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Common skin diseases

Contents

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Editorial

Dear Doctor,

We are happy to hand you over another edition of Info Medicus, the result of our continuous hard work to keep you informed about latest medical advancement and the best way of showing our gratitude to you for your remarkable support. Moreover we would like to appreciate your valuable suggestion to make our efforts more effective and fruitful.

In this edition as we kept all our previous segments along with two new sections we introduced in our previous edition and also tried to give them a new taste by discussing them more elaborately and keeping them more close to problem based pattern according to the need of the new age.

In review article, we discussed common skin diseases like Scabis, Tinea Pedis (Athelete's foot) and Seborrhoeic Dermatitis and their treatment plan.

We have selected Diabetic ketoacidosis for clinician's corner and often faced life threatening condition Myocardial Infarction (MI) in clinical focus. Vesicocervical fistula is the talk of the day these days for every clinician and people also getting more aware of this day by day. Hence, we highlighted on this and discussed elaborately about it in case review.

We also have other usual topics like clinical review where we focused on The management of ingrowing toenails and Transvaginal pudendal nerve blocking in clinical method.

And last but not the least; we thank you again for your continuous support and for valuable opinions to make our service better every day.

Thank you for being with us all the way through our journey.

Sincerely yours,

(Dr. S. M. Saidur Rahman)
Medical Services Manager

(Dr. Rumana Dowla)
Manager, Medical Information & Research

Common skin diseases

Scabies

Scabies (from scabere, "to scratch"), known colloquially as the seven year itch, is a contagious skin infection that occurs among humans and other animals. It has been classified by the WHO as a water related disease. It is caused by a tiny and usually not directly visible parasite, the mite Sarcoptes scabiei, which burrows under the host's skin, causing intense allergic itching. The mites that infest humans are female. They tunnel into the skin and lay eggs. About 40-50 eggs are laid in the lifetime of a mite. The eggs hatch into larvae after 3-4 days; these then grow into adults within 10-15 days. Less than one in 10 eggs becomes an adult scabies mite. Most of the symptoms of scabies infestation are due to the immune system response to the mites themselves, their saliva, their eggs or their faeces. The average number of mites on an infected person is around 12. Neglected children with scabies in underprivileged communities may have 100s of mites. Scabies infestation occurs worldwide and is very common.

Scraped skin under microscope showing mite

Mode of transmission

Direct skin to skin contact is the mode of transmission. Scabies mites are very sensitive to their environment. They can only live off of a host body for 24-36 hours under most conditions. Transmission of the mites involves close person to person contact of the skin to skin variety. It is hard, if not impossible, to catch scabies by shaking hands, hanging coat next to someone who has it, or even sharing bedclothes that had mites in them the night before. Sexual physical contact, however, can transmit the disease. In fact, sexual contact is the most common form of transmission among sexually active young people, and scabies has been considered by many to be a sexually transmitted disease (STD). However, other forms of physical contact, such as mothers hugging their children, are sufficient to spread the mites. Over time, close friends and relatives can contract it this way too. School settings typically do not provide the level of prolonged personal contact necessary for transmission of the mites. Scabies is spread by skin to skin contact with another person who has scabies. Pets and animals cannot spread human scabies. It is also not very likely for scabies to be spread by:

- A swimming pool
- Contact with the towels, bedding, and clothing of someone who has scabies, unless the person has what is called "crusted scabies"

Causes and incidence

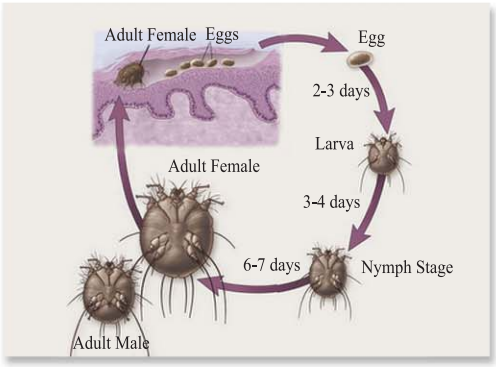
Scabies is found worldwide among people of all groups and ages. It is spread by direct contact with infected people, and less often by sharing clothing or bedding. Sometimes whole families are affected. Outbreaks of scabies are more common in nursing homes, nursing facilities, and child care centers. The mites that cause scabies burrow into the skin and deposit their eggs, forming a burrow that looks like a pencil mark. Eggs mature in 21 days. Mites may be more widespread on a baby's skin, causing pimples over the trunk, or small blisters over the palms and soles.

