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The essence of medical practice

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CONTENTS

EDITORIAL

Dear Doctors,

Happy New Year!

We are extremely thankful for all of you, our readers. You inspire us to continue to do what we love. Your letters, e-mails and involvement in Info Medicus Quiz App by liking and sharing our posts are what keep us motivated.

We hope you will continue to enjoy our newsletter info medicus and to celebrate life with us easy day.

Winter season has arrived as well as its various advantages and disadvantage come along it. One of the most disadvantage of winter season in Bangladesh is people are suffering from Asthma. Considering this fact, we have chosen "Management of Asthma" in our review article section.

The laryngeal mask airway is useful for the ventilation of patients in cardiac arrest and can be used as an alternative to traditional bag and mask ventilation when there is a need for prolonged resuscitation, when the personnel present do not have the competence to perform endotracheal intubation, when tracheal intubation cannot be performed or has failed. For this reason, we have discussed "Laryngeal mask airway in medical emergencies" in essential procedure section.

Buerger's disease is characterized by an inflammatory endarteritis that causes a prothrombotic state and subsequent vaso-occlusive phenomena. The condition is strongly associated with heavy tobacco use, and disease progression is closely linked to continued use. In the health care section, we have discussed "Buerger's disease".

Enjoy the season with your loved ones and make sure to reflect on all the things that make you happy.

Thanks and best regards!

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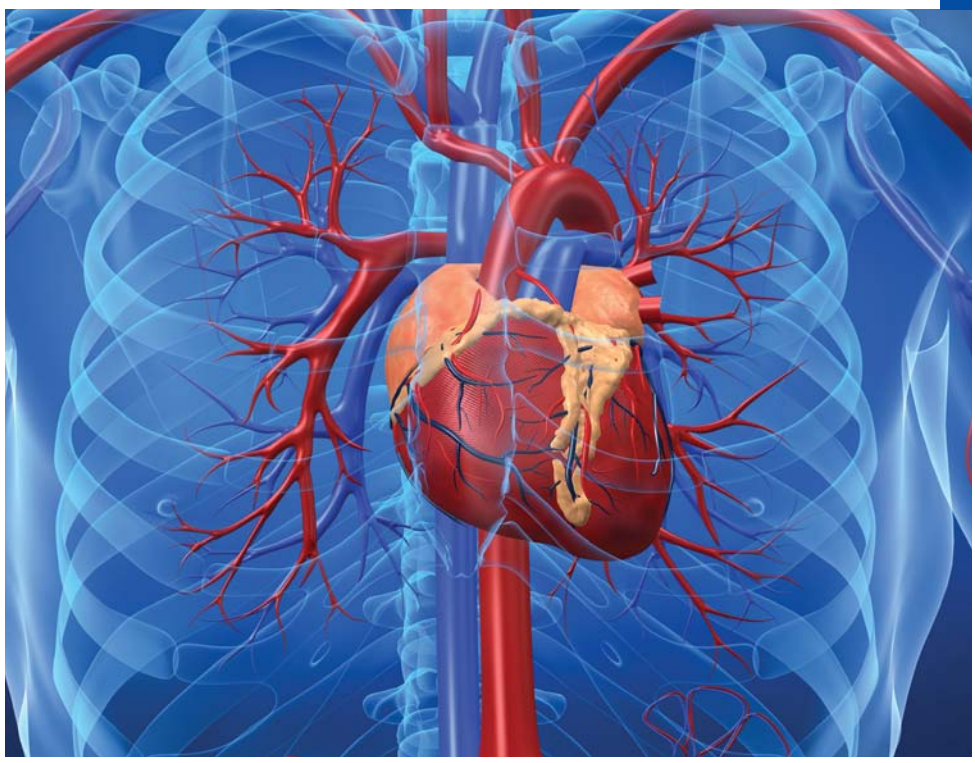
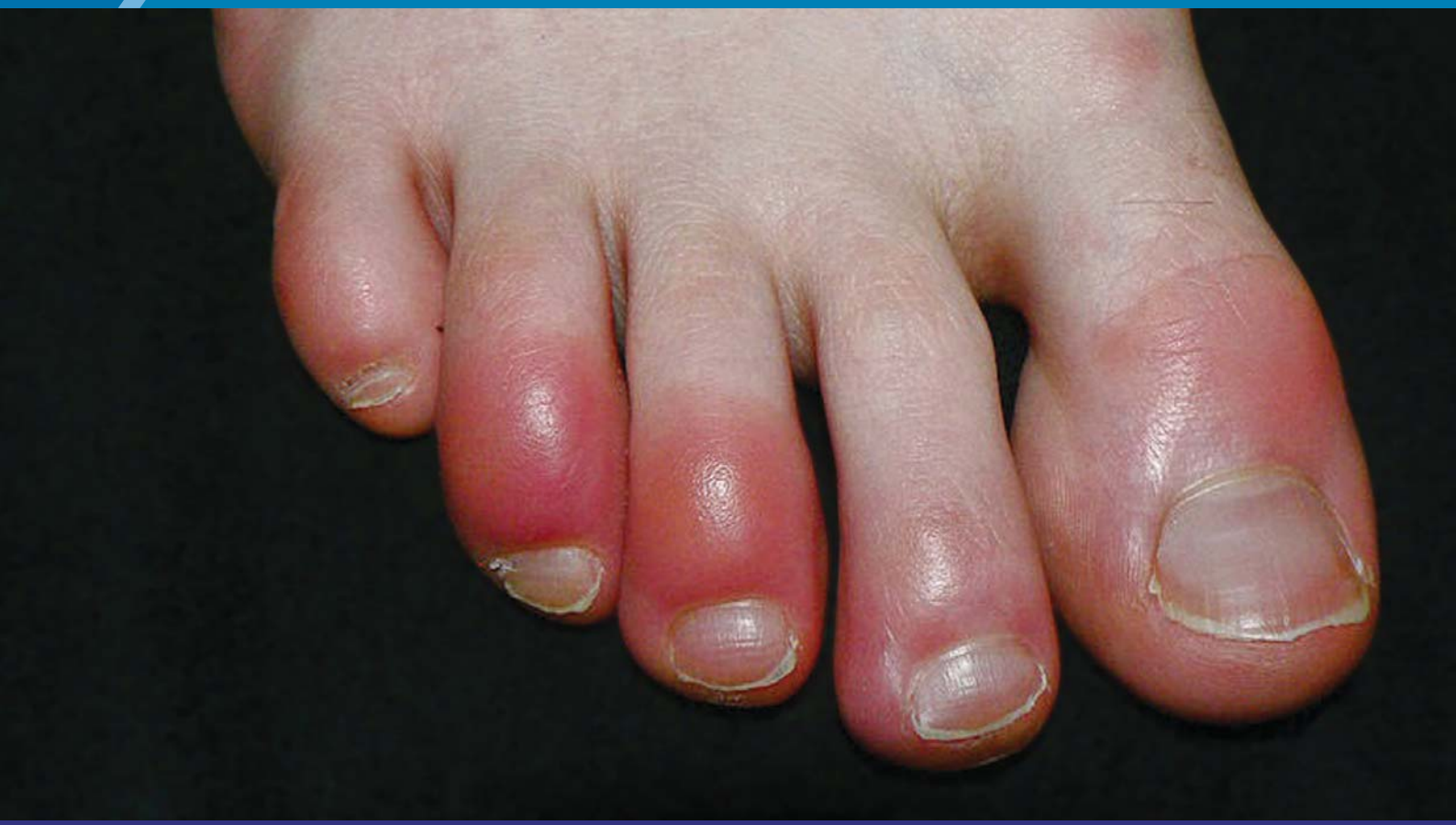
Four facts about human eye

1. Our human eye is 576 megapixel and able to process 36,000 pieces of information in a single hour
2. In the right conditions and lighting, human eye can see the light of a candle from 14 miles away
3. The cornea is the only tissue in the human body which doesn't contain blood vessels
4. Eyeballs stay the same size from birth to death, while nose and ears continue to grow

Four facts about human heart

1. The heart supplies purified blood almost 7.5 trillion cells of human body everyday
2. A woman's heart beats faster than a man's heart. Average heart rate is 78 per minute in woman and 70 per minute in man
3. The amount of energy produce by human heart is enough to drive up the moon from earth and back
4. Anatomy of heart is first describe by a French Anatomy Professor, Raymond Vieussens in 1706





Buerger's disease

Introduction

Buerger's disease also known as thromboangiitis obliterans is a chronic disease characterised by segmental inflammation and thrombosis of the small and medium sized arteries and veins of the peripheral upper and lower limbs. The thrombus leads to arterial ischaemia in the distal extremities and superficial thrombophlebitis, which may progress to gangrene and ulceration. The aetiology is unknown but the use of tobacco is the key factor in the development and progression of the disease. There is evidence that autoimmune factors may be involved. The pathophysiology is thought to involve endothelial cells, platelets, leukocytes and sensory neurons. In addition to smoking, male gender, genetic factors, infectious agents and mental stress due to poor socio-economic circumstances have all been suggested as possible trigger factors. Cardiovascular risk factors may also be important, especially glucose intolerance.

