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Overview of tremor

EDITORIAL

CONTENTS

Dear Doctor,

Wish you Happy New Year 2020!

As we welcome this new year, we take this opportunity to thank you for your love and support. Without you we cannot imagine our success. Its been a pleasure serving you over seventeen years, and we are looking forward to your continued support in the year ahead.

We have tried our best to provide you with necessary information on different topics of medical science through Info Medicus. This issue is unique as we have introduced two freshly brewed concepts. One of them is Clinical answer, where you will get an overview of recent frequently asked questions and their answers and the other one is the management of heel pain in Health care.

Pain is a distressing and unpleasant feeling that creates hindrance in our day to day activity. Therefore, we have selected diagnosis and management of heel pain in Health care. Closed reduction is preferred in case of fracture and dislocation where possible. For this, we have selected closed reduction of fractured and dislocated ankle as our topic in Essential procedure.

Tremor is very common among people especially among older age group. We have highlighted "Overview of tremor" as our Review article. We hope, you will get concise information about it from the article. A recent topic on heart is included in our Current health section, which you may find interesting.

We need your suggestion to serve you better and hope that we become integral part in your professional success and wish you health and happiness in the coming year.

Thanking you and warm regards,

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Heel pain: diagnosis and management

Introduction

Heel pain is a general term used to describe pain and discomfort experienced anywhere in or around the rear of the foot. Varieties of names are used to describe heel pain such as plantar fasciitis, tennis heel, jogger's heel, policeman's heel. Heel pain is a very common foot disease that may cause significant discomfort and disability. The patient frequently complains of pain on the posterior aspect of the calcaneus at the insertion of the Achilles. Other less common causes of heel pain, which should be considered when symptoms are prolonged or unexplained, include osteomyelitis, bony abnormalities such as calcaneal stress fracture or tumor. Heel pain rarely is a presenting symptom in patients with systemic illnesses but the latter may be a factor in persons with bilateral heel pain, pain in other joints or known inflammatory arthritis conditions.

Cause of heel pain

There are many causes of heel pain that can induce the mild to moderate chronic severe heel pain. Biomechanic factors are the most common etiology of heel pain. Other causes include neurologic, injury related, arthritic, infectious, neoplastic, auto immunological and other systemic conditions (Table-1). The anatomic location of the pain can be a guide to diagnosis (Figure-1). Examination should include inspection of the foot at rest and when weight bearing as well as palpation of bony prominences, tendon insertions and the foot and ankle joints. Any tenderness, defects or differences between the sides should be noted. Active range of motion of the foot and ankle should be assessed; if full range of motion is not present, passive range of motion should also be evaluated. Specific testing as detailed throughout this article, will also help to determinate the diagnosis.

Cause of plantar heel pain

Plantar fasciitis: Plantar fasciitis occurs when the plantar fascia ligament of the foot spanning the arch of the foot to induce the excessively inward rolling with either over flexion or stretching. The ligament becomes irritated and inflamed and small tears may develop in the tissue. The typical patient is between 40 years and 60 years old but it occurs in the earlier age of the joggers whose incidence is as high as 10%. Because of the high incidence in the joggers, it is the best postulated to be caused by recurrent minor injury. Several risk factors have been identified including occupations that involve in

Table-1: Cause of heel pain		
Plantar heel pain	**Posterior heel pain**	**Midfoot heel pain**
• Plantar fasciitis • Stress fractures • Heel neuritis • Heel Pad Syndrome • Plantar warts	• Achilles tendinopathy • Haglund's deformity (heel bump) • Sever's disease (calcaneal apophysitis)	• Tendinopathies • Tarsal Tunnel Syndrome • Sinus Tarsi Syndrome

excessive walking or standing, poorly cushioned footwear, obesity and running for exercise or competition. Patients who are excessive pronators (pes planus) or have reduced ankle dorsiflexion is also at a higher risk of developing plantar fasciitis. When plantar fasciitis is bilateral, it is associated with rheumatologic conditions such as rheumatoid arthritis, systemic lupus erythematosus and gout.

Stress fractures: Stress fractures of the foot and heel commonly occur in athletes or long distance runners who increase their running mileage over a short period of time. Repeated stress on the heel bone eventually leads to a break. Other factors that increase a person's chance of developing a stress fracture include: osteopenia, anorexia or bulimia and infrequent or absent monthly periods. A stress fracture causes significant pain that intensifies with activity and improves with rest. In addition to pain, swelling may be present along with tenderness felt in the area of the bone break.

Heel neuritis: Compression of a small nerve (a branch of the lateral plantar nerve) can cause pain, numbness or tingling in the heel area. In many cases, this nerve compression is related to a sprain, fracture or varicose (swollen) vein near the heel.

Heel Pad Syndrome: Heel Pad Syndrome is due to thinning of this fat pad that results from trauma, such as the consistent pounding of the foot in marathon runners or pressure put on the foot due to obesity. This causes a deep, aching pain felt in the middle of the heel that worsens with weight bearing activity.

Plantar warts: Plantar warts which are raised, skin lesions resulting from infection with the human papilloma virus, can cause heel pain. Lesions can be noted on inspection of the heel and may be tender to palpation. They are usually self limited; however, patients often desire quicker resolution.

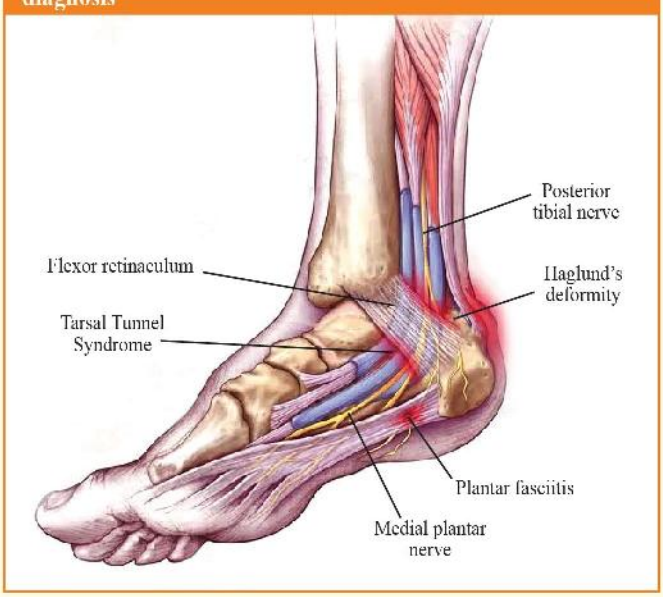
Cause of posterior heel pain

Achilles tendinopathy: The Achilles tendon constitutes the distal insertion of the gastrocnemius and soleus muscles into the calcaneus. It is the inflammatory process within the tendinous insertion of the Achilles. This condition also refers to Achilles tendonitis, paratenonitis (acute disease), tendinosis (chronic disease), tenosynovitis, peritendinitis and achillodynia. The acute phase of Achilles tendinopathy is secondary to acute over exertion, blunt trauma or chronic overuse and muscle. Achilles tendinitis is generally caused by overusing the affected limb, which refers to the repeated stress and strain and is similar to the case in endurance runners. Overusing can simply mean the increase of running, jumping or plyometric exercise intensity in a very short time. The most common theories are based on physiological, biomechanical and extrinsic properties (e.g., footwear or training types).

Haglund's deformity (heel bump): Heel bumps or exostoses occur just lateral to the Achilles tendon and cause particular worry to teenagers in whom they interfere with shoes wear. The soft tissue near the Achilles tendon becomes irritated when the bony enlargement rubs against shoes. This often leads to painful bursitis which is an inflammation of the bursa that produces the redness and swelling associated with Haglund's deformity. The bumps appear about the age of 11 and usually stop hurting when growth is completed.

Sever's disease (calcaneal apophysitis): Sever's disease, also known as calcaneal apophysitis, is an inflammation of the growth plate in the heel of growing children, typically adolescents. The condition refer to the pain in the heel and is caused by recurrent stress to the heel and thus is particularly common in active children. It usually resolves once the bone has completed growth or activities reduce. Sever disease (calcaneal apophysitis) is the most common etiology of heel pain in children and adolescents, usually occurring between 5 years to 11 years old. Sever's disease is directly related to

Figure-1: The common sites of heel pain with corresponding diagnosis



overuse of the bone and tendons in the heel. This can come from playing sports or anything that involves a lot of heel movement. It can be associated with starting a new sport or the start of a new season. Children who are going through adolescence are also at risk of developing it because the heel bone grows quicker than the muscles and tendons in these patients. Too much weight bearing on the heel can also cause it, as can excessive traction since the bones and tendons are still developing. It also occurs more commonly in children who over pronate and involves both heels in more than half of patients. Radiography is usually normal and therefore does not aid in the diagnosis but may reveal a fragmented or sclerotic calcaneal apophysis.