- In young children, the head, neck, shoulders, palms, and soles are involved.
- In older children and adults, the hands, wrists, genitals, and abdomen are involved.

The itchy rash is an allergic response to the mite.

Symptoms

- Mite tunnels may be seen on the skin as fine, dark, or silvery lines about 2-10 mm long. They most commonly occur in the loose skin between the fingers, the inner surface of the wrists, and the hands. However, they can occur on any part of the skin. Burrows may not be noticed until a rash or itch develops.
- Itching is often severe and tends to be in one place at first (often the hands), and then spreads to other areas. The itch is generally worse at night and after a hot bath. The infected person can have widespread itching, even with only a few mites.
- Rashes are especially seen in between the fingers. It usually appears soon after the itch starts. It is typically a blotchy, lumpy red rash that can appear anywhere on the body. The rash is



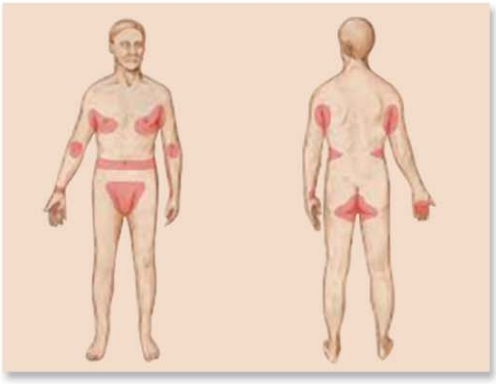
Life cycle of scabies

often most obvious on the inside of the thighs, parts of the abdomen and buttocks, armpits, and around the nipples in women. The appearance of the rash is often typical. However, some people develop unusual rashes which may be confused with other skin conditions.

- Scratching due to intense itching can cause minor skin damage. In some cases the damaged skin becomes infected by bacteria causing secondary skin infection. If skin becomes infected with bacteria it becomes red, inflamed, hot, and tender.
- Aggravation of preexisting skin conditions: Scabies can worsen the symptoms of other skin conditions, particularly itchy skin problems such as eczema, or problems such as psoriasis. Scabies can be more difficult to diagnose in these situations too.

Signs

- **Itching, mainly at night:** Itching is the most common sign. The itch can be so intense that it keeps a person awake at night.



Common areas affected by scabies in human being

- **Rash:** Many people get the scabies rash. This rash causes little bumps that often form a line. The bumps can look like hives, tiny bites, knots under the skin, or pimples. Some people develop scaly patches that look like eczema.
- **Sores:** Scratching the itchy rash can cause sores. An infection can develop in the sores.
- **Thick crusts on the skin:** Crusts form when a person develops a severe type of scabies called crusted scabies. Another name for crusted scabies is Norwegian scabies. These crusts house 100s to 1,000s of mites and the mites' eggs. With so many mites burrowing in the skin, the rash and itch become severe.

Diagnostic tests

According to the American Academy of Dermatology, the most common test involves applying a drop of sterile mineral oil to the suspected lesion. The site is then scraped with a scalpel and the scrapings are transferred to a slide. Under the microscope, it may be possible to find scabies mites, their eggs, or feces. Another option is an ink test, in which a blue or black felt tipped pen to the suspected areas. Then the skin surface is cleaned. Mite burrows can be revealed if the ink sinks into them.

Treatment

Cream: Mite killer like permethrin can be used. Creams are commonly used to treat scabies infections. The most commonly used cream is permethrin 5%. These creams are applied from the neck down, left on overnight, and then washed off. This application is usually repeated in seven days. Other creams include benzyl benzoate, sulfur in petrolatum, and crotamiton.

Oral medication: Ivermectin, is an oral antiparasitic medication that has also been shown to be an effective scabicide. It is taken at a dosage of 200 micrograms per kilogram body weight as a single dose, followed by a repeat dose two weeks later. Antihistamines, such as diphenhydramine, can be useful in helping provide relief from itching. Wash linens and bedclothes

Additional tips

- Treat sexual contacts or relevant family members.
- Cut nails, and clean under them thoroughly to remove any mites or eggs that may be present.
- Thoroughly vacuum rugs, furniture, bedding, and car interior and throw the vacuum cleaner bag away when finished.
- Try to avoid scratching. Keep any open sores clean.