Clinical feature

The onset of Buerger's disease occurs between 40 and 45 years of age, and men are most commonly affected. It begins with ischemia of the distal small vessels of the arms, legs, hands and feet. Involvement of the large arteries is unusual and rarely occurs in the absence of occlusive disease of the small vessels. Patients may present with claudication of the feet, legs, hands and arms. The pain typically begins in the extremities, but may radiate to more central parts of the body. As the disease progresses, typical calf claudication and eventually ischemic pain at rest and ischemic ulcerations on the toes, feet or fingers may develop. Limbs that are clinically not affected could present arteriographic abnormalities. Other signs and symptoms of the disease may include numbness and/or tingling in the limbs, skin ulcerations and gangrene of the digits. Superficial thrombophlebitis and Raynaud's phenomenon occur in approximately 40% of patients with Buerger's disease.

Although Buerger's disease most commonly affects the small and medium sized arteries and veins in the arms, hands, legs and feet, it has been reported in many other vascular beds. There are case reports of involvement of the cerebral and coronary arteries, aorta, intestinal vessels, and even multiple organ involvement. However, gastrointestinal involvement of Buerger's disease remains rare.

Diagnostic criteria

Since the specificity of Buerger's disease is characterized by peripheral ischemia of inflammatory nature with a self-limiting course, diagnostic criteria should be discussed from clinical point of view. Several different criteria have been proposed for the diagnosis of Buerger's disease.

Diagnostic criteria of Shionoya	Diagnostic criteria of Olin
• Smoking history • Onset before the age of 50 years • Infra-popliteal arterial occlusions • Either arm involvement or phlebitis migrans • Absence of atherosclerotic risk factors other than smoking	• Age under 45 years • Current or recent history of tobacco use • The presence of distal extremity ischemia indicated by claudication, pain at rest, ischemic ulcers or gangrenes and documented by non-invasive vascular testing • Exclusion of autoimmune diseases, hypercoagulable states and diabetes mellitus • Exclusion of a proximal source of emboli by echocardiography or arteriography • Consistent arteriographic findings in the clinically involved and non involved limbs

Diagnostic methods

No specific laboratory test for diagnosing Buerger's disease is available. Unlike other types of vasculitis, in patients with Buerger's disease the acute phase reactions (such as the erythrocyte sedimentation rate and C-reactive protein level) are normal. Recommended tests to rule out other causes of vasculitis include a complete blood cell count; liver function tests; determination of serum creatinine concentrations, fasting blood sugar levels and sedimentation rate; tests for antinuclear antibody, rheumatoid factor, serologic markers for CREST (calcinosis cutis, Raynaud phenomenon, sclerodactyly and telangiectasia) syndrome and scleroderma, and screening for hypercoagulability. Screening for hypercoagulopathy, including antiphosolipid antibodies and homocystein in patients with Buerger's disease is recommended.

The role of modern imaging methods, such as computerized tomography (CT) and magnetic resonance imaging (MRI) in diagnosis and differential diagnosis of Buerger's disease still remains unsettled. In patients with leg ulceration suspected of having Buerger's disease, the Allen test should be performed to assess the circulation in the hands and fingers.

Management

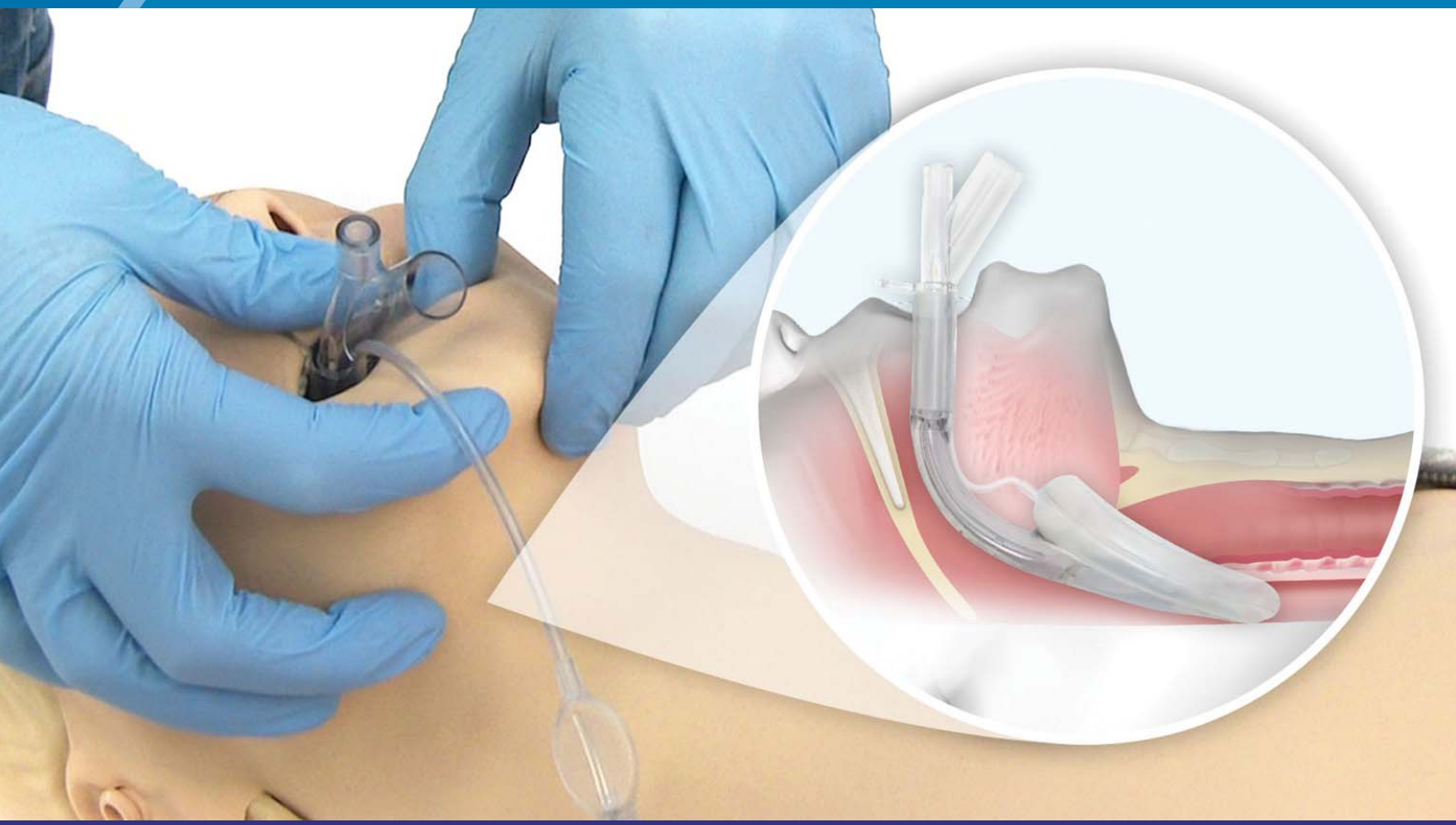
The most effective treatment for Buerger's disease is smoking cessation. It is therefore essential that patients diagnosed with Buerger's disease stop smoking immediately and completely in order to prevent progression of the disease and avoid amputation. Early treatment is also important, because Buerger's disease may provoke social problems that influence quality of life. If there is no gangrene when the patient discontinues smoking, amputation is avoided. Supportive care should be directed towards maximizing blood supply to the affected limbs. Care should be taken to avoid thermal, chemical or mechanical injury, especially from poorly fitting footwear or minor surgery of digits, as well as fungal infection. Vasoconstriction provoked by cold exposure or drugs should be avoided.

Despite the clear role of inflammation in the pathogenesis of Buerger's disease, anti-inflammatory agents, such as steroids, have not been shown to be of real benefit. The results of intravenous therapy with iloprost (a prostaglandin analogue) show that this drug is superior to aspirin in providing total pain relief at rest and complete healing of all trophic changes. It diminishes the risk of amputation. Although acetylsalicylic acid (aspirin) is often prescribed to patients with Buerger's disease, the benefit of this or other orally administered anti-clotting agents has not been confirmed by controlled studies. Intra-arterial thrombolytic therapy with streptokinase has been tested in some patients with gangrene or pre-gangrenous lesions of the toes or feet, with some success in avoiding amputation.

For patients with Buerger's disease, arterial revascularization is usually not possible due to the diffuse segmental involvement and distal nature of the disease. The benefit of bypass surgery to distal arteries also remains controversial because of the high incidence of graft failure. However, if the patient has severe ischemia and there is a distal target vessel, bypass surgery with the use of an autologous vein should be considered.