Cause of midfoot heel pain

Tendinopathies: Other less common tendinopathies can cause heel pain localized to the insertion site of the affected tendon. Medial heel pain may be triggered by the posterior tibialis, flexor digitorum longus or flexor hallucis longus tendon. Lateral heel pain can originate from the peroneal tendon. Ultrasonography of these tendons may aid in the diagnosis.

Tarsal Tunnel Syndrome: It is a nerve condition in which a large nerve in the back of the foot becomes pinched. Tarsal tunnel pain which described as aching or burning, may be felt in the heel but is more common in the bottom of the foot and near the toes. Similar to carpal tunnel syndrome in the hand, numbness and tingling may be present and the pain is often worse at night.

Sinus Tarsi Syndrome: The Sinus Tarsi, referred to as "the eye of the foot" refers to the space on the outside of the foot between the ankle and heel bone. This space while small, contains several ligaments as well as fatty tissue, tendons, nerves and blood vessels. Rolling out the ankle often triggers this syndrome which may lead to pain with weight bearing activities, a sensation of ankle looseness and difficulty walking on uneven surfaces like grass or gravel.

Diagnosis

The diagnosis is mostly based on clinical examination. Normally, the location of the pain and the absence of associated symptoms indicating a systemic disease strongly suggest the diagnosis. Diagnosis of heel pain may involve patient presenting a history of symptoms, physical examination and ordering appropriate imagining studies if indicated (e.g., x-rays, ultrasound and MRI). Sometimes further diagnostic test like blood analysis test is needed.

Physical examinations: The physical examination must include examination of the patient's foot at rest and in a weight bearing posture. A visual examination of the foot may show swelling, bony deformities, bruising or skin tear. Bony prominences and tendinous insertions close to the heel and midfoot should be palpated for observing any tenderness or palpable deformity. Passive range of motion of the foot and ankle joints should be examined for evidence of restricted movement. Also the foot posture and arch formation should be visually examined while the patient is bearing weight; the physician must look for abnormal pronation or other biomechanical irregularities. Observing of the foot when the patient is walking, to identify gait abnormalities that provide further diagnostic evidence.

Radiology: Imaging may demonstrate calcaneal spur formation or calcification at either the insertion of the Achilles tendon or the origin of the plantar fascia. Alternatively, the posterosuperior aspect of the calcaneus may be over prominent and protrude into the Achilles tendon, a condition known as "Haglund deformity" with the symptoms coming from the inflamed retrocalcaneal bursa. A bone scan sometimes reveals increased activity diffusely about the calcaneus, as may be seen in systemic diseases such as Reiter's syndrome or a discrete area of uptake as in a stress fracture. MRI may help delineate the degree of the Achilles tendon degeneration present in cases of Achilles tendinosis and can help identify rupture of the tendon if this is in question.

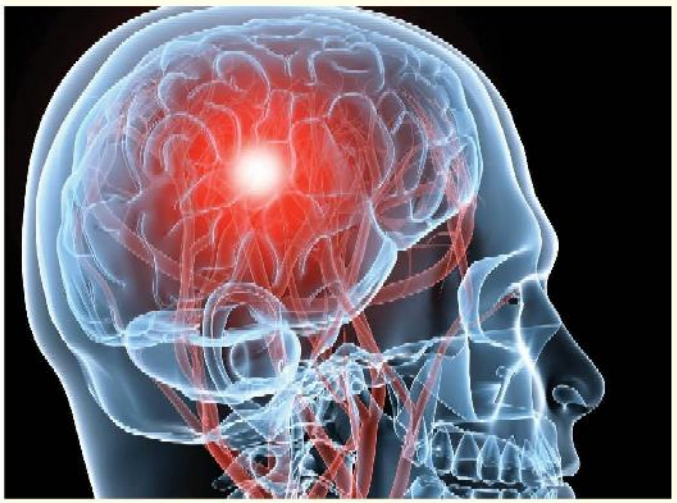
Treatment

Early treatment might involve oral or injectable anti-inflammatory medication, exercise and shoe recommendations, taping or strapping or use of shoe inserts or orthotic devices. Taping or strapping supports the foot, placing stressed muscles and tendons in a physiologically restful state. Physical therapy may be used in conjunction with such treatments. A functional orthotic device may be prescribed for correcting biomechanical imbalance, controlling excessive pronation and supporting the ligaments and tendons attaching to the heel bone. It will effectively treat the majority of heel and arch pain without the need for surgery. Only a relatively few cases of the heel pain require more advanced treatments or surgery. If surgery is necessary, it may involve the release of plantar fascia, removal of a spur, removal of a bursa or removal of a neuroma or other soft tissue growth.

Conclusion

There are many treatment options that exist when dealing with heel pain but there is lack of evidence to show which one is the most effective. Each doctor has a different treatment method. As we don't know yet which treatment works best, upgrading to better, more supportive shoes should be the important first line treatment option.

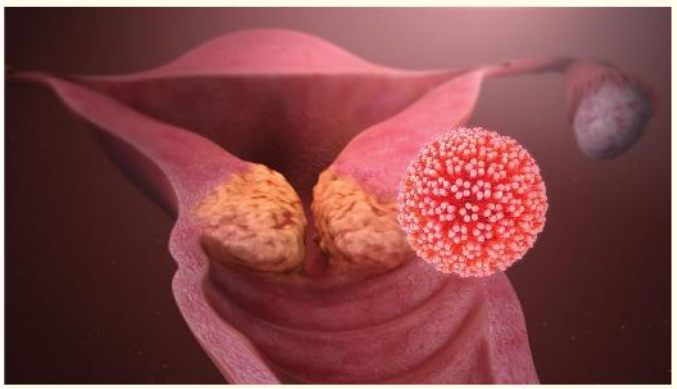
References: 1. Chin. Jour. of Traum., 2015, Vol. 18:164-169
2. Am. Fam. Phys., 2018; 97(2):86-93
3. www.verywellhealth.com/heel-pain
4. www.apma.org/heelpain



What is the role of imaging in mild traumatic brain injury?

Head computed tomography should not be routinely performed to assess patients with mild traumatic brain injury. Imaging should be used only to eliminate concerns of more significant injuries and not for evaluation of uncomplicated concussion.

Reference: Am. Fam. Phys., 01 October 2019; 100(7):400



When screening for cervical cancer should be stopped?

Evidence from randomized clinical trials and decision modeling studies suggests that screening beyond 65 years of age in women with adequate screening history would not have significant benefit. The current guidelines by the American Cancer Society, American Society for Colposcopy and Cervical Pathology and American Society for Clinical Pathology define adequate screening as three consecutive negative cytology results or two consecutive negative high risk human papilloma virus results within 10 years before stopping screening, with the most recent test performed within five years. The United States Preventive Services Task Force recommends against screening for cervical cancer in women older than 65 years who have had adequate prior screening and are not otherwise at high risk for cervical cancer.

Reference: Am. Fam. Phys., 15 September 2019; 100(6):331

What is the effective treatment for mild dehydration in children with gastroenteritis?

