

ANSWERS AND ADDITIONAL INFORMATION FOR ORTHOPAEDIC CLINICAL QUIZ

FOOT AND ANKLE DISORDERS

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Quiz 1

- Plain radiograph showed a flat second metatarsal head and widening of the metatarsophalangeal joint (MTPJ) relative to neighboring joints. CT-scan showed central joint depression and multiple loose bodies over the dorsal articulating surface of the metatarsal head.
- Avascular necrosis of the second metatarsal head (Freiberg disease).
- Differential diagnoses include stress fracture of the second metatarsal, synovitis secondary to systemic disorder such as hypercoagulopathy and SLE, chronic septic arthritis and neoplasm.
- Thompson-Hamilton classification (1987) divided the disease according to the degree of vascular insult, giving guidance for treatment. Stage-II disease – severe degenerative joint disease and loss articular cartilage
- For stage-II disease with collapsed metatarsal head, non-operative treatment is rarely successful. With osteophyte causes limitation of the MTPJ motion, a cheilectomy and joint debridement may be considered to eliminate the mechanical block. The procedures that restore articular congruity or address the degenerative features encountered in advanced stages of the disease may be offered. These include dorsiflexion osteotomy metatarsal neck and arthroplasty.

Freiberg Disease

Freiberg disease, first described by Freiberg in 1914, is an osteochondritis of the metatarsal head with predilection to afflict the second metatarsal. Affliction of the third and fourth metatarsals is rare. The second metatarsal epiphysis is vulnerable to repetitive trauma-induced ischemic insults during the early teen years. Its etiology remains unclear but adolescents with altered gait biomechanics due to long second metatarsal are at-risk to develop the disease. Avascular sequence of sclerosis, convex-site articular fragmentation and collapse of the metatarsal head followed by metatarsophalangeal joint incongruity and finally osteoarthritis are typical radiographic features.

It occurs predominantly in adolescence and more common in females than males in 5:1 ratio. The first symptom is pain in the ball of the foot with weight bearing and worsened with activities that require excessive toes dorsiflexion. The involved MTPJ is tender on palpation and painful limitation of dorsiflexion. Swelling may develop due to synovitis.

Treatment decision should be based on the stage of the disease and patient's compliance to therapy. In general, non-operative treatment should be used first especially for early disease with a normal shape MTPJ without radiographic subchondral crescent sign. This includes oral analgesic, activity modification and protected weight bearing using orthoses or shoe wear modification (a stiffened forefoot or rocker bottom sole) to reduce motion of the MTPJ. Surgical options for early disease with non-collapsing metatarsal head include core decompression and osteotomy.

Reference

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Quiz 2

- Congenital macrodactyly of the second and third toes of the right foot.
- Enlargement of the digit secondary to tissue-specific tumours including lipoma, haemangioma, lymphangioma, AVM, neurofibromatosis and fibrous dysplasia.
- A true congenital anomaly is characterized by enlargement of all structures of a digit unit including skin, subcutaneous fat, nerve, vessels, nail and phalanges.
- In foot, excessive proliferation of adipose tissue coupled with absence of any neural involvement. Hand macrodactyly demonstrated predominant involvement of the digital nerve.
- Non-operative treatment in-form of shoe wear modification has poor compliance. Surgery involves multiple procedures and is usually without successful outcome. Debulking procedure involves staged excision of half of soft tissue (skin and subcutaneous fat) and up to a quarter of phalangeal bone from each side usually from the convex side first. It is done on one side of digit at one time and multiple debulks are often needed. Epiphysodesis is performed when digit achieved adult length/size as parents' digits and excessive discrepancy in length may require finger shortening (removal of phalangeal segments). Ray amputation or amputation of the phalanx should be opted as the last salvage procedure for uncontrolled growth. Adjacent digit may have accelerated growth after ray amputation of the other.

Congenital Macrodactyly

Macrodactyly is a rare congenital anomaly manifested as enlargement of the toe or finger present at birth. This deforming lesion is aesthetically displeasing as it always large at birth and the affected digit is angulated and stiff.

