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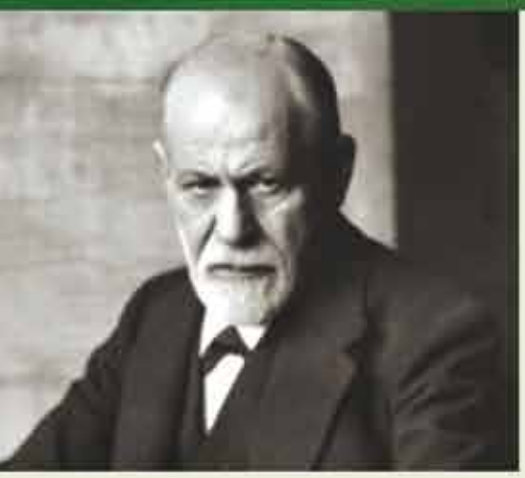
Info Medicus

The essence of medical practice

"The doctor should be opaque to his patients and like a mirror, should show them nothing but what is shown to him."

Sigmund Freud
(1856 - 1939)



Sigmund Freud was an Austrian neurologist and the founder of psychoanalysis, a clinical method for treating psychopathology through dialogue between a patient and a psychoanalyst. In 1882, Freud began his medical career at the Vienna General Hospital. Freud applied his theories outside the clinical setting in 'The Psychopathology of Everyday Life' (1901) and 'Jokes and their Relation to the Unconscious' (1905).

CONTENTS

EDITORIAL

Dear Doctor,

Wishing you a heartiest and happy Pahela Baishakh 1424!

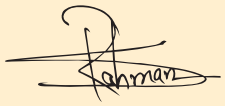
We are much cherished to present you the current issue of our Info Medicus. In this issue we have reminisced some commonly encountered problems which are crucial for practice, as well as patient care. In the present circumstances of the medical science, our objective is to proliferate updated information of diseases.

With pleasure we would like to inform you that there is a new theme in this issue where we have introduced an App for Info Medicus Quiz. In order to download Info Medicus Quiz App, you will have to write 'info medicus quiz' in google play store or apple store. You will find Info Medicus Quiz App as IMQ logo in google play store and apple store. The glimpse of the App is given in the last page of this issue. This will safe your valuable time to participate in the quiz competition at your convenience on go. All the best!

Osteoarthritis is becoming a major cause of pain and disability among elderly patients. This disease leads to an enormous social and financial burden. In this context we have elaborated 'Osteoarthritis: current concepts in diagnosis and management' in 'Review Article'. All other contents are given as usual.

The response received from you especially for the wealth of your suggestions is highly appreciative. We are looking forward to your continuous participation as it will make our endeavor worthwhile in the days to come.

Thanks and best regards,



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Osteoarthritis: current concepts in diagnosis and management

Osteoarthritis (OA) is defined by the American College of Rheumatology as a "heterogeneous group of conditions that lead to joint symptoms and signs which are associated with defective integrity of articular cartilage, in addition to related changes in the underlying bone at the joint margins." Osteoarthritis is usually classified as primary or idiopathic when there is no obvious predisposing cause, and secondary when there is some clearly defined predisposing pathology. Idiopathic OA is the most common form of arthritis and is a debilitating progressive disease that affects 60% of men and 70% of women over the age of 65 with enormous socioeconomic costs, rivaling those of ischemic heart disease. As the obesity is on the increase in the general population, OA will have an even greater impact on society in the future. Primary OA is a frustrating disease for both patient and clinician, because neither cause nor cure is known and once started, the disease cannot be halted, even though individual rates of joint degradation can vary considerably. Understanding of the various aspects of the disease has advanced sufficiently, however, to give rise to cautious optimism that other treatments beside joint replacement may become available.

Pathophysiology

Osteoarthritis (OA) is a degenerative joint disease, chronic and progressive, affecting synovial joints. The pathophysiology of osteoarthritis involves a combination of mechanical, cellular, and biochemical processes. The interaction of these processes leads to changes in the composition and mechanical properties of the articular cartilage. Cartilage is composed of water, collagen, and proteoglycans. In healthy cartilage, continual internal remodeling occurs as the chondrocytes replace macromolecules lost through degradation. This process becomes disrupted in osteoarthritis, leading to increased degenerative changes and an abnormal repair response. These processes result in different interactions between the joint cartilage and adjacent tissues in response to injury or chondrocyte extracellular matrix. This disease can affect upper and lower limbs. From the lesion starts matrix degradation by proteolytic enzymes such as Matrix Metalloproteinase (MMPs). The specific collagenases include MMP-1 (collagenase-1), MMP-8 (collagenase-2) and MMP-13 (collagenase-3). These enzymes are distinguished by the ability to degrade other regions of the triple helical helix of type I collagen, II and III. The gelatinases MMP-2 (gelatinase A) and MMP-9 (gelatinase B) is another group of enzymes that degrade collagen types IV, V, VII and XI. This group acts synergistically with collagenase in cleavage of collagen. In addition, degrade elastin, agrecans and cartilage link protein. Other enzymes are also able to degrade extracellular matrix, such as cathepsin D, degrade aggrecans; cathepsin B and L cleave telopeptides regions of collagen types I and II resulting in

depolymerized collagen fibrils, aggrecans and helical regions of the collagen IX and XI. There are still serine proteases, such as plasmin, which directly degrade extracellular matrix, or by activating metalloproteinase precursors. At the same time, the cartilage components are organized to control progressive degeneration. The decomposition of proteoglycan and collagen bundles triggers increased amount of water, the space between the fibrils followed by a superficial necrosis of chondrocytes and reduced density of these cells. Consequently, the joint surface will change affecting the joint capsule, subchondral bone, ligaments, muscles and tendons, including the synovial fluid. Increased hydration of cartilage and proteoglycans, promotes changes in mechanical properties of the tissue, triggering the loss of integrity of the articular surface and the presence of vertical cracks progressing to deep erosions with the consequent exposure of the subchondral bone.

Risk factor

Age: Even though aging is not sufficient for the development of OA, it is still the strongest risk factor for OA in all joints. Age related changes in articular cartilage likely play a role. They could be related to an alteration within the collagen network crosslinking resulting from the advanced glycation end products, a decreased response of chondrocytes to repair stimuli or altered levels of cartilage oligomeric protein and hyaluronic acid.