Sympathectomy may be performed to decrease arterial spasm in patients with Buerger's disease. A lapraroscopic method for sympathectomy has also been used. Sympathectomy has been shown to provide short term pain relief and to promote ulcer healing in some patients with Buerger's disease, but no long term benefit has been confirmed. Spinal cord stimulator and vascular endothelial growth factor gene therapy have been used experimentally in patients with Buerger's disease with promising results.

References: *1. Orph. J. of Rar. Dise., 2006, Vol. 1, N.14:1-5*
2. patient.info/doctor/buergers-disease-pro



Laryngeal mask airway in medical emergencies

Overview

During cardiopulmonary resuscitation (CPR), ventilation and chest compressions must be provided in a proper manner to be effective. Traditionally, ventilation is established through bag and mask ventilation, which is followed by endotracheal intubation with a cuffed endotracheal tube. However, because the level of skill required for successful placement of an endotracheal tube is high, only health care providers with experience in advanced airway placement techniques should perform this procedure. The laryngeal mask airway (often referred to as LMA) is an alternative airway device that is both efficacious and relatively easy to place, even by novices. It is routinely used for patients who are under general anesthesia, since it provides a patent airway for patients who are breathing spontaneously and for those who are receiving mechanical ventilation. The laryngeal mask airway has been successfully used as an airway device for patients in cardiac arrest, even by personnel with little experience in airway management.

Indications

The laryngeal mask airway is useful for the ventilation of patients in cardiac arrest, both before and after their arrival at the hospital. It has been recommended by the American Heart Association and the European Resuscitation Council as an acceptable device for use by non experts in endotracheal intubation when performing emergency airway management. The laryngeal mask airway can be used as an alternative to traditional bag and mask ventilation and is particularly useful when there is a need for prolonged resuscitation, when the personnel present do not have the competence to perform endotracheal intubation, when tracheal intubation cannot be performed or has failed and when movement of the head and neck may injure the patient.

Equipment and anatomy

All laryngeal mask airways contain an inflatable cuff, a connecting tube, a standard connector, and a tube for cuff inflation. Currently available laryngeal mask airways have either precurved or straight tubes or are designed for either a single use or multiple uses (Figure 1). Some devices have a separate channel for suctioning the stomach, and others are designed to facilitate orotracheal intubation after the laryngeal mask airway has been placed. The materials required for placement of the laryngeal mask airway include the device itself, in a size appropriate for the patient's weight, a syringe

for inflating the airway cuff (preferably with just enough air to achieve an adequate seal usually 20 to 40 ml for an adult sized laryngeal mask airway), lubricating jelly, and plastic or cloth tape to secure the device. Often, single use laryngeal mask airways come prepackaged with these supplies. A bag and valve mask device is required for the administration of positive pressure ventilation to the lungs through the laryngeal mask airway, and a stethoscope should be available to confirm breath sounds after placement of the device. It can be helpful to review the pharyngeal anatomy, including the tongue, hard palate, epiglottis, laryngeal inlet, and esophagus, to better understand the proper placement of the laryngeal mask airway and to aid in troubleshooting.

Preparation of the laryngeal mask airway

The laryngeal mask airway is prepared by lubricating the flat, posterior surface of the cuff. Most manufacturers recommend removing all air from the cuff before insertion. It is also important to make sure that all necessary supplies are present before insertion. Personal protective equipments including gloves, eye protection, and a face mask should be used if possible.

Placement of the laryngeal mask airway

With the patient in the supine position, stand behind the patient's head. Physician may open the patient's mouth with his hand, or physician may gently tilt the head backward. Do not perform the backward head tilt in an unconscious patient who is suspected of having a neck injury. For placement of a straight laryngeal mask airway, grasp the tube with the dominant hand, with the curved portion of the tube and the flat side of the cuff facing the patient.

Successful insertion depends on keeping the leading edge flat at the time of insertion. Place the first finger in the space between the tube and the cuff. Using the first finger, direct the cuff upward against the hard palate (maintaining constant upward pressure of the cuff on the hard palate at all times during insertion), and then guide it above the tongue and down through the oropharynx in a smooth, continuous motion. Continue the insertion until physician encounter the resistance, at which time 7 to 10 cm of the tube usually continues to protrude from the patient's mouth. To place a precurved laryngeal mask airway, hold the connecting tube so that the flat side of the cuff is pointing toward the patient; physician's hand should be in a neutral position, with the thumb on the upward surface of the tube. As with the straight tube, the flat upward side of the cuff is inserted along the hard palate with the use of firm pressure.

Guide the tube behind the tongue and toward the larynx using a circular motion of the wrist. Regardless of type of tube, placement should be accomplished with gentle pressure and without the use of force. Once the tube has been fully inserted, inflate the laryngeal balloon with approximately 30 ml of air (for adult sized laryngeal mask airways). Do not hold the tube while the air is being introduced; during inflation, the laryngeal mask airway often slides out of the mouth by 1 to 2 cm as it settles into its proper location. The final resting position is posterior to the tongue, at the laryngeal inlet. The cuff should not be visible when the tube is in its proper position. Connect the laryngeal mask airway to a positive pressure ventilation system verify placement through the auscultation of breath sounds, and secure the device with tape.

Figure 1: Types of commonly used laryngeal mask airways

Complications

The complications associated with the use of a laryngeal mask airway are similar to those seen with other instruments used for airway management. However, in some situations, the risk of certain complications may be lower than when bag and mask ventilation or endotracheal intubation is used. These complications include the potential for upper airway trauma, tooth dislodgment or damage, and the introduction of air into the stomach. Despite its numerous advantages for rescue ventilation, the laryngeal mask airway does not provide a secure airway and will not protect against pulmonary aspiration when the volume of gastric contents is large or under pressure. It may be difficult to ventilate a patient with a laryngeal mask airway when airway or thoracic pressures are high.

Summary

The laryngeal mask airway is a fast and effective alternative to endotracheal intubation and is used for an increasing number of clinical indications, including ventilation during cardiopulmonary arrest. Placement of this device is usually successful on the first attempt. Thus, it is an important procedure to learn and practice.

Reference: *N. Eng. J. Med., 14 November 2013, Vol. 369, No. 20:e26 (1-4)*

A case of intra-arterial thrombolysis with alteplase in a patient with hypothenar hammer syndrome but without underlying aneurysm

Abstract

Hypothenar hammer syndrome is a cause of symptomatic ischemia of the hand secondary to the formation of aneurysm or thrombosis of the ulnar artery in the setting of a complete or incomplete palmar arch. Acute occlusive thrombus or embolus of the hand represents a complex problem that often may require immediate surgical intervention. We report a case from "SAGE Open Medical Case Reports" of acute unilateral arterial hand ischemia requiring catheter directed thrombolysis with alteplase therapy in a patient with acute occlusive arterial thrombosis of the left ulnar artery.

Introduction

Vascular occlusive syndromes of the upper extremity are rare and often secondary to repetitive trauma, atherosclerosis, embolic events and hypercoagulable states. The symptoms can include pain, paresthesias and cold intolerance. Hypothenar hammer syndrome occurs when repetitive impact to the palmar aspect of the wrist and hand at the hypothenar eminence causes damage to the underlying ulnar artery. This damage may manifest clinically as in situ thrombosis or distal embolization with or without underlying aneurysm. This results in acute ischemia of the digits. Typically, this occurs in the setting of an incomplete palmar arch and therefore an inability of the radial artery to compensate for the ischemia.

Hypothenar hammer syndrome may occur in sports and occupations where the heel of the hand is used as a hammer or is subject to repeated force. Besides cases of acute trauma, it may occur in the dominant hand of players of racquet, stick, club sports, volleyball and practitioners of the martial arts. Other occupations such as auto mechanics, metal workers, miners, machinists, butchers, bakers, carpenters and brick layers are also at risk. It typically occurs in middle aged men who present with unilateral symptoms which upon physical exam are suggestive of vascular

pathology. Angiography is diagnostic. Conservative approaches such as cessation of offending activity, calcium channel blockers, antiplatelet therapy or anticoagulants are appropriate in most patients, whereas surgical options and thrombolytic therapy are reserved for patients with severe or refractory symptoms.

Case

A 57 year old gentleman presented to the emergency department with a 1.5 week history of progressively worsening pain and swelling that began in left hand and progressed to second to fifth distal interphalangeal joints (DIP). Initially, he felt that his fingers started to feel numb, which went away as the day progressed. However, several days prior to presentation, he noted persistent numbness with new discoloration of his fingertips that began to worsen as well. Upon presentation to the hospital, his left ulnar pulse was not palpable, and his left second to fifth fingertips appeared deeply cyanotic with early gangrenous changes. He denied any other symptoms.

He had a past medical history significant for anxiety, depression, alcohol abuse and chronic back pain. He was an active smoker of about 16 years. He also had a remote history of heroin and cocaine use, and his last relapse was documented over 10 years ago. He was a former truck driver who drove mostly with his left hand despite being right hand dominant.