Prognosis

Most cases of scabies can be cured without any long term problems. A severe case with a lot of scaling or crusting may be a sign that the person has a disease such as HIV.

Complications

The intense itching of scabies leads to prolonged and often intense scratching of the skin. When the skin is

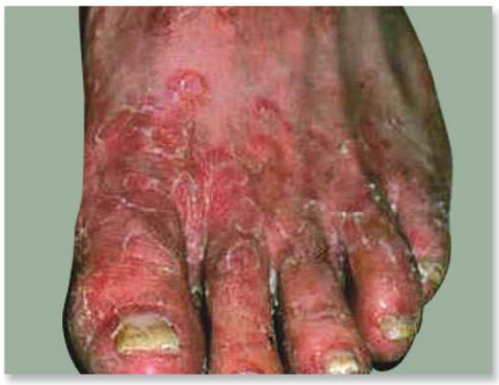
broken or injured due to scratching, secondary bacterial infections of the skin can develop from bacteria normally present on the skin, such as *Staphylococcus aureus* or β-hemolytic streptococci.

Prevention

Avoid contact with infected persons.

Tinea Pedis (Athlete's foot)

Tinea pedis is the medical term for Athlete's foot. Tinea = infections of the skin, nails, or hair caused by fungi; and pedis = foot. Tinea pedis is most commonly caused by *Trichophyton rubrum*, a dermatophyte initially endemic only to a small region of Southeast Asia and in parts of Africa and Australia. Interestingly, tinea pedis was not noted in these areas then, possibly because these populations did not wear occlusive footwear. The colonization of the *T. rubrum* endemic regions by European nations helped to spread the fungus throughout Europe. Wars with accompanying mass movements of troops and refugees, the general increase in available means of travel, and the rise in the use of occlusive footwear have all combined to make *T. rubrum* the world's most prevalent dermatophyte. It is also called Athelete's Foot.



Tinea Pedis

Causes and incidence

Athlete's foot occurs when a certain fungus grows on skin of feet. In addition to the toes, it may also occur on the heels, palms, and between the fingers.

The fungi that most often cause Athlete's Foot infections are:

- *Trichophyton mentagrophytes* often causes blister like infections. The infection appears suddenly, is often severe, and is usually easily treated.
- *Trichophyton rubrum* often causes the more chronic type of Athlete's Foot known as moccasin pattern infections. This condition may last for a long time and is often difficult to treat.

Athlete's foot is the most common type of tinea fungal infections. The fungus thrives in warm, moist areas. Risk for getting athlete's foot increases if :

- Wear closed shoes, especially if they are plastic lined
- Keep the feet wet for prolonged periods of time
- Sweat a lot
- Develop a minor skin or nail injury

Athlete's foot is contagious, and can be passed through direct contact, or contact with items such as shoes, stockings, and shower or pool surfaces.

Predisposing factors:

- The organisms that cause Athlete's foot thrive in damp, close environments created by wearing tight shoes that can squeeze the toes together and create warm, moist areas between them.
- Wearing damp socks and shoes. Warm, humid conditions that promote heavy sweating favor its spread.
- The fungus is carried on fragments of skin or other particles that contaminate floors, mats, rugs, bed linens, clothes, shoes, and moist surfaces. Just by walking barefoot on a contaminated surface is enough to cause Athlete's foot.
- Person to person contact is another means of transmission. Although transmission can occur within a household, the infection is more commonly passed along in public areas locker rooms, saunas, swimming pools, communal baths and showers.
- Cuts, cracks, and sores on the feet allow for easy penetration of fungi into the skin.

Symptoms

The most common symptom is cracked, flaking, peeling skin between the toes or side of the foot. Other symptoms can include:

- Red and itchy skin
- Burning or stinging pain
- Blisters that ooze or get crusty
- If the fungus spreads to nails, they can become discolored, thick, and even crumble.
- Athlete's foot may occur at the same time as other fungal skin infections such as ringworm or jock itch.