Gender: Women are more susceptible than men to OA as well as to higher disease severity. The significant increase in OA prevalence in women around the time of menopause has led to multiple investigations of the hormonal implication in the pathophysiology of OA. In contrast to other tissues (such as endometrium, breast and brain), joint tissues were traditionally thought to be non-responsive to estrogen or to its deficit. On the other hand, it was also reported that women on estrogen replacement therapy were less likely to require total knee or hip replacement. Indeed, those who had taken estrogen for 10 years or longer had a greater decrease in the risk for hip osteoarthritis.

Bone density: As is now well known, OA is certainly not limited to cartilage degeneration, but involves all components (tissues) of the joint.

Genetics: It has been suspected for nearly seven decades that genetics have a strong influence on the incidence of OA. Osteoarthritis is a multifactorial and polygenic late onset disease in which environmental factors are key modulators of gene expression. Moreover, it is becoming increasingly evident that synovitis and inflammation are also involved in the etiology of OA, and that inflammatory molecules including those related to the metabolism of cytokines, prostaglandins, and arachidonic acid are associated with susceptibility to osteoarthritis.

Obesity: Obesity is the strongest modifiable risk factor for OA, possibly due to the high mechanical stresses imposed on the joint. Indeed, it is well demonstrated that OA prevalence in knee and possibly hip and hand OA are increased in overweight people.

Nutritional factors: Nutritional factors have been the subject of considerable interest in OA even though the results are conflicting. One of the most important factors for bone development and remodelling is vitamin D. Since vitamin D influences bone quality, it has long been suspected that its status could have an effect on the risk of the development or progression of osteoarthritis. The systemic factors previously discussed are key modulators of OA susceptibility but they are frequently associated with local biomechanical and biochemical factors.

Biomechanical factors: Knee OA progression is found to be often driven by biomechanical forces. The subsequent pathological response of tissues to such forces leads to structural deterioration, symptoms, and reduced knee function.

Muscle strength: In contrast to high bone mass index or mal-alignment, muscle strength is a more easily modified risk factor for osteoarthritis.

Exercise: Despite the common misconception that exercise is injurious to one's joints, there is no evidence to support this concept in the absence of joint injury.

Joint injury: Although recreational physical activity and sports alone do not appear to be risk factors for developing OA, it has been clearly demonstrated that injuries can counteract the beneficial aspects of sports and lead to secondary OA. A variety of joint injuries are clearly related to the incidence of OA, particularly in the knee, but nearly all joints can be affected.

Classification

Considering the high prevalence and socioeconomic impact of OA, well designed fundamental and clinical studies are most importance. One of the first steps toward reproducible studies is universally accepted classification criteria. It may seem trivial, but these criteria are continually evolving, for example with the creation of different radiographic scoring systems, which make study comparison a real challenge. To date, the most widely accepted classification criteria are those of the American College of Rheumatology (ACR). As shown in Table 1, the ACR has classified OA into two broad categories: idiopathic, which can be localized or generalized, and secondary.

Table 1: American College of Rheumatology criteria for classification of osteoarthritis

Idiopathic

Localized

- Hands
  E.g., Heberden's and Bouchard's nodes (nodal), erosive interphalangeal arthritis (non-nodal), scaphometacarpal, scaphotrapenzial
- Feet
  E.g., hallux valgus, hallux rigidus, contracted toes (hammer/cockup toes), talonavicular
- Knee
  a. Medial compartment
  b. Lateral compartment
  c. Patellofemoral compartment
- Hip
  a. Eccentric (superior)
  b. Concentric (axial, medial)
  c. Diffuse (coxae senilis)
- Spine
  a. Apophyseal
  b. Intervertebral (disc)
  c. Spondylosis (osteophytes)
  d. Ligamentous
- Other single sites
  E.g., shoulder, temporomandibular, sacroiliac, ankle, wrist, acromioclabicular

Generalized: Includes 3 or more areas listed above

- Small (peripheral) and spine
- Large (central) and spine
- Mixed (peripheral and central) and spine

Secondary

Post-traumatic

Congenital or developmental disease

- Localized
  a. Hip disease: e.g., Legg-Calve-Perthes, congenital hip dislocation, chondral dysplasia
  b. Mechanical and local factors: e.g., obesity, un-equal lower extremity length, extreme valgus/varus deformity, hypermobility syndromes, scoliosis
- Generalized
  a. Bone dysplasias: e.g., epiphyseal dysplasia, spondylo-apophyseal dysplasia
  b. Metabolic disease: e.g., haemochromatosis, ochronosis, Gaucher's disease, hemoglobinopathy, Ehlers-Danlos disease

Calcium deposition disease

- Calcium pyrophosphate deposition disease
- Apatite arthropathy
- Destructive arthropathy (shoulder, knee)

Other bone and joint disorders

E.g., avascular necrosis, rheumatoid arthritis, gouty arthritis, septic arthritis, paget's disease, osteopetrosis, osteochondritis

Other diseases

- Endocrine disease: e.g., diabetes mellitus, acromegaly, hypothyroidism, hyperparathyroidism
- Neuropathic arthropathy
- Miscellaneous: e.g., frostbite, kashin-back disease, caisson disease

Diagnosis

Most patients with osteoarthritis need medical attention because of severe pain. However, diagnosis is made by clinical features and some laboratory investigations.

Clinical feature

Pain is the most common presentation of an osteoarthritic joint. The nature of the pain is often described as dull and ill defined; this is especially true for hip disease. Pain is exacerbated by joint use and relieved by rest. In advanced cases, pain also persists at rest and at night, because the protective muscle splinting mechanism around the joint has been lost. Joint pain is typically accompanied by morning stiffness and generally lasts less than an hour. The phenomena of "start-up" pain are commonly described by patients in the early stages of the disease, as is the transient stiffness due to "articular gelling". The latter usually only lasts for a few flexion-extension cycles and is especially prevalent in lower limb disease in the elderly.