On examination, his left second to fifth fingertips appeared profoundly cyanotic with early gangrenous changes. His fingers were cool to the touch. He had decreased sensation in left hand and was unable to make a fist. His left radial pulse was palpable but the ulnar pulse was not. His physical exam including contralateral arm neurovascular exam was otherwise unremarkable. He had a Doppler signal in the distal ulnar artery and in the palmar arch. Duplex examination of the upper extremity revealed an acute occlusive thrombus of the left distal ulnar artery suggestive of hypothenar hammer syndrome. He was taken for angiographic assessment.

Angiography from a right femoral retrograde approach revealed normal left upper extremity arterial tree with the exception of an ulnar occlusion at the wrist. The wire crossed the occlusion easily consistent with acute thrombosis. The deep arch and digital vessels reconstituted distally. Aspiration with a glide catheter yielded thrombus. Thrombolysis was therefore initiated with a 4-F UniFuse catheter with an infusion length of 20 cm. It was placed in the ulnar artery distally with about 5 cm hanging into the brachial artery across the bifurcation proximally (Figure 1).

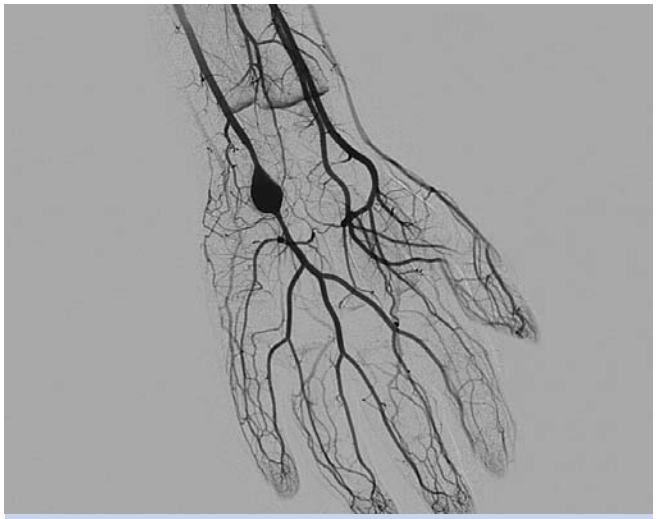


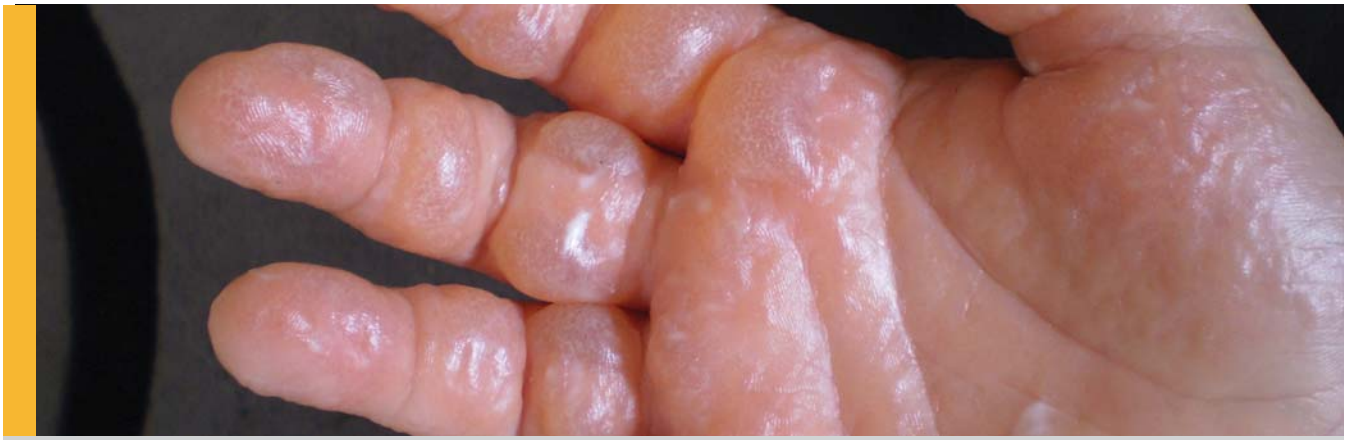
Figure 1: Initial angiogram revealing occlusion of the ulnar artery distally at the wrist

The catheter directed thrombolytic regimen consisted of alteplase 1 mg/h and intravenous heparin. Heparin was infused through the sheath side arm at a rate of 500 units/h. He was admitted to the intensive care unit where all patients undergoing thrombolytic infusions are observed per protocol. Repeat angiography was conducted after about 20 h of infusion (about 20 mg of alteplase). This revealed a widely patent ulnar artery and resolution of the thrombus with antegrade filling of the deep palmar arch. The digital vessels were seen now to continue from the arch to the digits. The ulnar artery appeared overall normal without corkscrew appearance. This confirmed the diagnosis of hypothenar hammer syndrome with ulnar artery occlusion but without underlying aneurysm. The thrombolytic infusion was discontinued but therapeutic anticoagulation was subsequently continued, and the patient was discharged on rivaroxaban therapy. The patient was initially given rivaroxaban therapy of 15 mg twice daily for 21 days followed by 20 mg once daily and continues on that dose.

Conclusion

Catheter directed thrombolytic therapy is a useful and important tool in the armamentarium for the treatment of acute limb threatening events. While off label, discussion of the potential complications and thorough risk benefit analysis with the patient may provide for an excellent alternative to open surgical revascularization. Alteplase infusion for acute limb threatening ischemia, even in the upper extremity, can provide an excellent option to treat hypothenar hammer syndrome especially without underlying correctable anatomic defect such as aneurysm.

Reference: *SAGE Open Medical Case Reports, 13 November 2017, Vol. 6:1 -3*



Extravasation of peripherally administered parenteral nutrition

A 60 year old woman with advanced Huntington's disease and a recent episode of aspiration pneumonia presented with erythema, edema, and blistering of the right forearm. An 18 gauge peripheral intravenous catheter had been partially displaced from the same site 10 hours earlier, resulting in infusion of an unknown quantity of parenteral nutrition solution (80 gm of dextrose per liter, 22 gm of amino acids per liter, 200 kcal of lipids per liter and 750 mOsm per liter) into the interstitial space. Peripherally delivered parenteral nutrition can act as a vesicant and cause blistering. Its potential to do so varies with the infusion site, rate of administration, solution composition, and total osmolarity. In this patient, treatment with sterile dressings, systemic glucocorticoids, and systemic antibiotic agents resulted in complete healing in 2 months.

Reference: *N. Eng. J. Med., 10 March 2011, Vol. 364, No. 10:e20*

Podoconiosis

A 36 year old man presented to an Ethiopian clinic with a 20 year history of skin nodules, pain, and edema involving his legs and feet. He was otherwise healthy and worked as a farmer. Circulating filarial antigen tests for the presence of *Wuchereria bancrofti* were negative; he was not tested for other types of filaria. This clinical presentation prompted a diagnosis of probable podoconiosis (also known as nonfilarial elephantiasis or mossy foot). This locally endemic, noninfectious condition is caused by the long term exposure of susceptible persons to irritant volcanic soil. Colloid particles are thought to be absorbed through the skin and taken up by macrophages, leading to lymphatic fibrosis and elephantiasis. Affected persons are typically barefoot agricultural workers in the highland tropics. Podoconiosis is preventable with fastidious shoe wearing and foot hygiene. Treatment is limited to compression bandaging and elevation. The patient was instructed to wear shoes, but additional nodules continued to develop on uncovered areas of his sandaled feet.

Reference: *N. Eng. J. Med., 24 March 2011, Vol. 364, No. 12:e23*

Management of Asthma

A chronic inflammatory airway disorder, Asthma is marked by airway hyper responsiveness with recurrent Episodes of wheezing, coughing, tightness of the chest, and shortness of breath. Typically, these episodes are associated with airflow obstruction that may be reversed spontaneously or with treatment. Asthma affects approximately 300 million people around the world. In children, males have a higher Asthma risk; in adults, females have a higher prevalence. Experts believe Asthma results from various host factors, environmental factors, or a combination. Host factors include gender, obesity, and genetics. Genetic factors include atopy. Defined as a genetic tendency to develop allergic diseases, such as Asthma and allergic rhinitis, atopy commonly is linked to an immunoglobulin E (IgE) mediated response to allergens. However, in this review we have discussed about pathophysiology, triggering factors, diagnosis and management of Asthma.