Diagnostic tests

Athlete's foot can simply be diagnosed by looking at the skin. If tests are needed, they may include:

- Skin culture
- Skin lesion biopsy
- Skin lesion KOH exam

Treatment

Antifungal powders or creams can help control the infection. These generally contain miconazole, clotrimazole, or tolnaftate. Keep using the medicine for 1 to 2 weeks after the infection has cleared to prevent the infection from returning. In addition:

- Keep the feet clean and dry, especially between the toes.
- Wash feet thoroughly with soap and water and dry the area very carefully and completely. Better to practice this at least twice a day.
- Wear clean, cotton socks and change socks and shoes as often as necessary to keep the feet dry.

Athlete's foot almost always responds well to self-care, although it may come back. Antibiotics may be necessary to treat bacterial infections that occur from scratching.

Prognosis

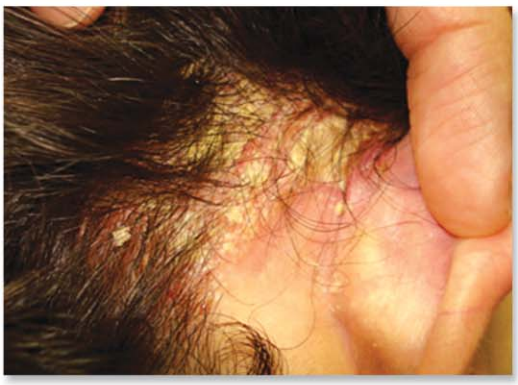
Athlete's foot infections range from mild to severe and may last a short or long time. They may persist or recur, but they generally respond well to treatment. Long term medication and preventive measures may be needed.

Complications

- Chance of relapse
- Bacterial skin infections such as cellulitis
- Lymphangitis, lymphadenitis

Seborrhoeic Dermatitis

Seborrhoeic dermatitis (also known as "seborrheic eczema") is an inflammatory skin disorder affecting the scalp, face, and torso. Typically, seborrheic dermatitis presents with scaly, flaky, itchy, and red skin. It particularly affects the sebaceous gland rich areas of skin. In adolescents and adults. Seborrheic dermatitis is a common, inflammatory skin condition that causes flaky, white to yellowish scales to form on oily areas such as the scalp or inside the ear or as mild to marked erythema of the nasolabial fold. It can occur with or without reddened skin.



Seborrheic Dermatitis of the scalp

Etiology

Seborrheic dermatitis frequently affects persons in post puberty. Additional evidence of hormonal influence is provided by research demonstrating that the human sebocyte responds to androgen stimulation. *Pityrosporum ovale*, a lipophilic yeast of the Malassezia genus, has been implicated in the development of this condition. It has been suggested that seborrheic dermatitis is an inflammatory response to this organism, but this remains to be proved. *P. ovale* is present on all persons. The colonization rate of involved skin by this organism may be lower than that of uninvolved skin.

Genetic and environmental factors, as well as other comorbid diseases, may predispose specific populations to the development of seborrheic dermatitis. Although seborrheic dermatitis affects only 3% of the general population, the incidence in persons with acquired immunodeficiency syndrome may be as high as 85%. The exact mechanism whereby human immunodeficiency virus infection promotes an atypical and explosive onset of seborrheic dermatitis is unknown, but many factors have been explored, including CD_4 positive T lymphocyte counts, *P. ovale* density and nutritional factors.

Persons with central nervous system disorders (Parkinson's disease, cranial nerve palsies, major truncal paralyses) also appear to be prone to the development of seborrheic dermatitis, tend to develop more extensive disease and are frequently refractory to treatment. It has been postulated that seborrheic dermatitis in these patients is a result of increased pooling of sebum caused by immobility. This increased sebum pool permits growth of *P. ovale*, which induces seborrheic dermatitis.

Causes and incidence

Seborrheic dermatitis is thought to be due to a combination of an over production of skin oil and irritation from a yeast called malessizia. Seborrheic dermatitis appears to run in families. Stress, fatigue, weather extremes, oily skin, infrequent shampoos or skin cleaning, use of lotions that contain alcohol, skin disorders (such as acne), or obesity may increase the risk. Neurologic conditions, including Parkinson's disease, head injury, and stroke may be associated with seborrheic dermatitis. Human Immunodeficiency Virus (HIV) has also been linked to increased cases of seborrheic dermatitis.