The origin of pain is poorly understood. Hyaline cartilage lacks nociceptors, but neighbouring structures do possess them. Pain from articular cartilage lesions results from mechanical irritation of loose flaps of cartilage, from synovial and capsular inflammation, and from subchondral bone sclerosis that acts on the periarticular nerve endings. The stimuli causing pain are related to, but fundamentally different from, those producing cartilage losses. As the disease progresses, the patient notices decreased range of motion due to joint space incongruity, muscle spasm and

contracture, capsular shrinkage, and mechanical block that results from osteophytes or loose bodies. Certain signs that point to the involvement of specific joints are not pathognomonic, but help to confirm the diagnosis. These include varus or bow-legged knees due to medial compartment collapse. The presence of bone spurs that form on the dorsal aspect of the distal interphalangeal joints of the fingers, known as Heberden's nodes, are also commonly seen in patients with established OA. On examination, patients with classic disease often demonstrate localized tenderness along the joint line most severely affected by the degeneration. This is especially true of the medial joint line in knee OA. There is often no demonstrable effusion, or increased local temperature. Osteophytes may be palpable around the affected hand, knee, foot, and ankle joints.

Investigation

- X-ray: A plain X-ray of the affected joint should be performed and often this will show one or more of the typical features of OA. In addition to providing diagnostic information, X-rays are used to assess the severity of structural change, which is useful if joint replacement surgery is being considered
- MRI: If nerve root compression or spinal stenosis is suspected, MRI should be performed
- Routine biochemistry
- Hematology
- Autoantibody
- Synovial fluid aspiration

Treatment

The primary goals of treatment are improved function and quality of life. Treatment should be tailored to the needs of the individual patient. Patient's education, rehabilitation, exercise, modification of activities of daily living, pharmacotherapy, alternative medicine and surgery are all treatment modalities that should be considered. Treatment choices fall into four main categories:

- Non-pharmacological
- Pharmacological
- Complementary and alternative medicine
- Surgical

In general, treatment should begin with the safest and least invasive therapies before proceeding to more invasive, expensive therapies. All patients with osteoarthritis should receive at least some treatment from the first two categories. Surgical management should be reserved for those who do not improve with behavioral and pharmacologic therapy, and who have intractable pain and loss of function. Table 2 presents a stepped care approach for treating osteoarthritis.

Non-pharmacological

Exercise: Non-pharmacological therapy often starts with exercise. Patients are often concerned that joint use will "wear out" a damaged joint. However, the available evidence shows that regular low impact exercise of osteoarthritic joints does not increase the development of osteoarthritis. The goals of an exercise program are to maintain range of motion, muscle strength and general health. All patients with osteoarthritis of the knee should be taught quadriceps strengthening exercises and should be encouraged to perform them every day.

Assistive devices: Many patients with osteoarthritis of the hip and knee are more comfortable wearing shoes with good shock absorbing properties or orthoses.

Weight management: There is a longitudinal association between obesity and osteoarthritis of the knee in men and women, although obesity is a greater risk factor in women. Therefore, primary preventive strategies may include measures to avoid weight gain, or to achieve weight loss in overweight patients.

Supplements: The lay media and books have widely proclaimed dietary supplements such as glucosamine sulfate and chondroitin sulfate to be "cures" for arthritis. At present, these supplements cannot be recommended for use in the treatment of osteoarthritis.

Pharmacological

Simple analgesics: A large number of medicines are prescribed for and consumed by patients with osteoarthritis, largely for the relief of pain. The recognition that pain in osteoarthritis is not necessarily due to inflammation has led to an increased awareness of the role of simple analgesics in the treatment of this disease. The ACR guidelines emphasize the use of acetaminophen as first line treatment for osteoarthritis of the hip and knee. The mainstay of treatment for mild osteoarthritis is acetaminophen. Patients should be instructed to take 650 to 1,000 mg of acetaminophen up to four times per day to relief osteoarthritic symptoms. The U.S Food and Drug Administration recommend no more than 4,000 mg of acetaminophen per day to avoid liver toxicity.

Table 2: Stepped care approach for the treatment of osteoarthritis

		Discuss total joint replacement for osteoarthritis of the hip, knee, or shoulder if steps below are unsuccessful
		Consider hyaluronic acid injection for persistent knee osteoarthritis
		Consider corticosteroid injection for acute exacerbation of knee osteoarthritis
		Consider opioid therapy, but monitor carefully for dependence and abuse
	Add combination glucosamine and chondroitin for moderate to severe knee osteoarthritis; discontinue if no change after three months, but continue if effect is noted	
	Start NSAID therapy, beginning with over-the-counter ibuprofen or naproxen; switch to different NSAID if initial choice is not effective; use generics if possible	
Begin with acetaminophen and continue if still effective, or step up to NSAID		
Encourage regular exercise throughout treatment and encourage weight loss if patient is overweight or obese Consider physical therapy referral for supervised exercise (land or water based); consider bracing and splinting		
Mild osteoarthritis	**Moderate osteoarthritis**	**Severe osteoarthritis**

Nonsteroidal anti-inflammatory drugs (NSAIDs): When acetaminophen fails to control symptoms, or if symptoms are moderate to severe, NSAID therapy is recommended. NSAIDs as a class are superior to acetaminophen for treating osteoarthritis. In patients requiring NSAID therapy, concurrent use of acetaminophen may allow the NSAID dosage to be reduced, thereby limiting toxicity. If an NSAID is to be used, safety is an important issue, especially in the elderly. The risk of NSAID induced renal and hepatic toxicity is increased in older patients and in patients with pre-existing renal or hepatic insufficiency. Thus, it is important to monitor renal and liver function.

When used as cotherapy in patients requiring chronic NSAID treatment, misoprostol, a synthetic prostaglandin E1 analog, helps to prevent gastric ulcers. Omeprazole, a proton pump inhibitor, appears to be as effective as misoprostol in healing NSAID induced ulcers and erosions, and it has the advantage of once daily dosing.

New developments: The presently available NSAIDs work through nonspecific inhibition of cyclo-oxygenase isoforms 1 and 2 (COX-1 and COX-2). COX-1 is expressed in gastric and renal tissues (among others), whereas COX-2 is inducible and is part of the inflammatory response.

Celecoxib is the first COX-2 inhibitor approved by the U.S. Food and Drug Administration (FDA) for the treatment of osteoarthritis and rheumatoid arthritis. In a recent study, celecoxib effectively alleviated pain and reduced inflammation but showed no evidence of inducing gastric ulcers or affecting platelet function. Although the risk of gastrointestinal bleeding is low, physicians should remain vigilant for signs of gastrointestinal bleeding. The most common side effects of celecoxib are dyspepsia, diarrhea and abdominal pain.