Pathophysiology

Airflow limitation in Asthma is recurrent and caused by a variety of changes in the airway. These include:

Bronchoconstriction: In Asthma, the dominant physiological event leading to clinical symptoms is airway narrowing and a subsequent interference with airflow. In acute exacerbations of Asthma, bronchial smooth muscle contraction occurs quickly to narrow the airways in response to exposure to a variety of stimuli including allergens or irritants. Allergen induced acute bronchoconstriction results from an IgE dependent release of mediators from mast cells that includes histamine, tryptase, leukotrienes and prostaglandins that directly contract airway smooth muscle. Aspirin and other non steroidal anti-inflammatory drugs can also cause acute airflow obstruction in some patients and evidence indicates that this non IgE dependent response also involves mediator release from airway cells. In addition, other stimuli (including exercise, cold air, and irritants) can cause acute airflow obstruction. Stress may also play a role in precipitating Asthma exacerbations. The mechanisms involved have yet to be established and may include enhanced generation of pro inflammatory cytokines.

Airway edema: As the disease becomes more persistent and inflammation more progressive, other factors further limit airflow. These include edema, inflammation, mucus hypersecretion and the formation of inspissated mucus plugs, as well as structural changes including hypertrophy and hyperplasia of the airway smooth muscle.

Airway hyper responsiveness: Airway hyper responsiveness an exaggerated broncho constrictor response to a wide variety of stimuli is a major, but not necessarily unique, feature of Asthma. The degree to which airway hyper responsiveness can be defined by contractile responses to challenges with methacholine correlates with the clinical severity of Asthma. The mechanisms influencing airway hyper responsiveness are multiple and include inflammation, dysfunctional neuro regulation and structural changes. Inflammation appears to be a major factor in determining the degree of airway hyper responsiveness.

Airway remodeling: In some persons who have Asthma, airflow limitation may be only partially reversible. Permanent structural changes can occur in the airway; these are associated with a progressive loss of lung function. Airway remodeling involves an activation of many of the structural cells, with consequent permanent changes in the airway that increase airflow obstruction and airway responsiveness. These structural changes can include thickening of the sub basement membrane, sub epithelial fibrosis, airway smooth muscle hypertrophy, hyperplasia, blood vessel proliferation and dilation and mucous gland hyperplasia and hyper secretion.

Features of airway remodeling

- Inflammation
- Mucus hyper secretion
- Sub epithelial fibrosis
- Airway smooth muscle hypertrophy
- Angiogenesis

Triggering factors

The airways of asthma patients are highly sensitive to certain things, which a people without asthma does not bother. These things are called triggers. When an asthma patient comes into contact with them, an asthma episode starts. The airways become swollen, produce too much mucous and are tightened up. Common triggering factors of asthma are given in Table 1.

Table 1: Triggering factors of Asthma

Allergens

1) **Outdoor allergens**

Pollen: From grass, trees and flowers

Molds: From some fungi

2) **Indoor allergens**

House dust, mites

Dander from skin, hair, feathers or excreta of warm blooded pets (e.g., dogs, cats, birds and rodents)

Insects: cockroach

3) **Food allergens**

Food allergens rarely cause an asthma attack. Though some foodstuff may cause allergy in some people, it is not wise to ban allergy producing foods in general for an asthmatic. Advice to avoid those food only which evoke an asthma/allergy attack within few minutes or hours after intake.

Common allergy producing foods are:

Beef, prawn, hilsha and some other fishes, seafood, duck egg, cow's milk, some vegetables and nuts.

Food additives: metabisulphate, tartrazine

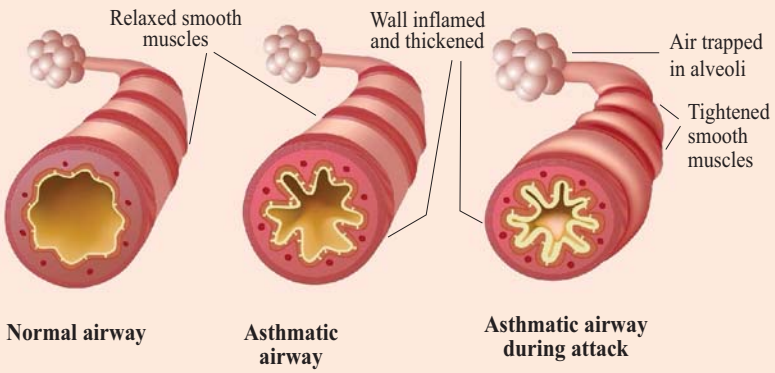
Irritants

Tobacco smoke, wood smoke, smoke from gas and other cooker, strong odors, perfume and sprays, cosmetics, paints, cooking of spices, toxic gases from automobiles and factories.

Others

- Upper respiratory tract infection: Viral infections, common cold
- Exercise: Strenuous physical activities
- Certain drugs: Beta blockers (even eye drops), aspirin and NSAIDs
- Changes in season, weather and temperature: Asthmatics experience more exacerbation during specific season (more in winter) and during the period of season change. It is also provoked during cold /or hot, humid days, during first and full moon and during thunder storms. These triggers are person specific and their underlying mechanism is poorly understood. It is noted that, asthma attack is likely if temperature lowers for 3^{o}C or more than the previous day
- Stress:
  - Emotion e.g., laughing, crying, sobbing, anxiety, mental depression
  - Surgery
  - Pregnancy
  - Fear of an impending attack

Difference between normal airway and Asthmatic airway



Diagnosis

There is no accepted standard diagnostic test for Asthma. Making the diagnosis of Asthma requires a critical evaluation of the patient's symptoms, medical history, physical examination, and diagnostic tests.

Clinical feature

The first step in making the diagnosis of Asthma is reviewing the patient's history and formulating the pre test probability of Asthma (Table 2). Patients typically present with intermittent symptoms of cough, wheeze, dyspnea or chest discomfort. These symptoms are often exacerbated by identifiable triggers such as tobacco smoke, perfume, pets, workplace exposure, or upper respiratory tract infection. Patients may experience symptoms during the daytime, nighttime or with exercise and the symptoms may vary depending on the time of year. Asthma is often associated with a history of atopy, and this association in a symptomatic patient is one of the strongest predictors of Asthma. Thus personal and family histories of allergies are key components of the medical history. Other important information to elicit includes early childhood breathing problems, occupational exposures, sensitivity to aspirin or non steroidal anti-inflammatory pain relievers, nasal polyposis, or sinusitis.

Table 2: Clinical features of Asthma

Symptoms

- Cough
- Wheeze
- Shortness of breath
- Chest tightness

History

- Environmental triggers
- Atopic or allergic history
- Symptoms with exercise
- Sensitivity to aspirin or non steroidal anti-inflammatories
- Nasal polyposis
- Family history of Asthma or allergies

Physical examination

- Hyper expansion of the chest cavity
- Prolonged expiratory time
- Expiratory wheezing
- Decreased air movement
- Use of accessory respiratory muscles
- Rash or eczema

Cough variant asthma is a subset of asthma characterized by cough as the predominant or sole symptom. In patients with chronic cough, asthma should always be considered as a possible diagnosis. The diagnostic and therapeutic approaches are similar to those for the typical form of asthma. The diagnosis of cough variant asthma should be confirmed by the resolution of cough in response to asthma therapy. Physical examination can be normal but often reveals wheezing, chest hyperinflation, or a prolonged expiratory phase, especially when patients are symptomatic. The use of accessory muscles may be apparent during a more severe exacerbation. Examination for signs of allergic rhinitis, conjunctivitis, and dermatitis should also be done.

Diagnostic testing

Spirometry: Spirometry is a pulmonary function test (PFT) that measures the amount (volume) or speed (flow) of air that can be inhaled and exhaled. The patients is typically asked to breathe normally and then to take the deepest possible breath and then exhale as quickly and as hard as possible. From that maneuver the forced expiratory volume in the first second (FEV_1) of exhalation is measured and compared to the entire volume of air that can be expelled in a forced expiration (forced vital capacity - FVC). Spirometry is indicated as part of the initial diagnostic evaluation for Asthma in all patients ³ 5 years old to test for airflow obstruction, the severity and the short term reversibility.

Bronchodilator response testing: Patients who have airflow obstruction on spirometry should undergo bronchodilator response testing. This is done by administering 2 to 4 puffs from an albuterol inhaler (90 g/puff), via a spacer or valved holding chamber. After waiting for 10 to 15 min, spirometry is repeated. Short acting anticholinergic agents can also be used but require a delay of more than 30 min before repeating spirometry. An improvement of > 12% or > 0.2 L in baseline FEV_1 or FVC has traditionally defined reversible airflow obstruction. An increase of ³ 10% of the

How to make a diagnosis of Asthma

Compatible clinical history plus either/or:

- FEV_1 ³ 15%* (and 200 ml) increase following administration of a bronchodilat/trial of corticosteroids
- > 20% diurnal variation on ³ 3 days in a week for 2 weeks on PEF (peak expiratory flow) diary
- FEV_1 ³ 15% decrease after 6 mins of exercise

** Global Initiative for Asthma (GINA) definition accepts an increase of 12%.*

predicted value is another criterion that has been used, and this may be less subject to bias. The presence of airflow obstruction and a good bronchodilator response is consistent with the diagnosis of Asthma, but lack of a bronchodilator response does not rule out Asthma.