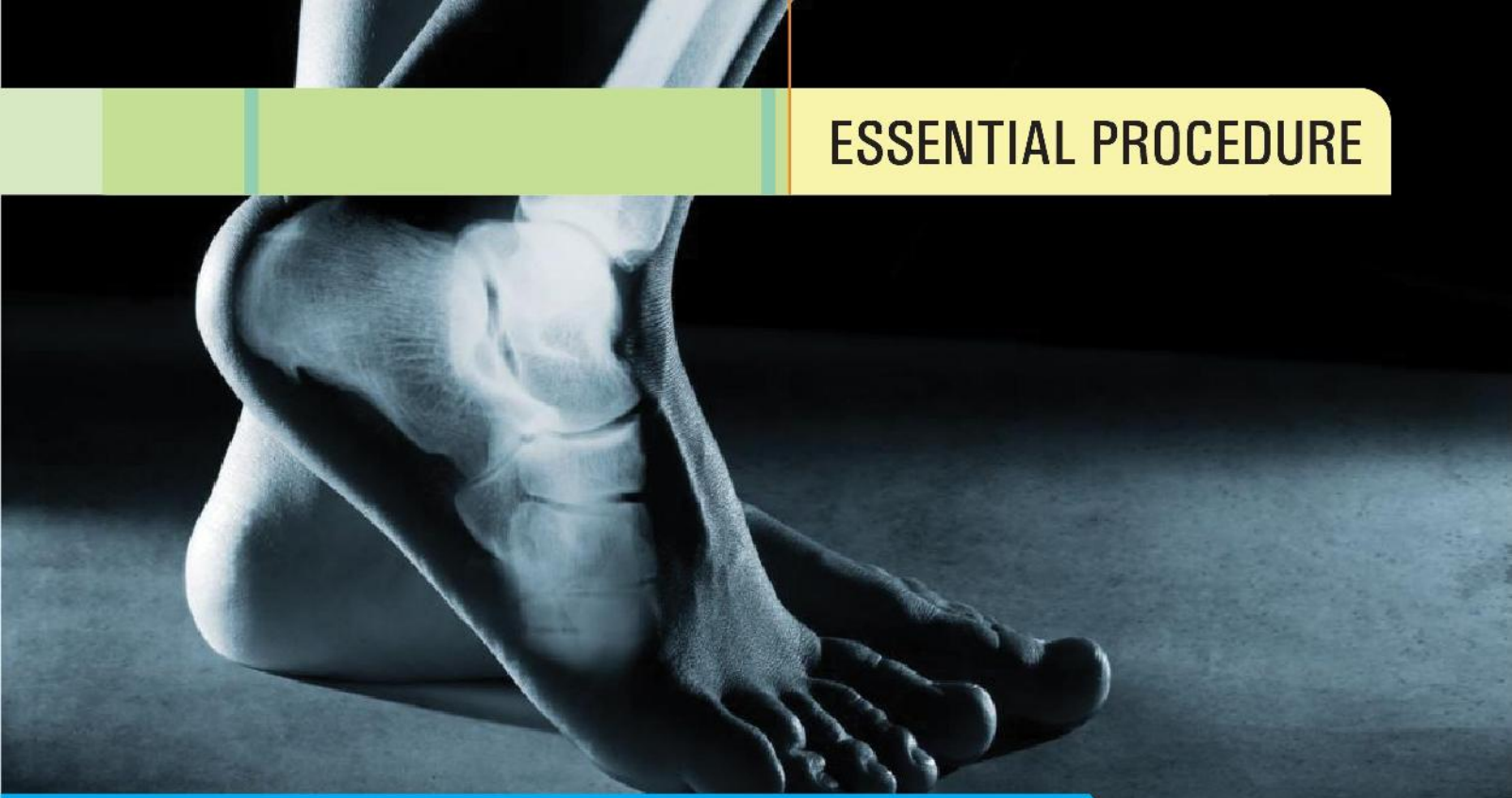
The specific electrolyte composition of oral rehydration solution is not important for mild dehydration. Half strength apple juice followed by preferred fluids (e.g., regular juices and milk) is an option for mild dehydration, because a study found this approach reduced the need for eventual intravenous rehydration compared with a formal oral rehydration solution.

Reference: Am. Fam. Phys., 01 September 2019; 100(5):273

Is manipulative therapy effective for back and neck pain?

Spinal manipulative therapy may be considered for patients with acute low back pain to provide modest improvement in pain and function at up to six weeks, comparable with other therapies. It may also be considered for patients with chronic low back pain. It results in modest improvement in pain and function for up to 6 months and is comparable with other therapies. Cervical manipulation and mobilization may be considered to provide short term improvement in pain relief and function in patients with neck pain.

Reference: Am. Fam. Phys., 15 August 2019; 100(4):208



Closed reduction of a fractured and dislocated ankle

Overview

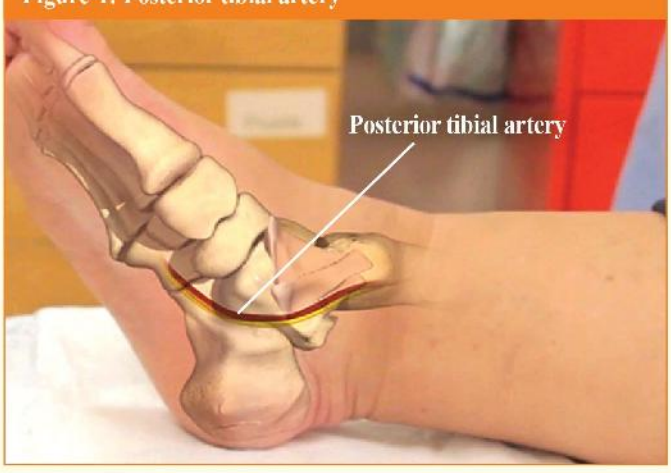
Ankle fractures are common injuries that may occur at any age and that typically result from a twisting or rotational injury. Prompt reduction of a displaced fracture is important in order to decrease the risk of trauma to the skin and soft tissues surrounding the ankle joint. Definitive management of displaced fracture of the ankle often involves surgical reduction and fixation; this review focuses on initial management. The use of a hematoma block for anesthesia is also discussed and techniques for reducing and splinting a displaced fracture of the ankle are reviewed.

Anatomy

The ankle forms a mortise joint, with the talus fitting snugly into a groove formed by the tibia and the fibula. Ankle stability results from the interplay among the articulations involving the talus, tibia and fibula and the medial and lateral ligaments of the ankle. The bony portion of the ankle joint consists of the distal flare of the tibia and the fibula. The medial malleolus has little soft tissue coverage and the skin covering the medial ankle is at particular risk for damage after a displaced fracture of the ankle has occurred. The ankle joint is medially constrained by the deltoid ligament and laterally constrained by the distal fibula and lateral ankle ligaments.

The most important neurovascular structures around the ankle including the posterior tibial artery (Figure-1) and tibial nerve, run posterior to the medial malleolus. These structures are at risk for damage when there is substantial displacement of the ankle. The anterior tibial artery, lies on the anterior aspect of the ankle, courses distally to become the dorsalis pedis artery. The deep and superficial branches of the peroneal nerve also cross the anterior ankle. Typically the anterior structures are not at risk after an ankle fracture, but these structures may be injured in patients with higher energy fractures of the tibial plafond. The medial and lateral malleoli and the posterior calcaneus are usually palpable, even in a grossly displaced fracture of the ankle or in dislocation. These surface landmarks are useful during reduction maneuvers.

Figure-1: Posterior tibial artery



Indication

Primary indication for performing closed reduction (e.g., reduction without surgery) of a displaced fracture of the ankle is subluxation of the talus from under the tibial plafond. A clinically significant deformity can put pressure on the overlying skin and soft tissues. An unreduced ankle fracture that threatens the integrity of the skin can lead to serious complications, including necrosis of the overlying skin and infection. Prompt reduction and splinting of the fracture can prevent such complications and decrease pain, making patients more comfortable as they await definitive treatment.

Equipment

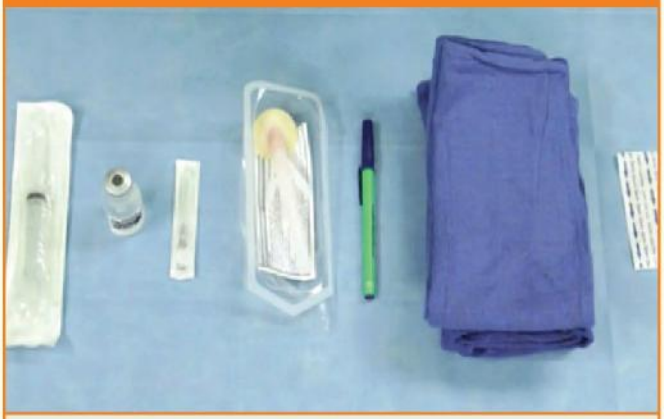
The equipment required for splinting includes:

- Non sterile water
- Non sterile gloves
- A non sterile gown, a cap and a face shield
- Splinting material includes a 10 cm plaster U-slab with 10 layers to 12 layers, a 10 cm or 15 cm plaster posterior slab with 10 layers to 12 layers and adequate padding, such as cotton under cast padding material. The under cast padding material can be placed on the splinting material or applied to the patient when the ankle is reduced
- A 10 cm or 15 cm elastic bandage that will be used to wrap the splint is also needed
- If plaster slab material is unavailable, premade fiber glass slabs can be used with nonsterile water
- For a closed reduction hematoma block, nerve block and conscious sedation with intravenous narcotic medication should be available
- A local hematoma block has been shown to be equivalent to intravenous sedation in providing anesthesia for ankle reductions
- The equipment needed for a hematoma block includes a sterile 10 ml syringe that is prefilled with sodium chloride and 1% lidocaine, a sterile 22 gauge needle, an antiseptic solution that contains chlorhexidine or povidone iodine, a sterile marker, sterile towels and a self adhesive bandage (Figure-2)

Patient preparation

Explain the procedure and its benefits and risks to the patient and obtain written informed consent. Ask the patient if he or she is allergic to any medications or skin preparation solutions. It is important to take universal precautions during the examination, the administration of the hematoma block and the performance of the reduction maneuver. All medical personnel present should wear gloves, gowns, caps and face shields. Begin by thoroughly examining the patient. Inspect the skin for any abrasions or tears that might indicate an open fracture. Open fractures require urgent surgical treatment to decrease the risk of infection. Next, assess the neurovascular status of the affected leg.

Figure-2: Equipment for hematoma block



The equipment needed to perform a hematoma block includes a sterile 10 ml syringe with sodium chloride and 1% lidocaine, a 22 gauge sterile needle, antiseptic solution, a sterile marker, sterile towels and a self adhesive bandage

First evaluate the sensory function of the peroneal, saphenous, sural and tibial nerves and then the skin color and the quality of capillary refill. Palpate the posterior tibial and dorsalis pedis arteries for pulses. Vascular compromise may result from a clinically significant deformity of the ankle. Reduction often improves vascular status. If vascular compromise is still suspected after reduction is performed, an urgent evaluation by a vascular or an orthopedic surgeon is required. If it is appropriate to proceed with fracture reduction, place an absorbent pad underneath the patient's ankle. Wash off any dirt or particulate matter from around the ankle.