An additional COX-2 inhibitor, rofecoxib has also been approved as a once daily medication for the treatment of osteoarthritis and acute pain. Clinical trials showed that rofecoxib was as effective as ibuprofen and diclofenac and was significantly superior to placebo in the treatment of pain in patients with osteoarthritis.

Opioid containing analgesics: It including codeine and propoxy-phene, can be used for short period to treat exacerbations of pain. These agents are not recommended for prolonged use because they cause constipation and increase the risk of falling, particularly in the elderly. Opioids are often used to treat pain and are an option for osteoarthritis pain. Because of the potential for abuse, opioids should be an option only if the patient has not responded to acetaminophen or NSAID therapy, or cannot tolerate them because of adverse effects.

Intra-articular corticosteroid injections: Patients with a painful flare of osteoarthritis of the knee may benefit from intra-articular injection of a corticosteroid such as methylprednisolone or triamcinolone. When a joint is painful and swollen, short term pain relief can be achieved with aspiration of joint fluid followed by intra-articular injection of a corticosteroid. A joint should not be injected more than three or four times in one year because of the possibility of damage of cartilage from repeated injections. Patients who require more than three or four injections per year to control symptoms are probably candidates for surgical intervention. These injections should be performed under fluoroscopic guidance. Intra-articular injections of corticosteroids or hyaluronic acid are another option for treating osteoarthritis. The use of intra articular corticosteroids primarily provides short term relief lasting four to eight weeks.

Many physicians inject a corticosteroid and a local anesthetic, such as lidocaine. The lidocaine can provide some immediate relief, which confirms that the medication was injected into the correct area. Patients should be warned of a potential flare-up of symptoms within the first 24 hours, followed by an improvement from baseline at 48 hours. Repeat injections are possible in the same joint, but usual practice is limited to four injections annually.

Intra-articular injections of hyaluronic acid like products: The major nonstructural component of the synovial and cartilage extracellular matrix is Hyaluronic acid. It confers viscoelastic and lubricating properties to the joint. In patients with osteoarthritis, the concentration and the molecular weight of hyaluronic acid are decreased. Intra-articular hyaluronic acid injections, also known as viscosupplementation, are widely used to treat osteoarthritis of the knee. Thus, viscosupplementation with hyaluronic acid like products is thought to be a possible treatment for osteoarthritis. The FDA has approved sodium hyaluronate and hylan G-F 20 injections for the treatment of pain caused by osteoarthritis of the knee. For patients experiencing chronic osteoarthritis pain, hyaluronic acid should be considered. The technique of injection is the same for either medication.

Complementary and alternative medicine

Acupuncture for osteoarthritis of the knee found only short term benefit which can be of benefit in chronic low back pain. The most widely used supplements for osteoarthritis are glucosamine and chondroitin. The results were favorable only for the combination of glucosamine and chondroitin, which appeared to be effective for moderate to severe osteoarthritis of the knee. Chondroitin alone did not show benefit for osteoarthritis of the knee or hip.

Balneotherapy is a heterogeneous group of treatments also known as spa therapy or mineral baths. Mineral baths were of some benefit to patients with osteoarthritis. Capsaicin cream is a topical analgesic derived from chili peppers. It has been found to be superior to placebo in treating osteoarthritis pain. It is widely available, relatively inexpensive, and can be used as an adjunct to standard osteoarthritis treatments.

There also is evidence supporting the use of the supplement S-adenosylmethionine (SAM-e) to reduce functional limitation, but not compared with placebo in patients with osteoarthritis pain. The effectiveness of SAM-e is comparable to that of NSAIDs in some studies but with fewer adverse effects.

Surgical

Surgery should be reserved for patients whose symptoms have not responded to other treatments. The well accepted indication for surgery is continued pain and disability despite conservative treatment. The most effective surgical intervention is total joint replacement, with excellent patient outcomes following total joint replacement of the hip, knee, and shoulder. Many different prosthetic devices are available. Patients can expect that most current joint prostheses will function well for 15 to 20 years. There are other surgical approaches to osteoarthritis treatment, but they have not equaled the success of total joint replacement. Randomized trials of arthroscopic debridement for osteoarthritis of the knee have consistently failed to show an advantage over maximal medical therapy combined with physical therapy.

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Sleep with anger makes bad thoughts unforgettable

The age old advice, "never go to bed angry" is getting some support from new research. Researchers found that men in the study were less able to suppress a negative memory after they slept with anger. Normally, sleep helps people process information from the day and store it in their memory. The new finding suggests that this process of solidifying memories during sleep also makes it harder to suppress negative memories, which people may not want to recall. The results suggest that people should try to resolve any arguments before going to bed and not sleep on their anger, said Yunz he Liu, a Ph.D. student in neuroscience at University College London.

In the study, the researchers asked 73 men in England to look at 26 neutral photos of people's faces. The photos were neutral, meaning they were not associated with either positive or negative emotions. But each of these neutral photos was paired with an upsetting image, such as a photo of corpses, crying children and injured people. This way the men learned to associate each face with an upsetting image. Shortly after the researchers showed the participants some of the photos of faces again and asked them to try to suppress or forget their memories of the associated unsettling images. Specifically, the people were 9 percent less likely to recall the upsetting images compared with other, baseline images that the researchers had shown the participants earlier on in the study to test their memory performance. The researchers repeated the memory suppression task the next day, after the participants got a night's sleep and found that this time around the participants reported that they had more trouble forgetting the upsetting images that had been paired with the faces. Specifically they were just 3 percent less likely to recall the upsetting images compared with other, baseline images that the researchers has shown their earlier on during the study to test their memory performance. These results suggest that sleep may make it harder for people to forget things they'd rather not remember, the researchers said.

The researchers also scanned the people's brains during the memory suppression task and compared the participants brain activity when they tried to suppress the negative memory before they slept with their memory suppression activity after they slept. There was a difference: when the men were asked to suppress their memory of the negative image before they slept, the hippocampus, the brain's memory center, was the part of the brain that was most involved in the task of suppressing memories. But after the men got a night's sleep, other regions of the brain became activated in the task as well, according to the study in the journal Nature Communications. This last finding may lead to a better understanding of conditions such as post-traumatic stress disorder, in which people cannot suppress traumatic memories, the researchers said.