Symptoms

Seborrheic dermatitis can occur on many different body areas. Usually it forms where the skin is oily or greasy. Commonly affected areas include the scalp, eyebrows, eyelids, creases of the nose, lips, behind the ears, in the outer ear, and middle of the chest.

Seborrheic dermatitis in infants, also called cradle cap, is a harmless, temporary condition. It appears as thick, crusty, yellow or brown scales over the child's scalp. Similar scales may also be found on the eyelids, ear, around the nose, and in the groin. Cradle cap may be seen in newborns and small children up to age 3. Cradle cap is not contagious, nor is it caused by poor hygiene. It is not an allergy, and it is not dangerous. Cradle cap may or may not itch. If it itches, excessive scratching of the area may cause additional inflammation, and breaks in skin may cause mild infections or bleeding.

In general, symptoms of seborrheic dermatitis include:

- Skin lesions
- Plaques over large area
- Greasy, oily areas of skin
- Skin scales look white and flaking, or yellowish, oily, and adherent dandruff
- Itching may become more itchy if infected
- Mild redness
- Hair loss

Diagnosis

The diagnosis is based on the appearance and location of the skin lesions.

Treatment

In general, One can treat flaking and dryness with over the counter dandruff or medicated shampoos. Shampoo the hair vigorously and frequently (preferably daily). Loosen scales with the fingers, scrub for at least 5 minutes, and rinse thoroughly. Active ingredients in these shampoos include salicylic acid, coal tar, zinc, resorcin, ketoconazole, or selenium. Shampoos or lotions containing selenium, ketoconazole, or corticosteroids may be prescribed for severe cases. To apply shampoos, part the hair into small sections, apply to a small area at a time, and massage into the skin. If on face or chest, apply medicated lotion twice per day. Recently, creams classified as topical immune modulators are being used. Seborrheic dermatitis may improve in the summer, especially after outdoor activities.

Involved areas of the face may be washed frequently with shampoos that are effective against seborrhea as detailed above. Alternatively, ketoconazole cream, 2%, may be applied once or twice daily to affected areas. Often, 1% hydrocortisone cream will be added once or twice daily to affected areas and will aid with resolution of erythema and itching. Sodium sulfacetamide, 10% lotion, is also an effective topical agent for seborrheic dermatitis. Seborrhea of the trunk may be treated with frequent application of zinc or coal tar containing shampoos or by washing with zinc soaps. Additionally, topical ketoconazole cream, 2%, and/or a topical corticosteroid cream, lotion or solution applied once or twice daily will prove useful. Benzoyl peroxide washes are also helpful in controlling seborrhea of the trunk. Patients should be cautioned to rinse thoroughly after application of these agents as they will bleach clothing and bed linens. These agents may be drying, and the patient may benefit from application of a moisturizer after treatment.

An occasional patient with severe seborrhea that is unresponsive to the usual topical therapy may be acandidate for isotretinoin therapy. Isotretinoin can induce up to a 90% reduction in sebaceous gland size, with a corresponding reduction in the production of sebum. Isotretinoin also has anti-inflammatory properties. Treatment with daily doses of isotretinoin as low as 0.1 to 0.3 mg per kg may result in improvement in severe seborrhea after four weeks of therapy. Thereafter, a dose as low as 5 to 10 mg per day may be effective as maintenance therapy over several years.

Prognosis

Seborrheic dermatitis is a chronic condition that can be controlled with treatment. It often has extended inactive periods followed by flare ups. A more extreme form of this condition overlaps with psoriasis of the scalp and is called sebopsoriasis.

For infants with cradle cap

- Massage the baby's scalp gently with fingers or a soft brush to loosen the scales and improve scalp circulation.
- Give the child daily, gentle shampoos with a mild soap while scales are present. After scales have disappeared, shampooing can be reduced to twice weekly.
- Be sure to rinse off all soap.
- Brush that the child's hair with a clean, soft brush after each shampoo and several times during the day.
- If scales do not easily loosen and wash off, apply some mineral oil to the baby's scalp and wrap warm, wet cloths around his head for up to an hour before shampooing. Then, shampoo as directed above.
- If the scales continue to be a problem or concern, or if the child seems uncomfortable or scratches his scalp, medical attention may be needed. A cream or lotion can be applied on baby's scalp several times a day.