Hematoma block

To perform a hematoma block, first identify the proper location at which to insert the needle. Locate the space that is bordered laterally by the anterior tibial tendon and medially by the anterior aspect of the medial malleolus. A palpable soft spot is often present. Mark the location with a sterile marker and then clean the skin with antiseptic solution. Attach the sterile 22 gauge needle to the prefilled syringe. Pierce the skin at an angle that is approximately 30 degrees from the horizontal plane and 30 degrees from the sagittal plane. If the bone of the tibia or talus is encountered, the needle should be retracted and redirected into the joint space.

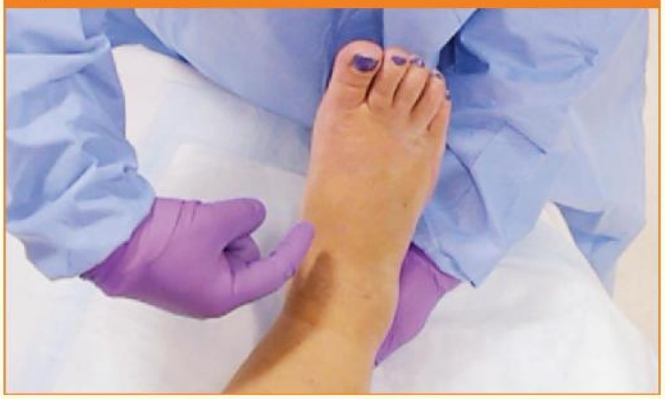
Aspirate a small amount of the fracture hematoma to confirm the intra-articular location, since most ankle fractures result in the formation of an intra-articular hematoma. Inject the solution into the fracture hematoma. Dress the puncture site with a self adhesive bandage. Approximately 10 minutes should be waited for the anesthetic agent to take effect.

Ankle reduction

The patient should be positioned so that the affected leg is placed at approximately 45 degrees of hip flexion and approximately 45 degrees

of knee flexion. This position facilitates reduction by relaxing the pull and deforming force of the gastrocnemius muscle. To perform the reduction maneuver, grasp the calcaneus and hind foot. If the ankle is fully dislocated, axial traction is used to pull the talus into position underneath the tibial plafond (Figure-3).

Figure-3: Ankle reduction



The most common fracture pattern involves lateral subluxation of the talus. The talus and hind foot resume their correct positions once the talus is partially under the tibial plafond. Hold the hind foot firmly in one hand, with the butt of the other hand at the level of the tibial plafond. Apply varus and medial force to the hind foot to slide the talus into its correct position. It may help to internally rotate the midfoot when pressure is applied. For cases in which medial subluxation of the talus is present, apply direct lateral force to reduce the talus and hind foot under the tibia. Once it appears that the reduction has been successful, Quigley's maneuver can be performed to keep the ankle in the reduced position.

To perform Quigley's maneuver, simply hold the big toe to maintain the varus and medial moment on the hind foot. This technique is primarily used for fractures that involve either lateral or posterior translation of the talus with regard to the tibia. The primary goal of reducing a displaced fracture of the ankle is to decrease the effect of the injury on the skin and soft tissues. As long as the skin is not threatened, an imperfect reduction may suffice until definitive treatment can be provided.

Once the ankle fracture have reduced, the splinting material can be dipped in water at room temperature and the posterior slab of the splint applied (Figure-4). The posterior slab should extend from just beneath the popliteal fossa to approximately 3 cm beyond the big toe. Wrap the slab with a single layer of cotton padding material to maintain its position. Next, apply the U-slab. Ensure that the slab covers the lateral and medial malleoli. An additional layer of padding is useful to hold the splinting material in position and to prevent the plaster from sticking to the elastic bandage. Wrap these slabs very loosely with an elastic bandage. It is crucial to avoid stretching the elastic bandage as it is applied. Stretching the bandage can cause pain and may even cause compartment syndrome. It is also important to splint the ankle in a neutral position to maximize stability and avoid

Figure-4: Application of the posterior slab to the ankle

contracture of the Achilles' tendon. Once the ankle is encased in the splinting material, gently mold the material to maintain the position. With one hand, apply gentle pressure around the distal calf, just proximal to the injury and with the other hand, cup the hind foot and apply gentle pressure to preserve the position until the plaster material hardens. Be sure to apply an even amount of pressure; any uneven areas can lead to pressure sores. To avoid the creation of undue pressure points on the underlying skin, apply pressure with the palms of hands rather than fingers. Take particular care when treating elderly patients, in whom pressure points can lead to major skin ulcerations. Hold the mold in place while the splinting material hardens. This process may take 5 minutes to 10 minutes. Once the material has hardened, place the injured ankle on several pillows and apply ice to help control edema. Instruct the patient to avoid bearing any weight on the affected leg until the injury has been assessed by an orthopedic surgeon.

Complication

Once the reduction is complete, it is important to reassess the neurovascular status of the affected leg. Any neurovascular compromise may lead to severe complications. Instruct the patient to avoid bearing any weight on the affected leg until the injury has been assessed by an orthopedic surgeon. In elderly patients, be aware of friable skin, since traction may lead to large abrasions. The use of hot water or the application of too many layers of plaster can induce burns. An incomplete reduction or a loss of reduction can cause skin damage. It is also important to use sufficient padding, especially around the bony prominences, to avoid skin breakdown.

Summary

Displaced ankle fracture is a common injury that can cause clinically significant pain and skin complications. Knowledge of the basic reduction technique is essential to decrease the risk of these complications.

Reference: N. Eng. J. Med., 19 September 2019, Vol. 381(12):e25

Development of an abdominal wall abscess caused by fish bone ingestion

Background

Foreign body ingestion sometimes occurs accidentally and is usually related to food, such as fish and chicken bones and includes ingestion of toothpicks. Most foreign bodies pass uneventfully through the gastrointestinal tract within a week. In a small percentage of cases, a foreign body impacts, penetrates or perforates the gastrointestinal tract. Endoscopic intervention is required in 10% to 20% of patients and surgical intervention to remove a foreign body is required in less than 1% of patients. Therefore, an abdominal wall abscess can develop in association with an ingested foreign body, such as a fish bone. Moreover, the laparoscopic approach for resecting a portion of bowel containing a foreign body is a useful option for selected cases.

Case presentation

A 55 year old man presented to the hospital with a complain of right lower abdominal pain. His medical and family histories were unremarkable. He worked in a factory. He occasionally consumed alcohol and smoked cigarettes. He denied having eaten fish during the previous few days. Three days prior to visiting the hospital, he noticed redness of the skin and pain involving his right lower abdomen. A physical examination revealed tenderness, swelling and redness at the right iliac fossa; however, he was afebrile (36.5 °C). His blood pressure and pulse were 122/80 mmHg and 85 beats per minute (bpm), respectively. A laboratory examination revealed an increased white blood cell (WBC) count of 10.4×10^3 cells/μl and C-reactive protein (CRP) level of 10.19 mg/dl. Except for this finding, laboratory testing revealed no abnormal values. Computed tomography (CT) showed a 42 × 22 mm low density area with rim enhancement in his right internal oblique muscle and a 20 mm long hyperdense, sharply pointed object in the wall of his cecum adjacent to the low density area. Although he was unaware of having ingested a sharply pointed object such as a fish bone, the object was suspected as a fish bone because of the shape. Thus, the findings were diagnosed as abdominal wall abscess due to a foreign body piercing the cecum. The abscess

was aspirated, but did not return fluid. A blood culture had no growth. He was treated conservatively with cephalosporin (2 g/day) for 2 weeks. After the treatment, his WBC and CRP level returned to normal and the abdominal wall abscess was not seen on CT. His symptoms of tenderness, swelling and redness at the right iliac fossa also improved. The hyperdense pointed object remained in the same location. Therefore, 24 days after the diagnosis, he underwent colonoscopy for removal of the object. However, no abnormality was visualized in his cecum. Although surgical removal was indicated, the patient was monitored carefully without performing surgery, based on a mutual agreement with the patient.

Two months after the initial treatment, he presented again with right lower abdominal pain. CT showed the same low density area and the foreign body in the same location in the cecum; the findings were diagnosed as recurrence of the abdominal wall abscess due to the foreign body. Laparoscopic surgery was performed to remove the object. He was treated with cephalosporin (2 g/day) for 7 days before laparoscopic surgery and a subsequent laboratory examination revealed that his CRP level had decreased from 8.57 mg/dl to 0.82 mg/dl. When he presented immediately before undergoing surgery, he no longer complained of right lower abdominal pain.