Reference: *www.livescience.com*

Iron deficiency anemia leads to hearing loss

New research suggests, iron deficiency anemia may lead to hearing loss, which is a combination of low levels of iron and red blood cells. The study found that people with iron deficiency anemia have more than twice the rate of hearing loss as people without the blood disorder. The association between hearing loss and iron deficiency anemia was particularly strong for two types of hearing loss one called sensorineural and combined sensorineural and conductive hearing loss. Sensorineural hearing loss occurs when the inner ear or the nerve pathway from the inner ear to the brain is damaged, according to the American Speech Language Hearing Association (ASHA). Conductive hearing loss is when sounds aren't efficiently conducted from the outer ear to the eardrum or middle ear. Combined hearing loss is a mixture of the two, according to ASHA. ASHA reports that sensorineural hearing loss is generally considered permanent.

If iron deficiency anemia plays a role in hearing loss, it's possible that correcting the condition might lead to improvements in hearing. But, researchers say it's too soon to tell if that would happen, and they aren't recommending that people with hearing loss get blood tests for anemia. The inner ear is very sensitive to changes in blood supply, so it's possible that the lack of oxygen in the blood of people with iron deficiency anemia might affect the inner ear. The part of the inner ear affected by sensorineural hearing loss is supplied by only one artery, which makes it susceptible to damage if low oxygen is present. For the new study, the researchers checked diagnoses of hearing loss in more than 300,000 U.S. adults from 2011 to 2015. They were between the ages of 21 and 90, with an average age of 50. Most were women. When the researchers looked at the type of hearing loss, the overall risk for sensorineural hearing loss in someone with iron deficiency anemia was 82 percent higher than for someone without the blood condition. People with anemia had a 2.4 times greater risk of combined hearing loss than folks without anemia.

ENT specialist Peter Steyger of Oregon Health & Science University said "iron is clearly required for normal functioning of the auditory system, as for many other organs and too little can result in anemia, the loss of hemoglobin in red blood cells to carry oxygen to the tissues in the body. Too little iron can also disrupt the workings of cells and even kill them, leading to hearing loss if that happens to hair cells in the inner ear. Once the sensory hair cells in the inner ear are damaged and die, they cannot be restored to restore auditory function. A healthy well balanced diet that meets the daily recommended intake of vitamins and other nutrients is crucial for everyone's general physical well-being, as well as for optimal hearing health".

Reference: *www.livescience.com*

Performing punch biopsy of the skin

Overview

A punch biopsy allows for the diagnosis of skin conditions by means of histologic examination of a sample of the full thickness of the skin. The procedure is easy to master and has a low risk of adverse events and complications.

Indication

Skin biopsies are simple office procedures that can provide useful information about undiagnosed lesions such as neoplasms, bullous disorders, keratoses, or dysplastic nevi. A diagnostic biopsy can also be the definitive treatment for some malignant, irritated, or precancerous lesions. The primary types of skin biopsies are incisional and excisional.

Incisional biopsies: They sample only some part of the lesion e.g., superficial shave, partial punch.

Excisional biopsies: Excisional biopsies also called deep scoop shave. They are used to remove the entire lesion for diagnostic purposes e.g., fusiform elliptical punch for 1 to 4 mm lesions, saucerization.

Selection of the biopsy site

Fresh lesions that have not been excoriated or subjected to secondary infection tend to provide the best diagnostic information. A specimen taken from dependent parts of the body, such as the lower legs, which are subject to venous stasis, may confound the histologic picture. In addition, the legs are typically susceptible to poorer wound healing and to infection. It is best to avoid punch biopsies of the legs. Biopsies performed on the back can have poor aesthetic results because the scars are often subject to stretching. Performing biopsies on the face should be avoided whenever possible. Never plunge a biopsy punch deep into the temple, the jaw, or the finger, since substantial injury to a nerve or artery could result.

Equipment

A standing tray should be prepared with a clean disposable cover. It is important to exercise clean technique, but it is not necessary to maintain a sterile field. Alcohol pads, a local anesthetic, gloves, toothed forceps, short scissors, suture material, gauze, a needle driver, the punch instrument, and a specimen bottle that contains formalin and has been properly labeled with the patient's name, the patient's identification number, the biopsy site, and the date are needed.

For a punch biopsy in which the area sampled is 2 to 3 mm in diameter, it is acceptable to use a hemostatic agent such as Monsel's solution (also known as ferric subsulfate solution), which can obviate the need to suture the defect. Although Monsel's solution is an excellent hemostatic agent, it may tattoo the skin, causing long

lasting hyperpigmentation. Consequently, some prefer to use aluminum chloride (usually a 35% solution) to achieve hemostasis.

The aesthetic outcome can be satisfactory to excellent when either of these hemostatic agents is used for a small wound (<3 mm in diameter) left to heal by secondary intention (i.e., without suturing). This option also has the added benefit of not requiring the patient to return for suture removal.

For punch biopsies of larger areas of skin, the wound should be closed with a non-absorbable suture, such as nylon or polypropylene. In general, for thick skin, such as the skin on the back or volar skin, it is advisable to use a 3.0 reverse cutting suture. A 5.0 suture is recommended for the face, and a 4.0 suture for the rest of the body. The vast majority of skin biopsy samples are transported in formalin in preparation for routine staining with hematoxylin and eosin.

Procedure

Performing the biopsy

The procedure should begin by cleaning the area with an alcohol swab. If the lesion is poorly demarcated, it can be outlined with a skin marking pen. The area is anaesthetized by inserting the needle parallel to the lesion and slowly raising an intradermal bleb beneath it. For the biopsy of pigmented lesions, it is important to center the punch over the lesion, which will help to secure a complete sample. The back of the punch can be used to make an imprint around the lesion that will serve as a guide to perform the biopsy.

First the skin stretched at an angle that is perpendicular to the skin tension lines. The punch is positioned over the biopsy site by holding the punch in one hand and placing the fifth finger of that hand adjacent to the lesion for stability. A gentle rotational and downward pressure is applied on the punch. Toothed forceps are used to grasp and lift the specimen, without crushing it. It is easy to remove the specimen without cutting the base as the fat layer is often loose. Scissors are also used to cut the base of the specimen.