Complications

- Psychological distress, low self esteem, embarrassment
- Secondary bacterial or fungal infections

Prevention

The severity of seborrheic dermatitis can be lessened by controlling the risk factors and by paying careful attention to skin care.

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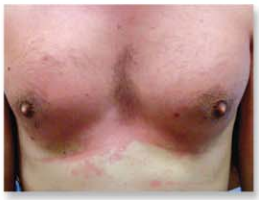
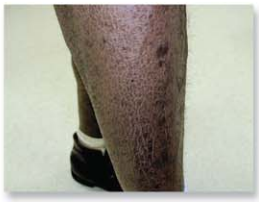
DERM DILEMMA



CASE 1

A 32 year old man presents with a 45 minute history of blanchable macular erythema involving his chest, back, upper extremities, neck, and face. He reports palpitations, acute onset of diarrhea, diaphoresis, headache, and a sense of anxiety. His vital signs are stable and he demonstrates no respiratory distress. The patient is otherwise healthy, and he states that the aforementioned signs and symptoms started 30 minutes after he ate an ahi salad.

What is your diagnosis?



CASE 2

A 53 year old man has had dry, itchy skin since early childhood. His medical history is positive for hypertension. He cannot recall whether any other family member was similarly affected. His current therapy is petrolatum ointment, which provides temporary relief of the pruritus but has no demonstrable effect on appearance. Examination reveals a striking fish scale appearance of the skin of his trunk and extremities, with his legs most severely affected.

What is your diagnosis?

Reference: Eme. Med. Nov. 2011, Vol. 43, No. 11: 17-18

Answers 17

People living alone are more depressed

People of working age who live alone increase their risk of depression by up to 80% compared with people living in families, says a Finnish study. The main factors are poor housing conditions for women and a lack of social support for men, who are both equally affected. Researchers said that living with other people could offer emotional support and feelings of social integration, as well as other factors that protect against mental health problems.

Living alone could be linked with feelings of isolation and a lack of social integration and trust, which are risk factors for mental health. Loneliness and isolation results in people having fewer outlets to talk about how they are feeling, which is something that we know can really help to manage and recover from a mental health problem. It is therefore essential that people who live alone are given the most appropriate treatment such as talking therapies, which provide safe, supportive environments to discuss and work through problems, rather than simply being left to rely solely on antidepressants.

Gene flaw linked to serious flu risk

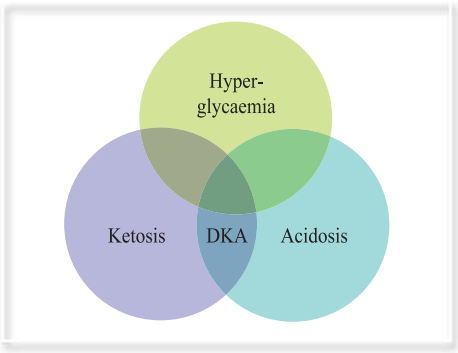
Scientists have identified a genetic flaw that may explain why some people get more ill with flu than others. The researchers said the variant of the IFITM3 gene was much more common in people hospitalized for flu than in the general population. It controls a malformed protein, which makes cells more susceptible to viral infection. Experts said those with the flaw could be given the flu jab, like other at risk groups. Evidence from genetic databases covering thousands of people showed the flawed version of the gene is present in around one in 400 people. This research is important for people who have this variant as we predict their immune defenses could be weakened to some virus infections. Scientists found during the recent swine flu pandemic, many people found it remarkable that the same virus could provoke only mild symptoms in most people, while, more rarely, threatening the lives of others. This discovery points to a piece of the explanation: genetic variations affect the way in which different people respond to infection. This important research adds to a growing scientific understanding that genetic factors affect the course of disease in more than one way. Genetic variations in a virus can increase its virulence, but genetic variations in that virus's host matter greatly as well.