The laparoscopic findings included fibrous adhesions between the cecum, tip of the appendix and right parietal peritoneum. The resection lines were based on the preoperative CT and the laparoscopic findings. First, the dissection was done at the appendix at its origin, then dissection was done in the adherent section of the cecum and then performed an en bloc resection of the foreign body with parietal peritoneum. Finally, removal of the foreign body was confirmed by a plain X-ray examination of the resected specimen. Then a percutaneous drain was placed in the abscess. A mucosal lesion was seen neither in the cecum nor the appendix of the resected specimen and most of the object was located in the wall of the cecum, except for the pointed tip, which had penetrated the tip of the appendix. The object was identified as a 2 cm long fish bone. These findings suggest that the fish bone pierced the cecal wall and then migrated within the cecal wall. After surgery, the patient was treated with cephalosporin (2 g/day) for 4 days and he had an uneventful postoperative course and was discharged on postoperative day 10. One year after surgery, he is doing well without recurrence of the abdominal wall abscess.

Discussion

Foreign body ingestion is usually related to food, with fish bones frequently being ingested accidentally, especially among populations that frequently eat fish. Ingested fish bones may be forgotten and there can be a time lag of months or even years between ingestion and the onset of symptoms. In previous reported cases of abscess formation due to fish bone ingestion, most patients denied any history of fish bone ingestion. Therefore, a clinical history might not be helpful. Moreover, fish bone complications in the gastrointestinal tract manifest with a variety of clinical presentations, ranging from impaction in the upper gastrointestinal tract, dysphagia, bowel obstruction and silent perforation to frank peritonitis. Peritonitis can be acute or chronic. However, only a few cases of abdominal wall abscess due to fish bone ingestion have been reported. Therefore, the medical history and clinical presentation alone do not provide information suggestive of fish bone ingestion.

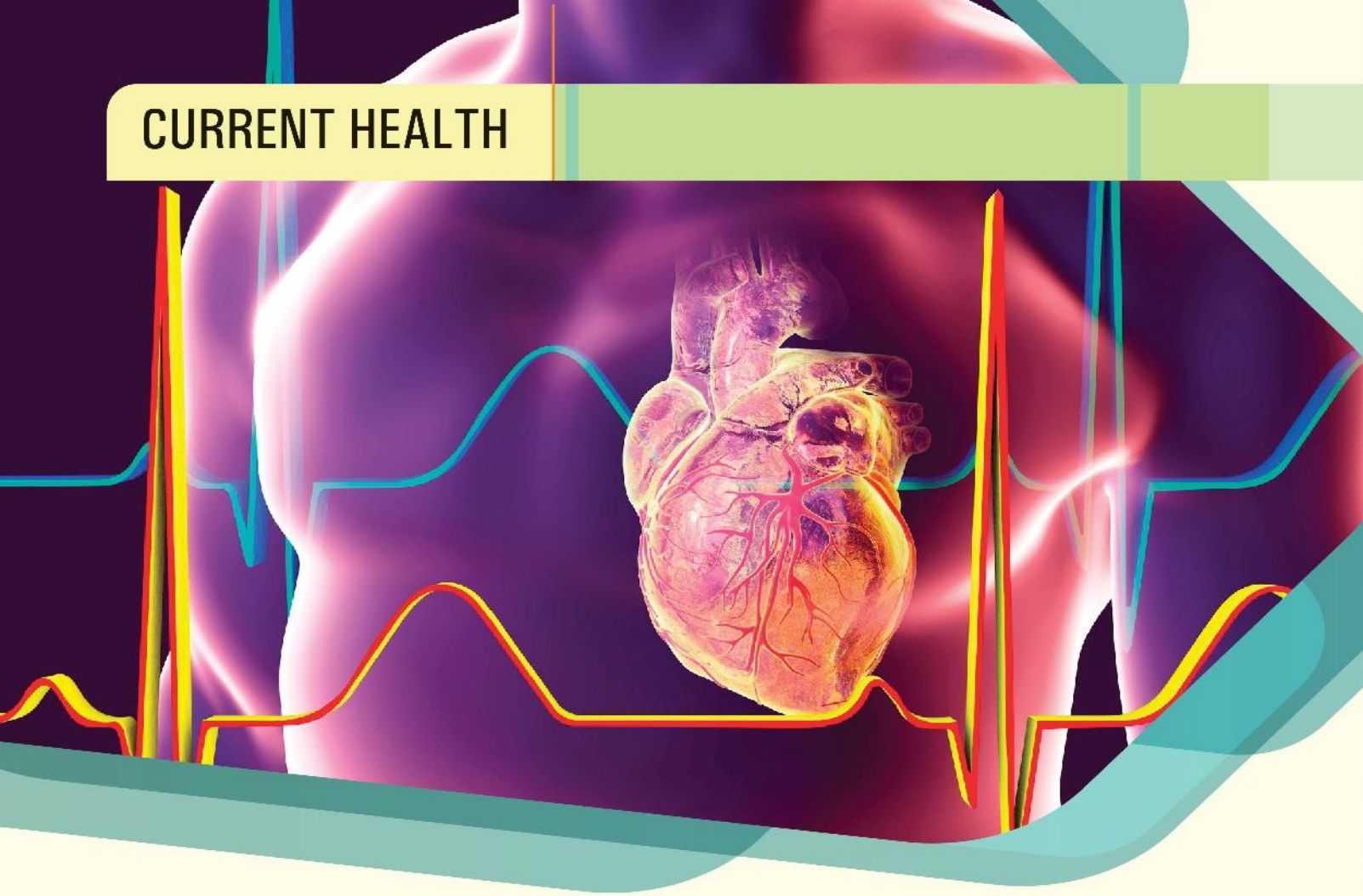
By contrast, with recent advances in the image quality of CT, the ability to identify a fish bone in a lesion has improved. In most of the reported cases, CT revealed not only inflammatory lesions but also linear hyperdense objects in the lesions. In this case, patient was unaware of ingesting a fish bone. However, CT revealed the fish bone like object adjacent to the abdominal wall abscess.

The management of an ingested foreign body depends on the patient's signs and symptoms and the type and location of the ingested object. In almost all asymptomatic patients, conservative management is appropriate, since most objects will pass uneventfully. In all cases of a foreign body in the esophagus, the foreign bodies require removal within 24 hours, even without the presence of signs or symptoms as the risk of complications increases dramatically with time. Sharp objects in the esophagus should be removed urgently, because of the high risk of perforation. Even if sharp objects pass through the stomach, complications have been described in up to 35% of patients. Moreover, objects in the stomach larger than 2 cm to 2.5 cm in diameter or longer than 5 cm to 6 cm should be removed, because they will not pass through the pylorus, duodenum or ileocecal valve. The objects should be removed endoscopically if possible. Surgical removal should be considered in patients who develop complications and for a foreign body that does not progress through the digestive tract.

Conclusion

In conclusion, first laparoscopically treated the case of a pyogenic abdominal wall abscess resulting from a fish bone migrating into the wall of the cecum. Even if a patient is unaware of ingesting a foreign body, aware should be carried out that an abdominal wall abscess can develop in association with an ingested fish bone that has migrated to the region and then remained adjacent to the abscess. Moreover, this case suggests that laparoscopic approach for performing a resection of the portion of the bowel containing a foreign body, which was planned based on the results of diagnostic imaging, is a useful option for selected cases. Such cases include with a foreign body that can be identified by computed tomography.

Reference: Jour. of Med. Cas. Rep., 2019, Vol. 13:369



Device for improving heart failure symptoms

Food and Drug Administration (FDA) approves new device to improve symptoms in patients with advanced heart failure. Heart failure is the fourth leading cause of death attributable to cardiovascular disease. An implantable neuromodulator device was approved for symptom improvement in patients with advanced heart failure who are not eligible for cardiac resynchronization therapy. Bram Zuckerman, MD, director of the FDA's Office of Cardiovascular devices said that the barostim neo system provides patients with a new treatment option for the symptoms associated with advanced heart failure. Patients with the advanced heart failure have limitations of physical activity, fatigue, palpitation or shortness of breath with activity and may not benefit from standard treatments, including currently marketed drugs and devices.

The barostim neo system improves heart failure symptoms by restoring autonomic cardiovascular balance. The device is implanted under the collar bone and delivers electrical stimulation to carotid artery baroreceptors. Baroreceptor activation sends signals to the brain, which in turn signals the heart, blood vessels and kidneys to relax blood vessels, slow heart rate and reduce fluid build up in the body and inhibit the production of stress related hormones to reduce heart failure symptoms. The device previously received approval for resistant hypertension. The device is intended for patients with a regular heart rhythm, a left ejection fraction of $\leq 35\%$ and NT-proBNP levels less than 1600 pg/ml despite appropriate treatment. The device is contraindicated in certain cases, which include certain nervous system disorders, anatomy impairing device implantation, uncontrolled and symptomatic slow heart rate and atherosclerosis near the implant site or known allergy to silicone or titanium.