Suturing the biopsy site

When the specimen has been obtained, the wound is sutured. Sutures should be placed perpendicular to the long axis of the punch defect or perpendicular to the skin tension lines. The needle is being placed through the edge of the wound. The curve wrist will naturally direct the blunt end of the needle toward the forceps. The needle driver is reloaded before the opposite edge of the wound is pierced. The suture is being circled around the needle driver twice before resting the driver on the thumbnail to prevent the suture from slipping. The first part of the knot is secured, by using a gauze to absorb excess bleeding. The suture is circled in the opposite direction to cinch a square knot. These steps are repeated three times, and then the suture is cut at about 1 cm. For making a second suture, the entire wound is pierced without going through the center.

Special situations

Although complications from a punch biopsy are rare, it is important to monitor the patient for bleeding, infection, and scarring. There are a few situations in which the simple suture may fail. The edges of large punch wounds in lax skin may invert with a simple suture; in such cases, a vertical mattress suture is preferred, since it will evert the edges of the wound for better approximation of the skin edges. When performing a punch biopsy on volar skin, the suture material may cut through the tissue; therefore, piercing the skin farther away from the punch defect is necessary. In addition, sutures with a larger caliber, such as a 3-0 nylon suture, will be less likely to cut through tissue than sutures with a smaller caliber.

Bleeding is often easily controlled with the local application of pressure and the proper placement of the first suture. In patients with thrombocytopenia or those being treated with anticoagulants, it is typical to place a pressure dressing over the sutured site to prevent late-onset bleeding. When performing a biopsy of the lip, which is highly vascular and extremely pliable, use a chalazion clamp to allow for easy handling and intraoperative hemostasis. Performing a punch biopsy in infants is fairly easy, since the subcutaneous fat will push the specimen up, making it relatively easy to grasp, there is no need to punch much beyond the dermis.

Aftercare

The application of a topical antibacterial agent after a punch biopsy is discouraged because allergic reactions are common. The biopsy site should be cleaned with soap and water twice a day. After dabbing it dry, the patient should apply petroleum jelly to aid healing. When sutures have been placed, they should be removed 1 to 2 weeks after the procedure, depending on their location. To minimize the risk of track marks and scarring, sutures placed on the face should be removed in 1 week, and sutures on other parts the body should be removed in 10 to 14 days.

Summary

The punch biopsy is the primary technique used for the diagnosis of many dermatologic conditions. Provided that the site of the biopsy is selected judiciously, that hemostasis can be achieved, and that there is a pathologist available who can assess and diagnose histopathological features of the skin, the punch biopsy is a helpful procedure for physicians across a broad spectrum of specialties.

References:

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Spontaneous spleen rupture: an unusual involvement of the spleen in SLE

Introduction

The involvement of the reticuloendothelial system in systemic lupus erythematosus (SLE) concomitant to the disease activity is highly variable, and well recognised but spontaneous splenic rupture is an unusual occurrence. Clinical features of splenic rupture include pain, tenderness and muscle guarding in the left upper quadrant of the abdomen along with features of haemorrhagic shock. In the absence of a history of trauma, other clinical diagnoses may be found on presentation, most commonly perforated peptic ulcer. The spleen can rupture after trauma or spontaneously to a normal or diseased spleen. The spontaneous rupture of the spleen, which occurs suddenly or insidiously, was first described by Aktinson and is a rare complication of infectious, haematological, neoplastic and rheumatic disorders, like systemic lupus erythematosus.

The clinical spectrum of patients with SLE is highly variable. The involvement of the reticuloendothelial system is a well-recognized concomitant of the disease. However, it is usually mild, its course is independent of other disease manifestations, and it is not useful as a little use as a prognosis marker. Spontaneous nontraumatic rupture is extremely rare and has been associated as an unusual event in patients with systemic lupus erythematosus.

Case report

A 35 year old female patient presented at the age of 31 with severe pericardial effusion, bilateral pleural effusion, ascites and polyarthralgia. At presentation, she had diagnostic criteria for SLE. Her laboratory tests revealed blood urea nitrogen of 87 mg/dl (normal 4mg/dl to 15 mg/dl), a creatinine level of 3.1 mg/dl (normal 0.6mg/dl to 1.3 mg/dl), 136 mEq/l of sodium, and a 5.3 mEq/l of potassium. Her complete cell blood count revealed lymphopenia (830/mm), a platelet count of 591,000/mm (normal 150,000/ ul to 400,000/ul) and anemia without reticulocytosis and with a negative indirect Coombs test. The urinalysis revealed albuminuria (3+) and a 24 hour urine collection demonstrated 304 mg proteins. Furthermore she had a positive ANA (1:640), anti-ds DNA antibodies (75 UI/ml) with positivity to anti SSA, SSB and Histone.

Considering the multisystemic involvement of the disease, she was treated with bolus therapy with three daily pulses of methylprednisolone one at a dose of 1,000 mg/day, followed by 0.5 mg/kg/day prednisolone. At the time, ultrasonography showed no changes in the spleen or liver. The disease has been controlled gradually and the patient closely monitored, using 5 mg/day prednisolone, 5 mg enalapril daily and hydroxychloroquine, with no further sign of disease activity.

Four years later, she was re-admitted to the hospital with complaints of dyspnea, left upper abdominal quadrant pain, diarrhea, fever for ten days, and painful left cervical lymphadenopathy, with no history of trauma. On physical examination, she presented symptoms compatible with Systemic Inflammatory Response Syndrome (SIRS)- her temperature was

38°C, cardiac rate of 120 bpm, and respiratory rate of 38/min. She also had joint pains, generalized maculopapular rash for one week and jaundice. Her abdomen was tense, tender with guarding to palpation and particularly painful in the left hypochondrium. The spleen was palpable, but its size could not be determined because of the presence of extreme abdominal tenderness and guarding.

Laboratory data included: white blood cells of 9.0 $\times 10^3/mm^3$ (normal $4000/mm^3$ to $11000/mm^3$) with segments 84.0%, band forms 10.0%, lymphocytes 5.0%, a platelet count of 50,000/ul (normal 150,000/ul to 400,000/ul), anemia with no reticulocytosis. Indirect Coomb's test was negative, total bilirubin level of 2.9 mg/dl (normal 0.2 mg/dl to 1.3 mg/dl), indirect bilirubin of 1.8 mg/dl (normal 0.2 mg/dl to 0.8 mg/dl), Erythrocyte Sedimentation Rate (ESR) of 50 mm/h (normal westgreen 0 mm/h to 20 mm/h), electrolytes within normal limits and normal liver and kidney function. Urine, blood and stool culture were negative and serologic tests for cytomegalovirus (CMV), Epstein Barr virus (EBV), human herpes virus type I (HHV-1) and hepatitis were negative, as well as anti-cardiolipin and serum antiphospholipid antibodies.