It has strong familial predilection with a slight male preponderance. Its occurrence per 10 000 births was estimated to be about 0.2%. Macrodactyly occurred more common to hands than feet. In most cases, only one foot or one hand is involved but more than one digit is involved. Index finger is most commonly affected. In contrast to secondary macrodactyly due to enlargement from tissue-specific tumour (lipoma, hemangioma, lymphangioma, neurofibromatosis, AVM and fibrous dysplasia), true or primary macrodactyly involves all elements of the digit. It co-exists with syndactyly in up to 10% of cases. An unusual variant of macrodactyly associated with lipofibromatous hypertrophy of subcutaneous tissues without direct involvement of the nerve is known as Proteus syndrome.

There are two types of macrodactyly: static type (grows proportionately with the rest of the body at normal rate) and progressive type (grows out of proportion to normal growth of unaffected digits). Foot macrodactyly is more often progressive.

The pathology of macrodactyly displays bizarre involvement of segment of the digit unrelated to the anatomical unit. It involves overgrowth of soft tissue components: skin, subcutaneous fat, nerves and vessels. The skin of the affected digit is markedly thickened and is of rubbery soft in consistency. Changes in phalangeal bones, joint and adjacent tendons and ligaments are secondary effects. The phalanges of the affected digit are longer in length and breadth with abundant fibroblastic tissue between the periosteum and cortex. The bone age of the affected phalanx based on the epiphyseal center is increased when compared to unaffected phalanx.

Surgical dissections have indicated that predominant subcutaneous adult-type fibrofatty tissue infiltration and remarkable digital nerve enlargement are two most striking features of foot macrodactyly whereas tortuous hypertrophied digital nerve with abundant epineural and perineural tissues predominate macrodactyly of the hand. Histologically, the hypertrophied tissues appear as benign-like neurofibromas. With this new knowledge, the correct pathology of macrodactyly might be digital-nerve orientated benign neurofibroma. Most of the pathological tissue bulk (neural and fibro-fatty tissues) is abundant on one side of the digit or adjacent sides of two digits. In a one-sided digital nerve lesion, the hypertrophied tissues will push the un-hypertrophied side away from the mid-line of the digit, giving rise a whole finger appearance of clinodactyly. If the hypertrophy affects the common digital nerve before it division to digital branches on the adjacent sides of the digits, both adjacent sides of two digits will be affected in-form divergent clinodactyly.

Surgical treatment of macrodactyly is often difficult as complications are likely to follow because of inadequate excision of extensive soft tissue pathology and a complete excision requires digital nerve sacrificing procedure. Radical yet intralesional excision of pathologic soft tissues around the digital nerve require longitudinal splitting of the nerve to leave a portion of intact nerve for sensory preservation. Bone shortening with or without excision of one growth plate and arthrodesis, are options for hypertrophic phalanges and joints. When dealing with adjacent two-digit macrodactyly, single digit amputation is contraindicated as accelerated growth of the other digit is highly likely to follow.

Reference

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Quiz 3

- Accessory soleus
- Differential diagnoses of soft tissue mass in the postero-medial region of the ankle include lipoma, ganglion, hematoma, encapsulated haemangioma and synovium.
- Obiteration of the pre-achilles fat pad (Kager triangle) by a subtle soft tissue mass without other significant abnormality. This sagittal T1-weighted image just medial to midline depicted a clearly defined accessory soleus muscle with projection toward insertion site on the calcaneum anteromedial to the insertion of the Achilles tendon.
- Reassurance for asymptomatic mass and non-operative treatment in-forms of orthoses, physiotherapy and activity modification if symptomatic. In cases with severely disabling symptoms affecting daily activities of living, surgical options: fasciotomy, tendon release and even excision or debulking of the muscle may provide symptomatic relief.

Accessory Soleus

The accessory soleus was first described in 1843 by Cruvelhier as an anatomical variant of soleus muscle. According to Petterson et al.(1987), the incidence of the accessory soleus muscle ranges from 0.7 to 5.5%. Recently, Kouvalchouk et al.(2005) estimated that it was present in 10% of all individuals.