Higher birth weight linked to grandmother gene

Scientists say a gene variation could contribute up to 155g (5.5oz) to a child's birth weight. They found a particular variant passed down from the mother can add 93g (3.3oz) to the birth weight, or 155g if passed down from the maternal grandmother. Scientists looked at a gene called PHLDA2 in nearly 9,500 DNA samples taken from mothers and their babies, collected in three separate studies. They found a gene variant called RS1 appeared to change the way in which the gene functioned, leading to higher birth weights. They found a genetic variant of PHLDA2 that when inherited from the mother, causes the baby to be 93g bigger on average, or even 155g bigger on average, if inherited successively from the mother's mother. The RS1 variation was found in around 13% of the individuals studied, with 87% possessing the RS2 variation. The scientists suggest that the more common RS2 gene variation, which is only found in humans, has evolved to produce a smaller baby as a protective effect to enhance the mother's survival during childbirth. The PHLDA2 gene is unusual in that only the copy inherited from the mother is active, while the copy inherited from the father is "silenced". This silencing of the paternal gene results from molecular processes around the DNA known as epigenetics. Scientists do not know why, but have speculated that it is to ensure birth weight is reduced to ensure the mother survives childbirth. Indeed the long term health consequences associated with extremes of birth weight might be due in part to this and other contributory genetic factors.

Reference: bbc.co.uk

Diabetic ketoacidosis

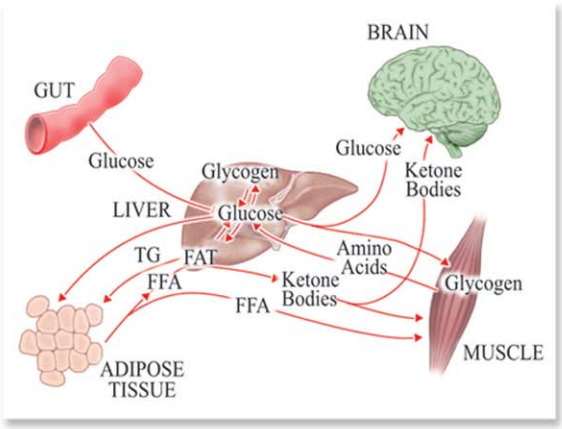


DKA - intermediary metabolism

Diabetic ketoacidosis (DKA) is an acute, major, life threatening complication of diabetes. DKA mainly occurs in patients with type 1 diabetes, but it is not uncommon in some patients with type 2 diabetes. DKA is a state of absolute or relative insulin deficiency aggravated by ensuing hyperglycemia, dehydration, and acidosis-producing derangements in intermediary metabolism. The most common causes are underlying infection, disruption of insulin treatment, and new onset of diabetes. DKA is defined clinically as an acute state of severe uncontrolled diabetes associated with ketoacidosis that requires emergency treatment with insulin and intravenous fluids. Biochemically, DKA is defined as an increase in the serum concentration of ketones greater than 5 mEq/L, a blood glucose level greater than 250 mg/dL (although it is usually much higher), and a blood (usually arterial) pH less than 7.3. Ketonemia and ketonuria are characteristic, as is a serum bicarbonate level of 18 mEq/L or less (< 5 mEq/L is indicative of severe DKA).

Pathogenesis

Diabetic ketoacidosis arises because of a lack of insulin in the body. The lack of insulin and corresponding elevation of glucagon leads to increased release of glucose by the liver from glycogen and through gluconeogenesis (a process that is normally suppressed by insulin). High glucose levels spill over into the urine, taking water and solutes (such as sodium and potassium) along with it in a process known as osmotic diuresis. Ketones also participate in osmotic diuresis and lead to further electrolyte losses. This leads to polyuria, dehydration, and compensatory thirst and polydipsia. The absence of insulin also leads to the release of free fatty acids from adipose tissue, which are converted, again in the liver, into ketone bodies (acetoacetate and β-hydroxybutyrate). β-hydroxybutyrate can serve as an energy source in absence of insulin mediated glucose delivery, and is a protective mechanism in case of starvation. However the ketone bodies have a low pKa and therefore turn the blood acidic (metabolic acidosis).



Pathogenesis of DKA

Incidence and causes

Diabetic ketoacidosis (DKA) is common in type 1 diabetes as this form of diabetes is associated with an absolute lack of insulin production by the islets of Langerhans. In type 2 diabetes, insulin production is present but is insufficient to meet the body's requirements as a result of end organ insulin resistance. Usually, these amounts of insulin are sufficient to suppress ketogenesis. If DKA occurs in type 2 diabetics, their condition is called "ketosis-prone type 2 diabetes".