According to the FDA, the most recent approval was based on a clinical trial involving 408 patients with advanced heart failure who were receiving guideline directed medical therapy. The 125 randomized patients implanted with the device showed significant improvements in a 6 minutes walking test and in quality of life compared with those who received medical therapy only. Potential complications include infection, low blood pressure, nerve or arterial damage, stroke and death.

Reference: JAMA, 15 October 2019; 322 (15):1442

Overview of tremor

Introduction

Tremor is an involuntary, rhythmic, oscillatory movement of a body part. It is important to note that the limbs and head, when unsupported, exhibit slight tremor, referred to as physiological tremor. Physiological tremor is generally not visible or symptomatic unless it is enhanced by fatigue or anxiety, whereas pathological tremor is usually visible and persistent. Tremor is classified along clinical characteristics, including historical features, tremor characteristics, associated signs and laboratory tests etiology. Accurate diagnosis of tremor is important because appropriate treatment depends on the accuracy of the clinical diagnosis. However, this article reviews the classification, diagnosis and the management of tremor elaborately.

Classification

Tremor is a manifestation of the various diseases. Tremors can be classified according to their specific clinical features or by etiology. Because of the numerous and ever expanding etiologies of tremor, etiologic classification is not helpful, whereas classification based on clinical features is more useful to the clinician. Tremors can be divided into the following types.

Phenomenological classification of tremor syndrome

Resting tremor

Resting tremor is evident when our body parts are relaxed and completely supported against gravity (e.g., with the hands in the lap). It is present during sitting, lying down and relaxed position. It attenuates when the body part is in the movement while doing activities. Classically it is seen in the Parkinson's disease; however, it may be a manifestation of a severe essential tremor, Wilson's disease.

Action tremor

Action tremor is initiated by voluntary muscle contraction. They are further sub classified into postural, kinetic and intention tremors.

Postural tremor: Postural tremor is conspicuous when limbs are voluntary maintained in an anti gravity position (e.g., extending arms in front of chest). It decreases when the body parts are supported. The various conditions associated with postural tremor are listed in Table-1.

Table-1: Cause of postural tremor

- Patient's age at onset of tremor
- Enhanced physiologic tremor
- Essential tremor
- Endocrine: hypoglycemia, thyrotoxicosis and pheochromocytoma
- Anxiety and stress
- Toxins: alcohol withdrawal, mercury lead and arsenic
- Dystonia

Kinetic tremor: Kinetic tremor appears while making a voluntary movement. It is appreciated during activities like eating, writing. This tremor is of special concern as it can hamper the daily activities of the patient. Essential tremor, cerebellar tremor, dystonic tremor and primary writing tremor are the common conditions where kinetic tremor is seen. Kinetic tremor is further classified into a simple and task specific tremor.

Intention tremor: Intentional tremor commonly known as cerebellar tremor is coarse tremor with a frequency of below 5 Hz, appears when precision is required to touch a target. It progressively worsens during the movement and reaches its maximal intensity near the target. The limb shakes side to side perpendicular to the line of travel. Classically it is seen in cerebellar disease of any etiology where it is associated with other cerebellar symptoms. However, one third of essential tremor had also intentional tremor.

Etiological classification of tremor syndrome

Essential tremor: Essential tremor (ET) is a bilateral, largely symmetric postural or kinetic tremor involving hands and forearms that are visible and persistent and in which there is no other explanation for the tremor. About half of essential tremor patients had a positive family history. It may involve the voice, head and rarely the legs. The usual frequency of essential tremor usually is 5-10 Hz and it has no latency to onset. Symptom severity often increases over time but the progression is very slow. The majority of patients do not show accompanying neurologic signs or symptoms but occasionally instability or more distinct cerebellar signs may be found during the examination, especially in long standing tremor. This tremor aggravated with stress and attenuated with intake of alcohol.

Parkinson's disease tremor: In Parkinson's disease (PD), about 75% patient had rest tremor during the course of the disease. The Parkinsonian rest tremor is characterized by the asymmetrical in onset, supination pronation, distal predominant, gradually progressive, pill rolling type of tremor. It attenuates during activities and sleep. It is associated with the tremor of lips, jaw and lower limbs. Resting tremor of Parkinson's disease is usually combined with postural and kinetic tremor.

Drug induced tremor: Drug induced tremor is usually symmetrical. It follows the temporal pattern with the initiation of drugs and decreases with the cessation of culprit drug. It is the diagnosis of exclusion and other causes of tremor should be ruled out. Risk factors for drug induced tremor are advancing age, poly pharmacy, underlying structural brain disease, anxious state and renal failure. The etiology of tremor in a certain subset of cases could be attributed to medications the individuals have been administered for certain underlying medical disorders as well. Hereby, a list of drugs causing tremors is given in Table-2.

Neuropathic tremor: Neuropathic tremor is also known as essential tremor like. It is characterized by the postural or kinetic distal predominant symmetrical tremor of. It is manifested in inherited or acquired large fiber predominant peripheral neuropathies. Its frequency ranges from 2.8 Hz to 5.5 Hz. Chronic inflammatory demyelinating polyneuropathy may be associated with this tremor, where it is associated with the IgG4 NF155 antibody. The development of these tremors occurs subacutely within weeks to months. On neurologic examination, other signs of peripheral neuropathy may be present. Serum electrophoresis, electrophysiological studies, cerebrospinal fluid analysis and sometimes nerve biopsy can help us come to a diagnosis.

Dystonic tremor: Tremor is part and parcel of primary dystonia. Dystonic tremor is a focal, postural or kinetic tremor in an individual with dystonia. This tremor may occur in the exact same part of the body as the dystonia or in a different area altogether. Both the frequency and amplitude are often irregular and variable. Subtle symptoms, in terms of mild blepharospasm, voice change of spasmodic dysphonia or slight torticollis, may be seen as important clues by the clinician. Responsiveness to sensory tricks (gestures antagonistic ques) and exhibition of null point (position of the body with no tremor) indicates a dystonic tremor. Task specific tremor such as tremor that only occurs when writing (primary writing tremor) or when performing other specific tasks, may be a form of dystonic tremor. Botulinum toxin injections can ameliorate dystonia and dystonic tremor and are accepted as the treatment of choice.

Psychogenic tremor: Psychogenic tremor is characterized by its sudden onset and its association with a stressful life event. It may manifest as a combination of resting, postural or intention tremors. It begins with the involvement of arms which is followed by involvement of the head and the legs. It shows a continuous or intermittent pattern with fluctuating frequency and amplitude. The majority of the patients have a maximal disability (46%) at its onset. Although various criteria are proposed, the diagnosis can be obvious in patients with abrupt onset generalized shaking. Entrainment sign and the co-activation sign are hallmarks to diagnose the psychogenic tremor. Entrainment sign requires the patient to maintain a tapping rhythm in an uninvolved body part at a different frequency than the suspicious tremor which automatically changes the frequency of involved part to the tapping enforced

Table-2: Drugs causing tremor	
Class of medication	**Examples**
• β-adrenergic agonists	• Terbutaline, adrenaline and salbutamol
• Antidepressants	• Bupropion, lithium and tricyclic antidepressants
• Neuroleptics	• Haloperidol
• Anticonvulsants	• Sodium valproate and carbamazepine
• Dopamine agonists	• Amphetamine
• Heavy metals	• Mercury, lead, arsenic and bismuth
• Xanthines or derivatives	• Coffee, tea, theophylline and cyclosporine
• Anti-arrhythmics	• Amiodarone and verapamil
• Endocrinologic agents	• Thyroxine and corticosteroids
• Dopamine antagonist	• Metoclopramide

frequency. Co-activation sign is the presence the increased tone of the involved limb during its passive movement and decrease of the tremor with the decrease in muscle tone. This tremor has unpredictable course and usually attenuate with sedatives.

Orthostatic tremor: Orthostatic tremor is characterized by high frequency (13 Hz to 18 Hz) tremor occurring in the legs of a person when erect and causes postural instability. Women are affected slightly more frequently than men. The mean age of onset of orthostatic tremor is the sixth decade. Most cases are sporadic. The syndrome can be primary or secondary and may be associated with a variety of disorders, most commonly Parkinsonism. Postural and kinetic tremor is most common, characterized by unsteadiness on standing. The symptoms improve markedly on sitting or walking. At times, the urge to sit down or to move can be so strong that patients often avoid situations where they have to stand still for a period of time, such as when queuing. The high tremor frequency leads to a partial fusion of the single muscle contractions and it can be easier to listen to the contractions through a stethoscope applied to thigh or calf muscles. The sound has been compared with that of a helicopter.