The soleus muscle takes origin from the distal posterior aspect of the tibia and deep fascia of the normal soleus or other flexor tendons. It typically inserted via a separate tendon on the calcaneus, anteromedial to the Achilles tendon. The presence of accessory soleus may be assumed for the splitting of the anlage of the soleus early in development. Accessory soleus can be divided into 5 types based on its insertion site onto: i) Achilles tendon; ii) upper surface of the calcaneus with a fleshy muscular insertion; iii) superior surface of the calcaneus with a tendinous insertion; iv) medial aspect of the calcaneus with a fleshy muscular insertion; and v)medial aspect of the calcaneus with a tendinous insertion.

The accessory soleus is generally enveloped within its own fascia and derives its blood supply from a single artery, the posterior tibial artery. The posterior tibial nerve supplies both the soleus proper and the accessory soleus muscle.

Although it is congenital lesion in origin, itsusual manifestation in the second and third decades of life may be related to an increase in muscle mass. However, its association with resistant clubfoot or equinus deformity has been documented in three case reports. The clinical symptoms vary from painless ankle swelling (25%) to pain and swelling about the ankle (67%). The pain generator in symptomatic accessory soleus is assumed to be ischemic origin from exercise-induced compartment syndrome or compression of tibial nerve within the tarsal tunnel. As such, the appropriate surgical treatment depends on findings at exploration. If accessory soleus is associated with tarsal tunnel syndrome, debulking or excision of the anomalous muscle may relieve the foot pain. Patient with chronic exercise-induced compartment syndrome may benefit from simple fasciotomy.

References

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Quiz 4

- a. Fracture-dislocation of the talo-navicular joint with dissociation of Lisfranc joint and early fragmentation of the left navicular.
- b. The above features are consistent with a diagnosis of bilateral acute (Eichenholtz stage-I) Charcot neuroarthropathy of the Lisfranc joints (Sander-Frykberg type-II) and Schon et al. type-B deformity.
- c. Full blood count is useful to indicate septic arthritis or chronic osteomyelitis as differential diagnoses or co-existing lesion. PET scans is the best imaging study to demonstrate both conditions. Semmes-Weinstein test using monofilament 5.07 helps to identify peripheral neuropathy with loss of protective sensation. HbA1c is important to monitor glycemic control and assessing suitability for surgery. However, the best confirmatory investigation remains synovial biopsy (showing synoviocytes and macrophages containing cartilage debris).
- d. Charcot neuroarthropathy is classified based on the course of the disease (staging) and the location of the destruction. Charcot changes typically occur in 3 stages (Eichenholtz, 1966) based on radiological findings:
Stage-I (stage of development), initiation of the fragmentation process.
Stage-II (stage of coalescence), the active period of the disease process when bony destruction and deformity occur.
Stage-III (stage of reconstruction), commencing when the destructive process 'burns out' with sclerotic bone.
The location of joint and bone destruction is best classified according to Sander-Frykberg system (1993). Type-I toes and distal metatarsals, type-II tarsometatarsal joints (Lisfranc joint), type-III Chopart joint including naviculo-cuneiform joints, type-IV ankle and type-V subtalar joint and calcaneus.
- e. The primary aims of treatment of Charcot foot are to prevent a permanent change to the shape of the foot by creating a stable, well-aligned plantigrade foot amenable to bracing or shoe wear and reduction of pressure points, and preventing future foot problem: ulceration, infection and amputation.

Charcot Foot

Charcot neuroarthropathy remains a mysterious self-limiting disease of unknown pathogenesis. Neuropathy is the main trigger and it well-documented link with diabetes mellitus and peripheral neuropathy speculates the French neuropathic theory and the German vascular theory of Charcot pathogenesis. Disturbance in the microvascular circulation caused by autonomic neuropathy and repeated microtrauma alter the integrity of bone and joint of the foot with recent evidences indicating different activation of regional bone metabolism via RANK-L complex interfering the function of osteoclasts.