Abdominal ultrasound revealed the presence of subcapsular collection in the spleen and enlarged left ovary (5.0 × 7.0 cm). An abdominal computed tomography with contrast showed no evidence of gallbladder stones or abnormality of the liver, but revealed a left subphrenic collection with dense fluid in close contact with the spleen measuring 9.0 cm in diameter and also the presence of bilateral pleural effusions associated with subsegmental parenchymal consolidations.

A laparotomy was performed and the exploration confirmed hemoperitoneum secondary to ruptured spleen with subcapsular hematoma. Splenectomy and a left oophorectomy were performed. Grossly, the spleen was 566 g, 140 × 100 × 70 mm. Its surface was lobulated, with lacerations. Microscopic examination revealed splenic follicle atrophy, with an onion skin appearance of the splenic central artery and histocytes proliferated. There was no significant vascular lesion.

She was admitted to the intensive care unit for post-operative care where she remained for three days. Jaundice was attributed to infectious process and it disappeared after surgery and the patient had clinical improvement being discharged home after 15 days of admission without symptoms. Her follow-up care after discharge was performed regularly at the hospital clinic and routine blood and urine tests, ESR, liver and kidney functions remained normal. Three years after the event, the disease remains stable and the patient is currently taking 400 mg/day of hydroxychloroquine and 5 mg/day of prednisolone to control her articular symptoms. The autoantibodies are still present in her serum.

Discussion

Splenic rupture in the absence of trauma, called spontaneous splenic rupture, is a rare complication of infectious, haematological and neoplastic disorders. Causes of a traumatic splenic rupture can be divided into six main categories: infectious, neoplastic, inflammatory, congenital or structural, iatrogenic and finally idiopathic. It complicates about 0.2% of cases of infectious mononucleosis. Others conditions reported in the paediatric age group include severe meningococcal septicaemia, congenital a fibringenemia and malaria. An increased risk for pathologic splenic ruptures has also been reported in patients with systemic vasculitis and several rheumatic conditions.

SLE is a multisystem disease and its clinical manifestations vary according to the organs involved. The immune system functions are closely linked to the spleen. Involvement of the reticuloendothelial system in SLE is a well-recognized and can present with lymphadenopathy and splenomegaly, abscesses, abnormal spleen function, histological abnormalities, capsulitis, infarction, and spontaneous rupture. Its reported frequency varies widely, from 9% to 46%. However, spontaneous spleen rupture is an extremely rare complication. It is also note worthy that the histopathological findings in analysis of the spleen can guide the etiological diagnosis of the rupture. The typical histological feature of splenic involvement in SLE is the onion skin lesion of periarterial fibrosis. Other pathological features include capsulitis and small infarcts, which are presumably secondary to arterial thrombosis, observed in association with the presence of a raised titer of anti-cardiolipin antibodies, not identified in this case. In agreement with previous reports, the microscopic examination revealed splenic follicle atrophy, onion skin appearance of the splenic central artery and histocytes proliferated with no significant vascular lesion.

Because of its rare occurrence, the clinical and pathophysiologic factors that may predispose the spleen to spontaneous rupture have not been defined. It continues to pose a diagnostic challenge and dilemma in management. Then, knowledge of this condition allows the identification of risk factors and the patients characteristics with this rare disease. The survival of these patients seems to be uncertain and an early diagnosis may be the only way to a proper and effective treatment improving survival.

Reference: *Clin. Med. Rev. Cas. Rep., 2016, Vol. 3 (3); 3:096*

Deviated nasal septum

The nasal septum is the wall dividing the nasal cavity into halves; it is composed of a central supporting skeleton covered on each side by mucous membrane. The front portion of this natural partition is a firm but bendable structure made mostly of cartilage and is covered by skin that has a substantial supply of blood vessels. The ideal nasal septum is exactly midline, separating the left and right sides of the nose into passageways of equal size. A deviated nasal septum occurs when the septum is severely shifted away from the midline.

Etiology

Direct trauma

Many septal deviations are a result of direct trauma and this is frequently associated with damage to other parts of the nose such as fractures of nasal bone. Fractures involving nasal bones are the commonest fractures involving the facial skeleton.

Birth molding theory

Many patients with septal deviation do not give history of trauma. Birth molding theory was propounded by Gray. According to him abnormal intrauterine posture may result in compression forces acting on the nose and upper jaws. Displacement of septum can occur in these patients due to torsion forces that occur during parturition.

Differential growth between nasal septum and palate

This is the most acceptable theory today. When the nasal septum grows faster in certain individuals than the palate then the nasal septum starts to buckle under pressure.

Classification

Deviated nasal septum is classified into:

- Spurs
- Deviations
- Dislocations

Spurs

These are sharp angulations seen in the nasal septum occurring at the junction of the vomer below, with the septal cartilage and or ethmoid bone above. This type of deformity is the result of vertical compression forces. Fractures that occur through nasal septum during injury to the nose may also produce sharp angulations. These fractures can be healed by fibrosis that extend to the adjacent mucoperichondrium. This increases the difficulty of flap elevation in this area.

Deviations

In patients with septal deviation a compensatory hypertrophy of the turbinate's and bulla may occur on the side opposite to the deviation. If compression forces are involved the septal deviations are often asymmetrical and may also involve the maxilla, producing flattening of the cheek, elevation of the floor of the affected nasal cavity, distortion of the palate and associated orthodontic abnormalities. The maxillary sinus is usually slightly smaller on the affected side. Anterior septal deviations are often associated with deviations in the external nasal pyramid. Deviations may affect any of the three vertical components of the nose causing

- Cartilaginous deviations
- The C deviation
- The S deviation

Cartilaginous deviations: In these patients the upper bony septum and the bony pyramid are central, but there is a dislocation deviation of the cartilaginous septum and vault.

The C deviation: Here there is displacement of the upper bony septum and the pyramid to one side and the whole of the cartilaginous septum and vault to the opposite side.