Other tremor syndrome

Physiologic tremor: Physiologic tremor is an action tremor and is present in every healthy person. It becomes more pronounced during periods of muscular fatigue, anxiety, emotional stress and fear or excitement. Other causes of enhanced physiologic tremor are thyrotoxicosis, pheochromocytoma, catecholamine infusion, methyl xanthine administration, drug withdrawal states and alcohol intoxication. These tremors are mostly reversible if the cause of the tremor is identified and corrected. The frequency of physiologic tremor in young adults is 8 Hz to 12 Hz, gradually decreasing with the age to around 6 Hz to 7 Hz in persons older than 60 years. β-receptor agonists enhance physiologic tremor whereas non selective β-blockers and β-2 antagonists are effective in preventing such tremor.

Enhanced physiological tremor: All persons have an asymptomatic physiologic tremor. It is a low amplitude, high frequency tremor at rest and during action. This tremor can be enhanced by certain medications, anxiety, stress and metabolic conditions. Patients with a tremor that comes and goes with anxiety, medication use, caffeine intake or fatigue do not need further testing.

Isolated focal tremors: Patients with no signs of dystonia in the vocal apparatus and no tremor, dystonia or other neurological signs elsewhere are considered to have isolated voice tremor. Cases with hyper abduction or adduction of the vocal cords, as observed in laryngeal dystonia or with dystonia in other body parts are classified as dystonic voice tremor of known or idiopathic etiology. Voice tremor may be observed in dystonia 6 and *ANO3* mutation carriers without clinical dystonia. Voice tremor in ET occurs by definition, in combination with hand tremor.

Head tremor: Head tremor is common in the context of ET. It is also a common manifestation of tremulous dystonia. The relationship between isolated head tremor and focal tremulous cervical dystonia is a topic of ongoing controversy.

Essential palatal tremor: Essential palatal tremor presents with the symptom of an ear click, mostly attributed is usually attributed to a lesion in the dentato-olivary pathway. Thus, symptomatic palatal tremor is typically a combined tremor syndrome and olivary pseudo hypertrophy is usually observed on MRI.

Task specific tremors: Task specific tremors are not uncommon in persons who perform the affected motor task repetitively and frequently.

Primary writing tremor: Primary writing tremor is probably the most common form of task specific tremor. It occurs only when writing or attempting to write. Task specific tremors of the hand or mouth occur in musicians and sportsmen. Yips in golfers is mostly considered a task related dystonia, sometimes manifesting with tremor as the main symptom.

Classical Parkinsonian rest tremor: Rest tremor combined with Parkinsonism is usually asymmetric and commonly unilateral in onset. A characteristic feature of rest tremor in PD is that it ceases or greatly subsides, at least transiently, when the muscles are activated voluntarily to execute a posture or movement. The subsidence of rest tremor may be followed by delayed re-emergence of tremor when a new limb posture is sustained (re-emergent tremor).

Intention tremor: Intention tremor is usually caused by a lesion in the cerebellothalamic pathway. Focal or unilateral intention tremor is rarely an isolated tremor syndrome. Cerebellothalamic dysfunction is caused by a wide range of disorders and syndromic associations help in the differential diagnosis for each patient.

Holmes tremor: Holmes tremor usually occurs with other localizing signs, but is rarely isolated. Dystonia and abnormal proprioception are often present when the underlying pathology is in the thalamus.

Myorhythmia: Myorhythmia has been recently reviewed and is distinguished from palatal tremor, rhythmic skeletal myoclonus and epilepsia partialis continua. It is usually caused by pathology in the brainstem, diencephalon or cerebellum and it is usually associated with other brainstem or cerebellar signs.

Indeterminate tremor syndrome: Patients with indeterminate tremor syndrome have classic essential tremor in addition to other neurologic signs not sufficient to make a diagnosis of a recognizable neurologic disorder.

Isolated chin tremor: Isolated chin tremor, also called geniospasm which is an autosomal dominant hereditary syndrome characterized by episodic, usually stress induced, high frequency, contraction of the mentalis muscle. The onset is typically in infancy or childhood. Usually there is no evidence of any other nervous system abnormality, although abnormal electroencephalographic findings, sleep disorders and involvement of other facial muscles have been described in rare cases.

Isolated voice tremor: Isolated voice tremor occurs in 2 variants. One is considered to be a form of focal dystonia of the vocal cords and the other is considered to be a variant of essential tremor.

Monosymptomatic resting tremor: Monosymptomatic resting tremor is a resting and/or postural tremor in the absence of bradykinesia or rigidity significant enough to diagnose Parkinson disease.

Palatal tremor: Palatal tremor can be either symptomatic due to brainstem and/or cerebellar lesions or essential without any demonstrable brain lesion. In symptomatic palatal tremor, olivary hypertrophy can be demonstrated on magnetic resonance imaging. In the essential palatal tremor, the patient usually has ear clicks which do not occur in symptomatic variety. The symptomatic form is often associated with pendular vertical nystagmus.

Rhythmic movements of tensor veli palatini and levator veli palatini muscles occur in essential and symptomatic palatal tremor, respectively. Symptomatic palatal tremor has been observed in patients with cerebrovascular disease, encephalitis, multiple sclerosis, trauma and neurodegenerative diseases including olivopontocerebellar atrophy, Alexander disease, spinocerebellar degeneration, progressive supranuclear palsy and other neurodegenerative diseases of uncertain origin. Approximately two thirds of patients with palatal tremor and neurodegenerative diseases have evidence of rhythmic tremors in other parts of the body, involving the eyes, larynx, neck and diaphragm.

Psychogenic tremor: Psychogenic tremor is usually a combination of resting and postural or intention tremors. Onset and remission of the tremor is sudden and tremor amplitude decreases during distraction. The tremor does not involve the fingers. There may be a history of somatization and additional unrelated neurologic signs might appear.

Wilson's tremor: Wilson's disease is a rare but important cause of treatable tremor usually presenting under 40 years of age with wing beating pattern in its characteristic form. It is confirmed by serum cerruloplasmin and 24 hours urinary copper excretion. Additional clinical features include ascites, jaundice and chronic liver disease in a young nonalcoholic patient and by presence of Kayser-Fleischer ring, dystonia, dysarthria, drooling in neurological phenotype patients.

Diagnostic approach

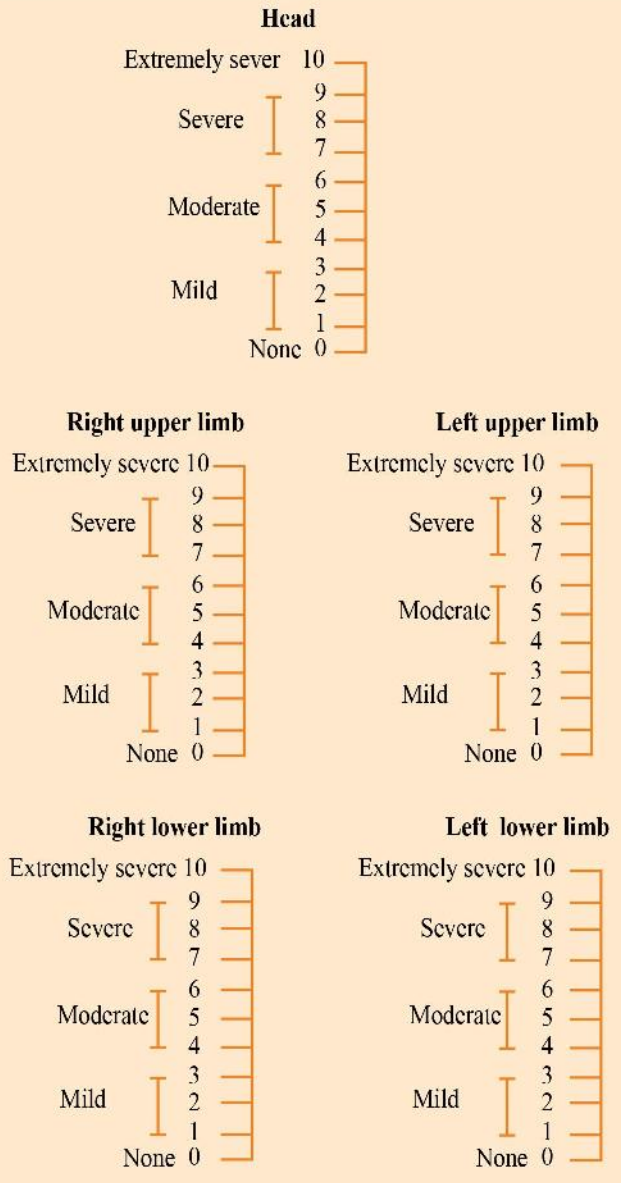
The diagnostic approach to patients with tremor involves the history, physical examination and selected laboratory studies. Firstly, tremor should be classified on basis of its activating stimulus (e.g., rest, kinetic, postural, isometric), frequency and topographic distribution. Action tremor is most common and of these, ET and enhanced physiological tremor are the most frequent diagnoses. Patients with tremor due to other disorders, such as hyperthyroidism, Parkinson's disease, dystonia or Wilson's disease which frequently have additional signs or symptoms that help point to the diagnosis, although this is not always the case.

