for fixation without screw fixation of the graft[25]. This method requires the donor costal to be carefully molded to form a new metacarpal head.

Joint replacement

Metacarpophalangeal joint replacement has been used in patients with rheumatoid arthritis and osteoarthritis[52,53]. Kim *et al.*[54] reported a case of metacarpal head necrosis following pyrolytic carbon hemiarthroplasty. The pyro-carbon MCP joint is considered to have excellent bone-implant incorporation, long survival rate, and fewer implant complications, with adequate pain relief and improved functional scores. Hemiarthroplasty requires that the joints at the base of the proximal phalanx be well preserved. It could be considered a relatively simple procedure that could reserve more bone stock, in contrast to total joint replacement[54].

For patients with secondary osteoarthritis of Dieterich's disease, total metacarpophalangeal joint replacement can be used[40]. Schmidt reported a case of metacarpal head necrosis with secondary osteoarthritis of the metacarpophalangeal joint. An uncemented cobalt-chrome alloy metacarpal hemispherical head and a cemented ultra-high molecular weight polyethylene phalangeal component were used during the operation[40]. Similarly, silicone elastomers and pyrolytic carbon total joint replacement systems exist[55]. Artificial joint replacement is a technically demanding procedure that requires sufficient bone mass and soft tissue to maintain stability. The long-term effects are still controversial due to implant fatigue fracture, stiffness, and other complications[52]. Instability or complications from soft tissue problems may require revision.

Resection arthroplasty and arthrodesis

Resection arthroplasty involves the removal of bone to correct deformity and allow movement as well as joint surface replacement or tissue intervention to prevent painful impingement or fusion[55]. Joint surface replacement may use the fascia lata, extensor tendon, palmaris plate, and extensor support band[55]. This method, which causes some degree of limb shortening, is rarely used. Arthrodesis is the fixation of the MCP joint at a certain angle using a Kirschner wire, plate screw, or stapler. They can be performed when bone reserves are insufficient to provide prosthesis implant fixation, or when ligamentous support is lacking to provide implant stability[55,56]. Arthrodesis can make joints stable and painless, but they can affect grip strength and function[57]. It should be avoided as a primary surgical procedure and can attempt when joint replacement failure[40]. The patient's functional needs and physiologic age must be evaluated when choosing metacarpophalangeal joint fusion.

Metacarpophalangeal joint denervation

Joint denervation is a minimally invasive surgical option for hand arthritis, which preserves the joint structure while treating pain and reducing interference with biomechanics and range of motion of the joint[58]. Arenas-Prat described the technique for MCP joint denervation as a treatment for painful degenerative or posttraumatic osteoarthritis. A dorsal incision is used to identify and divide the sensory articular branches from the dorsal digital cutaneous nerves. Meanwhile, all palmar sensory articular

branches are divided from the radial and ulnar digital nerves[59]. Bailey applied the MCP joint denervation technique to a patient with avascular necrosis of the metacarpal head, who achieved satisfactory pain relief and grip strength[60]. Surgeons should be aware of the potential for painful neuroma dorsally and neurapraxia of the digital nerves when applied MCP joint denervation[59]. Even so, MCP joint denervation may be a new option for treating AVN of the metacarpal head.

The optimal treatment for AVN of the metacarpal head has not been established, and the long-term prognosis is unknown. Most current recommendations on treatment options are based solely on an individual's experience. In this review, non-steroidal anti-inflammatory drugs is the most important nonoperative treatment. Curettage and cancellous bone grafting are good choices for early-stage patients with intact articular surfaces. Osteochondral autograft transplantation surgery is also a good treatment for filling the defect of the metacarpal head with intact bone and hyaline cartilage as an autologous scaffold. The above methods are widely used in literature and have achieved good prognosis. Arthroplasty can be used as an option for advanced metacarpal head avascular necrosis to restore metacarpophalangeal joint movement and hand function.

Limitations and foresight

Due to the rarity of Dietrich disease, the present review consists of case reports and small case series, and the number of patients included is relatively small, which may affect the results. No major prospective or retrospective studies were retrieved during the exhaustive database search. Meanwhile, most current recommendations on treatment options are based solely on an individual's experience. Hence, specific attention should be paid during application.

The aetiology of avascular necrosis of the metacarpal head may be multifactorial. Potential risk factors need to be explored further. Although uncommon, this review may enhance the awareness and medical management of AVN of the metacarpal head. In the future, more systematic large sample size studies and even multicenter studies are needed, rather than just case reports. Further research is needed to achieve a consensus on AVN treatment.

Conclusions

This study describes the performance and treatment of a rare entities. Although uncommon, Dietrich disease should be considered as a possible diagnosis for unexplained metacarpal pain in the absence of significant studies on routine blood tests and plain film imaging. The aetiology of avascular necrosis of the metacarpal head is unclear. However, the risk factors include trauma, corticosteroid use, SLE, and others. Clinical symptoms vary in severity, ranging from asymptomatic to pain, swelling, and limited movement of the metacarpophalangeal joint. MRI is the most sensitive tool for the diagnosis of AVN of the metacarpal head. An early understanding of this unusual disease will provide an optimal clinical outcome, restoring joint activity, and resolving pain. Nonoperative treatment may be recommended for young people to take advantage of the regenerative potential of growing bones. When nonoperative treatment fails, preservation surgery of the metacarpal head may be attempted in younger patients. Arthroplasty or arthrodesis could be reliable options for older, lower-demand patients. MCP joint denervation may be a new option for treating AVN of the metacarpal head.

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Ethical review and approval were waived for this study, due to the article being a review.

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Conflicts of interest disclosure

The authors declare that they have no conflict of interest.

Research registration unique identifying number (UIN)

1. Name of the registry: not applicable.
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Guarantor

Xiao-Lei Fan, Wen-Tao Wang, and Rui Xiao.

Data statement

The data in this review are not sensitive in nature and are accessible in the public domain. The data are therefore available and not of a confidential nature.

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