Inhalation challenge test: To assess bronchial hyper reactivity, inhalation challenge tests are safe and useful diagnostic tools. Typically, the patient is exposed to an agent or activity that could provoke bronchoconstriction during serial spirometry. Methacholine is used to directly stimulate airway smooth muscle. Other agents or activities indirectly provoke bronchoconstriction by inducing an airway inflammatory response (e.g., mannitol, cold air, hypertonic saline, exercise). Methacholine challenge is best used in patients with no baseline obstruction who can perform good quality spirometry.

Radio allergosorbent test and allergen skin test: Atopy, a high total immunoglobin E (IgE), any positive allergen skin test or any high specific IgE level increases the probability of Asthma in a patient with respiratory symptoms. Elevated IgE is consistent with the diagnosis of Asthma, but very high IgE (> 1,000 ng/ml) should prompt consideration of allergic broncho pulmonary aspergillosis. Skin testing or in vitro testing for specific IgE antibodies should be based on a careful history to ascertain likely aeroallergen exposures. Skin testing is performed by introducing an allergen into the skin and observing for wheal and flare. Skin test results are available within one hour and are visible to the patient, which may encourage compliance with environmental control practices.

Exhaled nitric oxide: The measurement of certain biomarkers in the diagnosis and assessment of Asthma has gained increased attention. These include induced sputum, exhaled gases and exhaled breath condensate. Induced sputum is collected by asking the patient to inhale nebulized saline and to then expectorate. Exhaled gases such as nitric oxide are measured by having the patient exhale to maintain a specified flow into a balloon or a measuring device. Exhaled breath condensate is obtained by passive breathing through a cooling device that contains a tube to collect the liquid sample. Of these, sputum eosinophil count and exhaled nitric oxide have shown the most promise for diagnosing Asthma. Induced sputum eosinophil count can distinguish patients with and without Asthma and predict responsiveness to inhaled corticosteroids. However, the methods for obtaining and processing the samples are time consuming and not standardized, so the use of induced sputum for assessing Asthma is still most appropriate in the clinical research setting.

Radiographic imaging: Chest radiographs and high resolution computed tomography are often used in diagnosing Asthma, to rule out other lung diseases. The chest radiograph is typically normal in patients with Asthma. Radiographic abnormalities can help identify alternative diseases, such as heart failure (pulmonary vascular congestion) and COPD (emphysematous changes). Parenchymal abnormalities can be detected in diseases such as cystic fibrosis and lymphangioleiomyomatosis.

Management

Setting goals

Asthma is a chronic condition but may be controlled with appropriate treatment in the majority of patients. The goal of treatment should be to obtain and maintain complete control, but may be modified according to the circumstances and the patient. General long term objectives of Asthma management include:

- Achieving symptom control and maintaining normal physical performance
- Minimizing the risk of exacerbations, fixed airway obstruction and side effects of the therapy

Immediate assessment of acute severe Asthma

Acute severe Asthma

- PEF 33% - 50% predicted (< 200 l/min)
- Respiratory rate ³ 25 breaths/min
- Heart rate ³ 110 beats/min
- Inability to complete sentences in 1 breath

Life threatening features

- PEF < 33% predicted (< 100 l/min)
- SpO_2 < 92% or PaO_2 < 8 kPa (60 mmHg) (especially if being treated with oxygen)
- Normal or raised $PaCO_2$
- Silent chest
- Cyanosis
- Feeble respiratory effort
- Bradycardia or arrhythmias
- Hypotension
- Exhaustion
- Confusion
- Coma

Near fatal Asthma

- Raised $PaCO_2$ and/or requiring mechanical ventilation with raised inflation pressures

Control based Asthma management

Modern Asthma treatment is based on the concept of Asthma control, which has been shown to improve the treatment success. This concept is based on a cycle of assess, adjust, and review. Symptom control is usually associated with reduced Asthma exacerbations. In the case of more severe forms, symptom control can occasionally not be paired with a reduced exacerbation rate. That makes it important to consider both factors of Asthma control (symptoms and exacerbation risk). As an alternative, other concepts such as therapy based on sputum or fractional exhaled nitric oxide (FeNO) may be used in special cases, e.g., severe or difficult to treat Asthma.

Drug therapy

Inhalation therapy is the application of choice for Asthma. Three pharmaceutical categories are generally distinguished for long term treatment (Table 3):

Controller: These therapy should be taken regularly. It reduces inflammation and exacerbation risk and controls symptoms.

Reliever: These therapy is taken as necessary to reduce symptoms in case of Asthma exacerbations. Also used for the short term prevention of exercise induced bronchoconstriction. It is a key objective of Asthma management to keep the need for reliever to a minimum.

Add on therapy: Used in patients with severe Asthma and persistent symptoms or exacerbations despite high dose combination therapy with ICS and optimization of modifiable risk factors.

Stepwise approach for adjusting Asthma treatment

The adjustment of Asthma therapy is based on Asthma control, and follows a step up or step down algorithm to increase or reduce the medication (Table 4). Regular follow up should occur in a period of 2 to 3 months to optimize the treatment strategy. The gold standard in asthma therapy is still a low dose ICS as a controller together with an on demand short acting beta-2-agonist (SABA). An LTRA (leukotriene receptor antagonist) can be tried as a second choice. There are also considerations that a combination product with low dose ICS and long acting beta-2-agonist (LABA) should be established in adults at this treatment stage (step 2) to ensure rapid treatment success. A further step up from step 3 for adults calls for ICS and LABA combination therapy, with ICS in low doses. For children over the age of 12 years, an increased ICS dose is preferred over a combination therapy in this case. Two inhalers are also approved for additional rescue therapy, formoterol or budesonide and formoterol or beclomethasone as basic therapy should be administered in the morning and at night and may also be inhaled by patients as needed (exacerbation).

The ICS concentration in the combination product increases with the severity of the disease. Tiotropium may now be added to the management regimen as an additional treatment option. The use of theophylline preparations is still defined in the guidelines, but they are rarely employed in practical situations. A further innovation in the step up algorithm is the application of an add on therapy (e.g., anti IgE for patients with severe asthma, who show a corresponding allergic predisposition prior to a systemic steroid therapy).

In the same way as the step algorithm provides for step up options, it is important to reduce (step down) the therapy after the corresponding controls (after approximately 2 to 3 months) with good asthma control. Again, this requires a highly sensitive approach to quickly detect deteriorating symptoms and lung function, which may indicate an elevated risk of exacerbations. In principle, the goal should be to use the lowest ICS concentration that can guarantee optimal therapeutic success.

Table 3: Medication categories for Asthma treatment

Controller	Reliever	Add-on therapy
• Inhaled corticosteroid (ICS) • ICS/LABA (long acting beta-2-agonist) combination • Leukotriene receptor agonists (LTRA) • Long acting anticholinergics (LAMA) • Methylxanthines (theophylline) • Chromones (practically no longer in use)	• Short acting beta-2-agonists (SABA) • Long acting anticholinergics (LAMA)	• Anti-IgE therapy • Systemic/oral corticosteroids (OCS) • Anti-IL5 therapy • Special (phenotype specific) treatments and interventions by specialized centers

Initial treatment after diagnosis

For the best outcomes, regular daily treatment should be initiated as soon as possible after the diagnosis of Asthma is made, because:

- Early treatment with low dose ICS leads to better lung function than if symptoms have been present for more than 2 to 4 years
- Patients not taking ICS who experience a severe exacerbation have lower long term lung function than those who have started ICS
- In occupational Asthma, early removal from exposure and early treatment increase the probability of recovery

Asthma therapy for preschoolers basically has the same objectives as approaches for older children and adults and also follows a step scheme (step up or step down). In this age group, minimization of pharmaceutical side effects (e.g., body growth limitations from use of ICS) is especially important.