History: The history concerning the onset of tremor is usually obvious due to its visibility. Examination of previous handwriting samples may be useful in determining the precise time of onset. Precipitating, aggravating or relieving factors, such as coffee, alcohol, drugs of abuse, medications, exercise, fatigue or stress should be elicited. Patient with sudden onset of tremor should arouse suspicion about brain tumor, stroke, cerebellitis, multiple sclerosis, psychogenic tremor and intoxication, while gradual onset points toward Parkinsonism. Generally, tremors are symmetric and asymmetry would point to focal lesion like brain tumor. Family history in ET reflects an autosomal dominant pattern of inheritance

in approximately half of patients, while approximately 5% to 10% of Parkinson's disease cases. History should also assess for severity and disability.

Examination: Examination begins with observations of the tremor during the interview. Many patients with tremor are more symptomatic during the early part of the examination. Patients should be observed while seated, lying down with the affected body part fully supported and while walking. Neurological examination should aim to discover the anatomical location of tremor, the tremor types or components present at those sites and their severities (on a 0-10 rating scale) (Figure-1). In addition it is useful to record specimens of the patient's handwriting and a drawing of a spiral, as estimates of tremor severity (on a 0-10 rating scale).

Figure-1: A validated (0±10) rating scale for scoring tremor severity



Furthermore, other neurological signs should be sought, particularly those related to the conditions listed in Table-3. Thyroid function should be routinely tested in patients with an action tremor. A cerebral magnetic resonance scan may identify a focal lesion for example, in Holmes tremor it can be useful if symptoms and signs are entirely confined to one side of a patient. Nerve conduction studies or electromyogram (EMG) confirm the presence of a peripheral or focal neuropathy.

Table-3: Clues to pathogenesis obtained from examination of the patient

Signs of:

- Parkinsonism: bradykinesia, rigidity and postural instability
- Cerebellar disease: eye movements and speech
- Dystonia: spasmodic torticollis, vocal dystonia and writer's cramp
- Neuropathy: pes cavus, lower motor neuron signs and sensory signs
- Drug induced dyskinesia: orofacial dyskinesia
- Multiple sclerosis (MS): other signs of MS usually evident
- Orthostatic tremor: unsteadiness and palpable leg tremor on standing
- Alcoholism: signs of liver disease
- Wilson's disease: Kayser-Fleischer rings, splenomegaly and hepatosplenomegaly

Laboratory studies: The routine laboratory evaluation of tremor should include tests of thyroid function, glucose, complete blood count (CBC), liver function test, gamma glutamyl transpeptidase (GGT), other diagnostic studies to exclude Wilson's disease and screening for heavy metal poisoning such as mercury or arsenic, if an environmental cause is suspected. Wilson's disease should be suspected in anyone under age 40 who has tremor or other involuntary movements. Hypoglycemia and pheochromocytoma may need to be ruled out in patients with enhanced physiologic tremor. Lithium levels might be done in those suspected of intoxication.

Treatment

Medical treatment

The first line of treatment for tremor is oral medication. β-blockers, anticholinergic medication and levodopa are useful modalities for resting tremor. Kinetic tremor may respond to β-blockers, primidone, anticholinergic medication and alcohol.

Essential tremor: Alcohol intake will temporarily cause dramatic tremor reduction lasting 45 minutes to 60 minutes in the majority of patients with essential tremor. However, this temporary improvement is followed by a rebound phenomenon when the alcohol effect wears off. Moreover, tolerance develops to the effect of alcohol and with time larger amounts of alcohol may be needed to cause tremor reduction. The mechanism of action of alcohol is unknown. However, in a positron emission tomography study, alcohol has been

shown to reduce the over activity of cerebellar connections seen in essential tremor. Propranolol was discovered by chance to improve essential tremor. β-adrenergic blocking drugs (e.g., mainly a non selective β-blocker such as propranolol or a β-selective blocker) have been the mainstays for the treatment of essential tremor. They are however, less effective in the treatment of essential voice and head tremor. Propranolol reduces tremor amplitude but not tremor frequency. The clinical response to propranolol is variable and often incomplete. Nipradilol, a new β-blocker has been shown to be effective in essential tremor. Gabapentin has been used in the treatment of essential tremor. Theophylline reduced tremor to the same extent as propranolol. Primidone has been shown to be effective in the treatment of essential tremor. Benzodiazepines have been used in the treatment of tremor.

Parkinsonian tremor: The response of Parkinsonian tremor to treatment is variable. Several drugs have been tried. Both trihexiphenidyl hydrochloride and carbidopa-levodopa combination have been shown to significantly reduce the tremor of Parkinson disease. In a study comparing the effects of trihexiphenidyl, the carbidopa-levodopa and amantadine hydrochloride, tremor amplitude was reduced by 59% with trihexiphenidyl, 55% by carbidopa levodopa and 23% by amantadine. Dopaminergic and anticholinergic agents are equally effective in patients with Parkinsonian tremor, but dopaminergic substances additionally improve other Parkinsonian signs. Propranolol has been shown to reduce the amplitude of resting tremor by 70% and that of postural tremor by 50% and so can be used as adjunctive therapy in the treatment of Parkinsonian tremor. Apomorphine hydrochloride has been shown to reduce the resting tremor of Parkinson disease. Clozapine has also been shown to be effective in Parkinsonian tremor.

Orthostatic tremor: Orthostatic tremor rarely responds to β-blocker therapy but can be ameliorated by clonazepam alone or in combination with primidone. Levodopa or gabapentin may also improve orthostatic tremor.

Dystonic tremor: Pharmacologic treatment of dystonic tremor is usually disappointing; however, clonazepam or anticholinergic may be tried. Treatment of the underlying dystonia with botulinum toxin often results in significant improvement of tremor.

Physiologic tremor: Usually no treatment is required for physiologic tremor. However, it may interfere with activities requiring extreme precision. Treatment of exaggerated physiologic tremor requires identification and removal or treatment of the precipitating cause such as thyrotoxicosis, hypoglycemia, emotional stress and use of tricyclic antidepressants, neuroleptics and lithium. In cases in which the precipitating cause cannot be removed or highly skilled fine motor function is desired, the treatment with propranolol may be effective.

Cerebellar tremor: There is no effective treatment of cerebellar tremor. However, some success has been reported with clonazepam. It may also respond to levodopa and anticholinergic agents or clozapine when a clinically significant resting tremor is present.

Holmes tremor: Treatment of Holmes tremor is usually unsuccessful. Some success with carbidopa-levodopa and clonazepam has been reported.

Neuropathic tremor: Treatment of neuropathy may or may not improve neuropathic tremor. The tremor of hereditary motor sensory neuropathy often responds to treatment with propranolol and alcohol.

Surgical treatment

The minimal criteria for a patient to be considered a candidate for neurosurgery are a lack of response to medical treatment, tremor resulting in severe disability and the absence of contraindications to neurosurgery. Thermocoagulation (thalamotomy) and deep brain stimulation target nucleus ventralis intermedius thalami. Thalamotomy and thalamic stimulation cause an improvement of the tremor in 80% to 90% of patients with Parkinson disease. Unilateral thalamotomy improves the contralateral tremor in 90% of patients. Deep brain stimulation has similar benefits to thermocoagulation but fewer side effects, including lower perioperative mortality. Other targets for the treatment of parkinsonian tremor are internal pallidum (pallidotomy) and subthalamic nucleus. Stimulation of subthalamic nucleus improves not only tremor but also akinesia by about 70%. Thalamotomy can achieve a permanent satisfying tremor relief in the contralateral extremities of 69% to 93% of patients with essential tremor. Thalamic stimulation has the advantage of less morbidity and the possibility of bilateral surgical treatment, as is needed in most patients with essential tremor.

Conclusion

Tremor disorders should be approached systematically. The tremor should be initially classified into rest or action tremor. Reversible and benign condition like enhanced physiological tremor should be ruled out first by taking detail history regarding the stress, caffeine use, drugs intake. Though the essential tremor is the most common movement disorder in general population, other organic cause of tremors like Parkinson's disease or Wilson's disease should be excluded first before reaching to diagnosis of essential tremor. Detail neurological examination should be performed to find out the subtle associated findings. It is important to recognize and diagnose them accurately and confidently for their successful management.

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Every 4 February is the world cancer day, the global uniting initiative led by the Union for International Cancer Control (UICC). It originated in 2000 at the first world summit against cancer, which was held in Paris. At this meeting, leaders of government agencies and cancer organizations from around the world signed the "Charter of Paris" against cancer, a document containing 10 articles that outlined a cooperative global commitment to improving the quality of life of cancer patients and to the continued investment in advancement of cancer research, prevention and treatment. 2020 marks the midway point of the 3 year (2019-2021) 'I Am and I Will' campaign. 'I Am and I Will' is an empowering call to action urging for personal commitment and represents the power of individual action taken now to impact the future. The aims of this day are to help in saving millions of preventable deaths each year by raising awareness and education about cancer and pressing to governments across the world to take action against the disease.

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