Charcot foot is a multifaceted disease with one end patients have severe deformation, ulcer, infection and life-threatening sepsis, and at another end patients with typical stable end-stage disease unknowingly continue to wear normal shoes. In the worst scenario, it may be difficult to differentiate acute Charcot and infected Charcot. A swollen, red, warm and often painless foot in an insensate diabetic with mild inflammatory signs is rather an acute Charcot than an infection. In unilateral disease, local skin temperature difference of 2°C indicates an acute Charcot. Full blood count and C-reactive protein are non-specific parameters. Imaging studies: MRI and PET scans are expensive. Acute trivial fracture-dislocations of the foot or ankle in insensate patients are potentially worst scenarios as they need more stable fixation constructs and a longer post-operative off-loading period to prevent surgical-triggered Charcot reaction.

Classification of Charcot foot should ideally incorporate a combination of staging, location of the disease, and information related to joint stability, status of soft tissue ulcer and/or infection status and vascular status. The course of Charcot foot is staged into three radiological stages: stage-I stage of development, stage-II stage of coalescence and stage-III stage of reconstruction (Eichenholtz, 1966). Today, with MRI assistance, stage-0 (stage of beginning) is seen as bone bruise or marrow edema. This classification helps to decide option for treatment with stage-0 and –I needing non-operative treatment to prevent joint and bone damage, and preclude unnecessary surgical intervention as surgery may aggravate Charcot reaction during stage-I disease. Of recent interest, drug therapy to accelerate conversion of early stages to stage-III disease may help to reduce bony destruction and foot deformity. The Sander-Frykberg classification (1993) is based on anatomical location of the affected joint and bone. It helps the surgeon to decide on type of reconstructive option. For Charcot lesions of the Lisfranc and Chopart joints, Schon et al.(1998) described destruction patterns into type-A (normal plantar arch), type-B (flat foot) and type-C (rocker bottom foot) to guide treatment decision. Type-A and –B can be treated conservatively if the joint of the foot is stable. Type-C foot will eventually ulcerate by the so-called internal decubitus and the foot is mechanically unstable due to breakdown of the arch. For type-C foot, surgical interventions: bony correction and stabilisation, are needed.

The primary treatment is non-surgical by aiming for a plantigrade and stable foot with permanent change to the shape of the foot. The only effective way to achieve these is to detect individual at-risk with stage-0 and bring them to stage-III disease by off-loading the affected foot and protect the bones of foot from damage. This requires some form of cast immobilization including total contact cast or protective orthoses such as Charcot Restraint Orthotic Walker (CROW) or even surgical off-loading procedures: excision of offending bony prominence or Achilles tendon lengthening, whenever appropriate. Parenteral bisphosphonate, pamidronate has been demonstrated to alter the natural history of the disease by accelerating conversion to stage-III disease through deactivation of osteoclast activity. While many clinicians have used oral bisphosphonates with unpublished anecdotal success, the United States Food and Drug Administration (FDA) have not approved oral bisphosphonates. Similar anecdotal reports of success have also been reported with the use of magnet therapy.

The role of elective surgery is to address long-term deformities includes excision of any pathological bony prominence that could cause pressure points and where feasible to perform reconstructive procedures to correct any residual bony deformities. Some authors however have reported success in early reconstructive surgery to stabilise the affected joints and in so doing prevent the development of deformities and its associated complications.

Dealing with infected Charcot foot remains a challenge as all sorts of surgery, antibiotics and selection of treatment options may be considered to justify the rule life before limb in patients with insensate foot. Surgical debridement and subsequent second look debridement may eventually serve as creeping amputation. Following control of infection with adjunctive antibiotics and staged wound dressing and closure, reconstruction phase is usually recommended with positioning of the foot is best controlled by the external or internal fixation. This often requires expensive circular frame external fixator (either Ilizarov construct or Taylor-spatial frame) or region-specific anatomical plate or interlocking nail. Secondary procedure to enhance positioning of the foot including lengthening of the Achilles tendon may be needed.

Reference

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