Table 4: Step care management of Asthma

Therapy of choice	Step 1	Step 2	Step 3	Step 4	Step 5
Preferred controller choice		Low dose ICS	Low dose ICS/LABA**	Medium/high dose ICS/LABA	Refer for add-on treatment e.g., tiotropium*† anti-IgE anti-IL5*
Other controller option	Consider low dose ICS	Leukotriene receptor antagonist (LTRA) Low dose theophylline*	Medium/high dose ICS Low dose ICS + LTRA (or + theophylline*)	Add tiotropium*† High dose ICS + LTRA (or + theophylline*)	Add low dose OCS
Reliever	As-needed short-acting beta-2-agonist (SABA)		As-needed SABA or low dose ICS/formoterol#		

Note:
Step 1: Occasional use of inhaled short-acting β_2-adrenoreceptor agonist bronchodilators
Step 2: Introduction of regular preventer therapy
Step 3: Add on therapy
Step 4: Poor control on moderate dose of inhaled steroid and add on therapy: addition of a fourth drug
Step 5: Continuous or frequent use of oral steroids

ICS - Inhaled corticosteroid, LTRA - Leukotriene receptor antagonist, LABA - Long acting beta-2-agonist, OCS - Oral corticosteroid, IgE - Immunoglobulin E, IL5 - Interleukin 5

**Not for children < 12 years. ** For children 6-11 years, the preferred Step 3 treatment is medium dose ICS. # Low dose ICS/formoterol is the reliever medication for patients prescribed low dose budesonide/formoterol or low dose budesonide/formoterol for maintenance and reliever therapy. †Tiotropium by mist inhaler is add-on treatment for patients with a history of exacerbations*.*

Differential diagnosis

Adults

- Chronic obstructive pulmonary disease (COPD)
- Hyperventilation syndrome and panic attacks
- Congestive heart failure
- Pulmonary embolism
- Mechanical obstruction of the airways
- Pulmonary infiltration with eosinophilia
- Vocal cord dysfunction

Infants and children

- Allergic rhinitis and sinusitis
- Foreign body in trachea or bronchus
- Vocal cord dysfunction
- Enlarged lymph nodes or tumor
- Viral bronchiolitis or obliterative bronchiolitis
- Cystic fibrosis
- Bronchopulmonary dysplasia
- Congenital heart diseases
- Dysfunction or gastroesophageal reflux disease

Prognosis

The outcome from acute severe Asthma is generally good. Death is fortunately rare but a considerable number of deaths occur in young people and many are preventable. Failure to recognize the severity of an attack, on the part of either the assessing physician or the patient, contributes to delay in delivering appropriate therapy and to under treatment. Prior to discharge, patients should be stable on discharge medication (nebulized therapy should have been discontinued for at least 24 hours) and the PEF should have reached 75% of predicted or personal best. The acute attack should prompt a look for and avoidance of any trigger factors, the delivery of Asthma education and the provision of a written self management plan. The patient should be offered an appointment with a general practitioner or Asthma nurse within 2 working days of discharge, and follow up at a specialist hospital clinic within a month.

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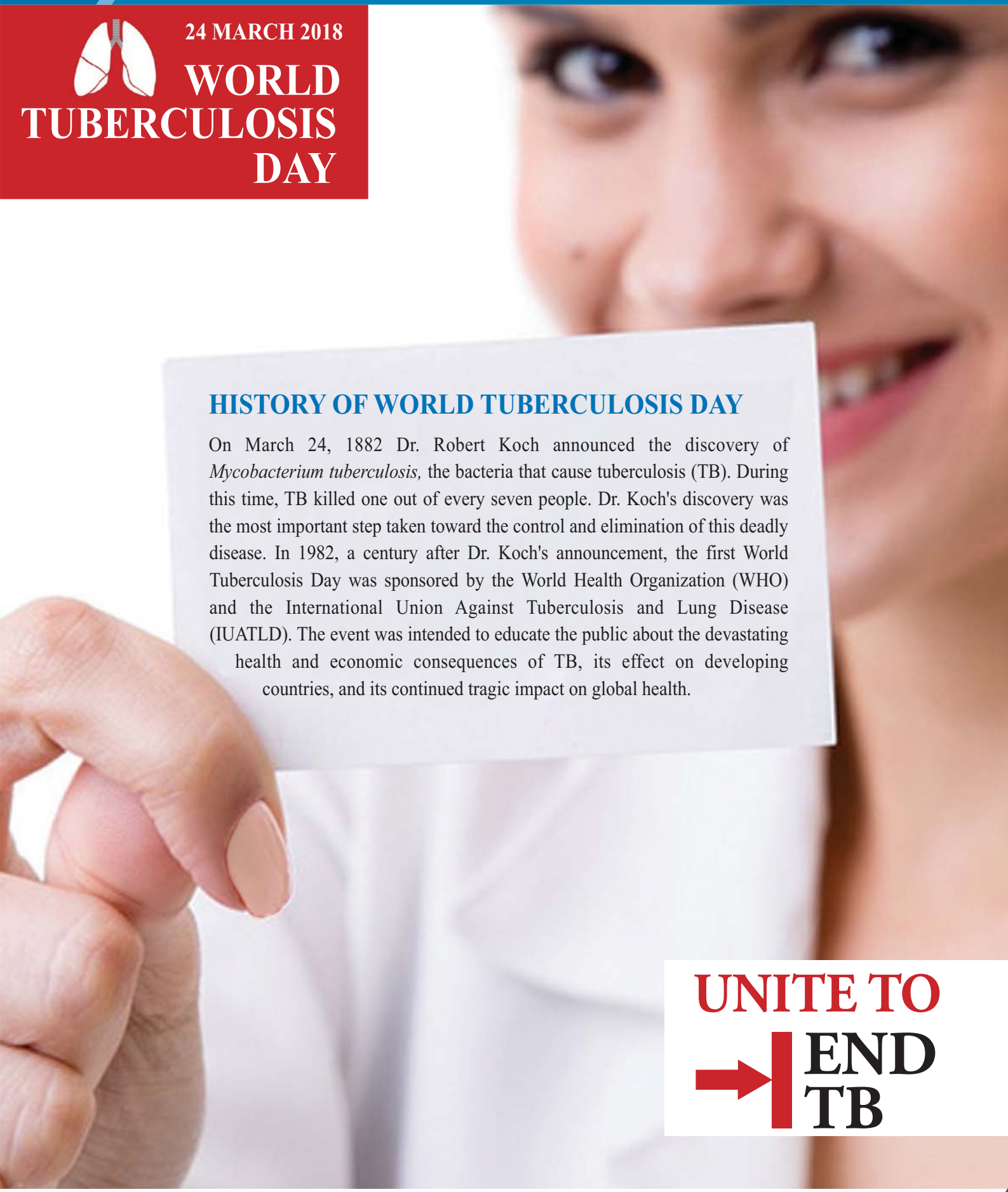
HEALTH DAY