The S deviation: Here the deviation of the middle third (the upper cartilaginous vault and associated septum) is opposite to that of the upper and lower thirds. With deviations of the nose, the dominant factor is the position of the nasal septum. The first step, therefore in treating the twisted nose is to straighten the septum, and if this objective is not achieved, there is no hope of successfully straightening the external pyramid.

Dislocations

In this the lower border of the septal cartilage is displaced from its medial position and projects into one of the nostrils.

Clinical feature

A deviated septum may cause one or more of the following symptoms:

- Blockage of one or both nostrils
- Nasal congestion
- Frequent nose bleeding
- Frequent sinus infections
- At times, facial pain, headaches
- Noisy breathing during sleep

Management

Initial treatment of a deviated septum may be directed at managing the symptoms of nasal obstruction and drainage.

Decongestants: Decongestants are medications that reduce nasal tissue swelling, helping to keep the airways on both sides of nose open. Decongestants are available as a pill or as a nasal spray.

Antihistamines: Antihistamines are medications that help prevent allergy symptoms, including obstruction and runny nose. They can also sometimes help nonallergic conditions such as those occurring with a cold.

Nasal steroid sprays: Prescription of nasal corticosteroid sprays can reduce inflammation in the nasal passage and help with obstruction or drainage.

Surgery: It may be the recommended treatment if the deviated septum is causing nose bleeding or recurrent sinus infections. The most common surgical procedures for Deviated nasal septum are

- Sub Mucosal Resection (SMR)
- Septoplasty

Sub mucosal resection: Sub mucosal resection of nasal septum is ideally performed under local anesthesia. Infiltration is done at the mucocutaneous junction on both sides just behind the columella. The floor of the nasal cavity is also infiltrated on the concave side. Killian's incision is preferred for SMR operations. Killian's incision is the commonly used incision. It is an oblique incision given about 5mm above the caudal border of the septal cartilage. The cartilaginous and bony nasal septum is exposed by elevation of mucoperichondrial and mucoperiosteal flaps on both sides. Flaps are elevated on both sides of the nasal septum. The cartilage is fully exposed from both sides and is remove using a Luc's forceps or a Ballenger's swivel knife. The flaps are allowed to fall back in place and wound is closed with catgut.

Septoplasty: Septoplasty is a surgical procedure performed entirely through the nostrils, accordingly, no bruising or external signs occur. The surgery might be combined with a rhinoplasty, in which case the external appearance of the nose is altered and swelling/bruising of the face is evident. Septoplasty may also be combined with sinus surgery. The time required for the operation averages about one to one and a half hours, depending on the deviation. It can be done with a local or a general anesthetic, and is usually done on an outpatient basis. After the surgery, nasal packing is inserted to prevent excessive postoperative bleeding. During the surgery, badly deviated portions of the septum may be removed entirely, or they may be readjusted and reinserted into the nose.

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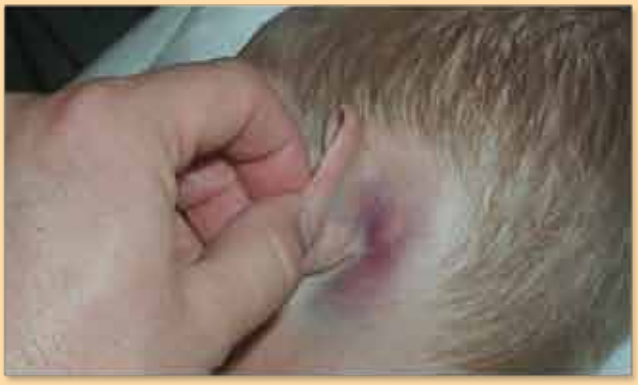
2. WebmedCentral.com
3. www.mayoclinic.org

Battle's sign

A 46 year old man presented to the otolaryngology clinic with hearing loss and a sensation of fullness in the left ear. The symptoms had started a few days before presentation. He had no other neurologic symptoms. Otoscopy revealed a hemotympanum through the eardrum. In addition, a subcutaneous hemorrhage was observed in the left postauricular region. The patient reported no history of head injury but said that he had been having partial memory blackouts since drinking alcohol at a New Year's party 5 days earlier. Computed Tomography revealed an acute epidural hematoma, accompanied by a left temporal bone fracture. The patient was sent to the neurosurgery department for evaluation, where it was determined that no intervention was required. The patient's symptoms were improved over the next several weeks, with the exception of slight sensorineural hearing loss in the left ear, detected on audiometry. Battle's sign represents ecchymosis around the mastoid process from head trauma that has caused a



temporal bone fracture. The sign can suggest that a patient has sustained a significant blow to the skull, even if the medical history is obscure.

Reference: *N. Eng. J. Med., September 20, 2012; 367;12:e1135*

Hutchinson's nail sign

A 37 year old man presented with a brownish black nail on the great toe of the right foot. A darkly pigmented linear patch had been started to form within the toe nail 4 years earlier and had widened and darkened over time. Two months before the current presentation, the dark pigment had begun to involve the hyponychium and the proximal and lateral nail folds. The patient's personal and family medical histories were otherwise unremarkable. The results on routine laboratory testing were within normal limits. An incisional biopsy of the nail matrix showed atypical melanocytes and inflammatory cells along the basal layer of the epidermis, findings consistent with acral lentiginous melanoma in situ. Subungual melanoma, a variant of acral lentiginous melanoma, arises from the nail matrix, most commonly in the great toe or thumb. Hutchinson's nail sign is an important clinical clue to subungual melanoma and is

characterized by extension of brown or black pigment from the nail bed, matrix, and nail plate to the adjacent cuticle and proximal or lateral nail folds. The patient underwent amputation of the great toe, and he remains healthy 8 years later.

Reference: *N. Eng. J. Med., May 5, 2011; 364;18:e38*

Important Health Day

April-June 2017

7 April 2017	:	World Health Day
17 April 2017	:	World Hemophilia Day
19 April 2017	:	World Liver Day
25 April 2017	:	World Malaria Day
2 May 2017	:	World Asthma Day
8 May 2017	:	World Thalassemia Day
17 May 2017	:	World Hypertension Day
28 May 2017	:	International Women's Health Day
8 June 2017	:	World Brain Tumor Day
14 June 2017	:	World Blood Donation Day

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