24 MARCH 2018

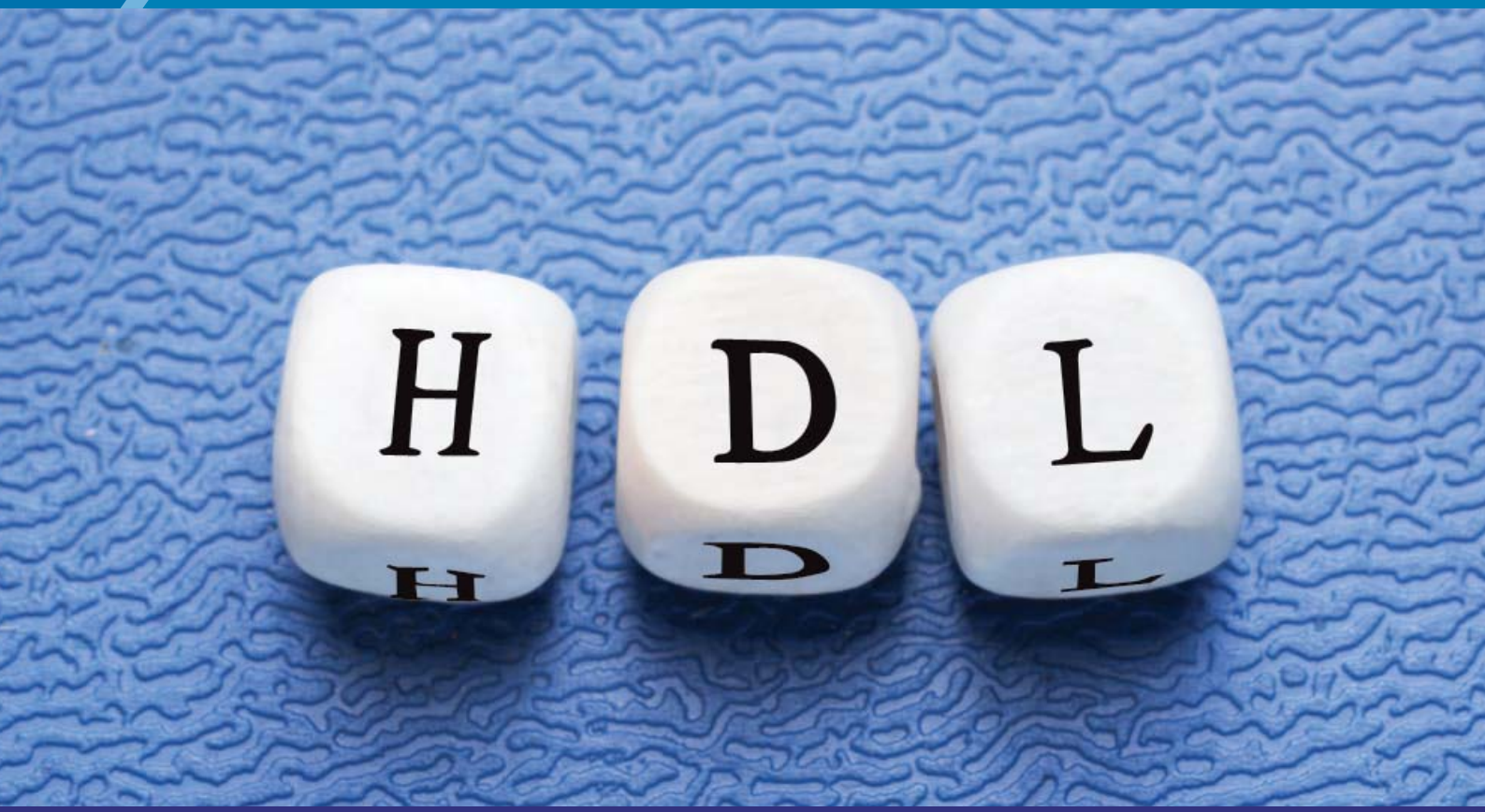
WORLD TUBERCULOSIS DAY



HISTORY OF WORLD TUBERCULOSIS DAY

On March 24, 1882 Dr. Robert Koch announced the discovery of *Mycobacterium tuberculosis,* the bacteria that cause tuberculosis (TB). During this time, TB killed one out of every seven people. Dr. Koch's discovery was the most important step taken toward the control and elimination of this deadly disease. In 1982, a century after Dr. Koch's announcement, the first World Tuberculosis Day was sponsored by the World Health Organization (WHO) and the International Union Against Tuberculosis and Lung Disease (IUATLD). The event was intended to educate the public about the devastating health and economic consequences of TB, its effect on developing countries, and its continued tragic impact on global health.

UNITE TO END TB



'Good' cholesterol might actually be bad

Traditionally, we have been told by physicians not to worry about "good" cholesterol, which is scientifically known as high density lipoprotein (HDL). New research finds an alarming association between high levels of this cholesterol type and excessive mortality. A new study published in the European Heart Journal finds that "good" cholesterol, or high density lipoprotein (HDL) cholesterol, may raise the risk of premature death. By and large, the medical community suggest that higher levels of the good kind of cholesterol are desirable, as it may protect against heart disease and stroke. By contrast, it is the "bad" cholesterol, or low density lipoprotein (LDL), that blocks the arteries. It is the first time that a study has drawn a connection between high HDL cholesterol levels and excessive mortality in the general population. Madsen and colleagues combined data from the Copenhagen City Heart Study, the Copenhagen General Population Study, and the Danish Civil Registration System. In total, they examined data on more than 116,000 people and clinically followed them for an average period of 6 years. During which time more than 10,500 people died. Blood tests for both types of cholesterol levels were taken non fasting, and statistically, the researchers adjusted for all known variables that are normally associated with all cause mortality. Such factors included age, body mass index (BMI), smoking, alcohol consumption, physical activity, and diabetes. Overall, 0.4% of the men and 0.3% of the women had extremely high levels of HDL in their blood. Extreme levels were defined as equal to or higher than 3.0 millimoles per liter for men, and equal to or higher than 3.5 millimoles per liter for women. The study found that men with extreme levels of HDL in their blood had a 106% higher chance of dying prematurely than men with normal levels of this type of cholesterol. Women with extremely high levels of HDL cholesterol were 68% more likely to die prematurely than women with normal levels. Additionally, the mortality rate in men with "very high" levels of the supposedly good kind of cholesterol also had a 36% higher mortality rate than men with normal levels.

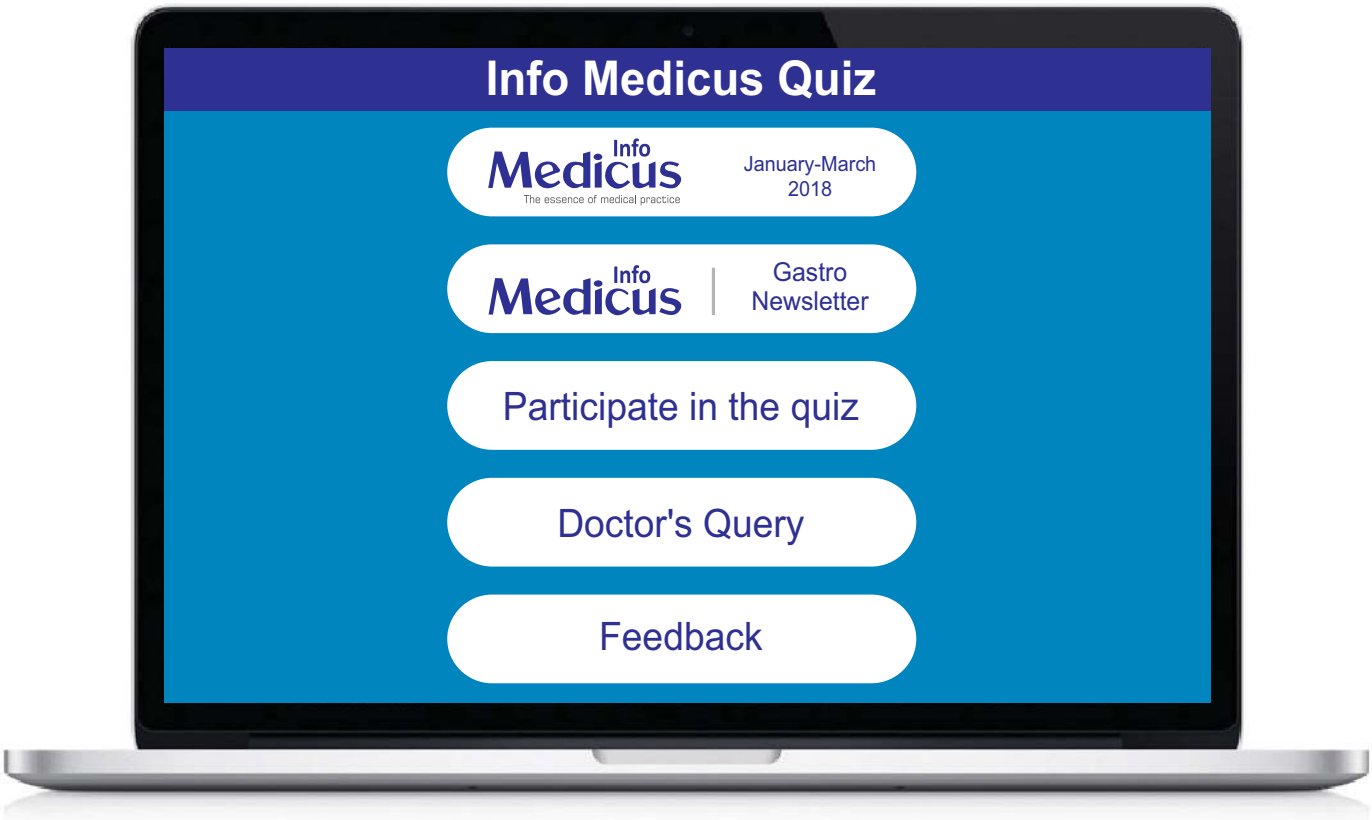
Reference: *www.medicalnewstoday.com*

How video games affect the brain

More than 150 million people in the United States play video games regularly or for at least 3 hours per week. The average American gamer is a 35 year old adult, with 72% of gamers aged 18 or older. For video game use by children, most parents 71% indicate that video games have a positive influence on their child's life. A growing body of evidence, however, shows that video gaming can affect the brain and furthermore, cause changes in many regions of the brain. A total of 22 of the reviewed studies explored structural changes in the brain and 100 studies analyzed changes in brain functionality and behavior. Results of the studies indicate that playing video games not only changes how brains perform but also their structure. For example, video game use is known to affect attention. The studies included in the review show that video game players display improvements in several types of attention, including sustained attention and selective attention. Furthermore, the regions of the brain that play a role in attention are more efficient in gamers compared with nongamers, and they require less activation to stay focused on demanding tasks. Game addicts have functional and structural changes in the neural reward system. Evidence also demonstrates that playing video games increases the size and competence of parts of the brain responsible for visuospatial skills - a person's ability to identify visual and spatial relationships among objects. In long term gamers and individuals who had volunteered to follow a video game training plan, the right hippocampus was enlarged. Researchers have discovered that video gaming can be addictive, a phenomenon known as "Internet gaming disorder." In gaming addicts, there are functional and structural alterations in the neural reward system, a group of structures associated with feeling pleasure, learning, and motivation. Researchers have recently collected and summarized results from 116 scientific studies to determine how video games can influence the brains and behaviors. The findings of their review were published in Frontiers in Human Neuroscience. "Games have sometimes been praised or demonized, often without real data backing up those claims. Moreover, gaming is a popular activity, so everyone seems to have strong opinions on the topic," says Marc Palaus, first author of the review.

Reference: www.medicalnewstoday.com

January
March
2018



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