People with type 1 diabetes do not have enough insulin, a hormone the body uses to break down sugar in the blood for energy. When glucose is not available, fat is broken down instead. As fats are broken down, acids called ketones build up in the blood and urine. In high levels, ketones are poisonous. This condition is known as ketoacidosis. Diabetic ketoacidosis is often the first sign of type 1 diabetes in people who do not yet have other symptoms. It can also occur in someone who has already been diagnosed with type 1 diabetes. Infection, injury, a serious illness, or surgery can lead to diabetic ketoacidosis in people with type 1 diabetes. Missing doses of insulin can also lead to ketoacidosis in people with diabetes.

People with type 2 diabetes can develop ketoacidosis, but it is rare. It is usually triggered by a severe illness.

Risk factors

The risk of diabetic ketoacidosis is highest if:

- Type 1 diabetes
- Younger than age 19

However, diabetic ketoacidosis can occur in other situations whether one has type 1 diabetes, type 2 diabetes or gestational diabetes. In a few cases, diabetic ketoacidosis is the first sign that a person has diabetes.

Treatment of DKA: Insulin

Symptoms

Symptoms can include:

- Polyuria
- Weight loss
- Blurred vision
- Weakness
- Nausea and vomiting
- Stomach pain

Other symptoms that can occur include:

- Abdominal pain
- Breathing difficulty while lying down
- Decreased appetite
- Decreased consciousness
- Dulled senses that may worsen to a coma
- Fatigue
- Frequent urination or thirst that lasts for a day or more
- Headache
- Muscle stiffness or aches
- Shortness of breath

Signs

- Dehydration
- Hypotension (postural or supine)
- Cold extremities / peripheral cyanosis
- Tachycardia
- Air hunger (Kussmaul breathing)
- Smell of acetone
- Hypothermia
- Confusion, drowsiness, coma

Diagnosis

Ketone testing may be used in type 1 diabetes to screen for early ketoacidosis. The ketones test is done using a urine sample. Ketone testing is usually done:

- When the blood sugar is higher than 240 mg/dL
- During an illness such as pneumonia, heart attack, or stroke
- When nausea or vomiting occur
- During pregnancy

Other tests for ketoacidosis include:

- Amylase blood test
- Arterial blood gas
- Blood glucose test
- Blood pressure measurement
- Potassium blood test

This disease may also affect the results of the following tests:

- CO_2
- CSF study
- Magnesium blood test
- Phosphorus blood test
- Potassium urine test
- Sodium blood test
- Sodium urine test
- Urine pH

Management of diabetic ketoacidosis

Fluid replacement

- 9% Saline (NaCl) IV
  1 litre over 30 min
  1 litre over 1 hour
  1 litre over 2 hour
  1 litre over next 2-4 hours
- When blood glucose < 15mmol/l (270 mg/dl)
  Switch to 5% dextrose, 1 litre 8 hourly
  If still dehydrated, continue 0.9% Saline and add 5% dextrose 1 litre per 12 hrs
- Typical requirement is 6 litres in first 24 hrs but avoid fluid overload in elderly patients
- Subsequent fluid requirement should be based on clinical response including urine output

Insulin

- 50 units soluble insulin in 50ml 0.9% Saline IV via infusion pump
  6 units/hr initially
  3 units/hr when blood glucose < 15mmol/l (270mg/dl)
  2units/hr if blood glucose declines < 10mmol/l (180mg/dl)
- Check blood glucose hourly initially-if no reduction in first hour, rate of insulin infusion should be increased
- Aim for fall in blood glucose of 3-6 mmol/l (55-110 mg/dl) per hour

Potassium

- None in first liter of IV fluid unless < 3.0mmol/l
- If potassium < 3.5 mmol/l, give 40 mmol/l added potassium
  Give in 1 litre of fluid
  Avoid infusion rate of > 20 mmol/hr
- If plasma potassium is 3.5-5.0 mmol/l, give 20 mmol added potassium

If plasma potassium is > 5.0 mmol/l, or patient is anuric, give no added potassium

Treatment

DKA should be treated in hospital preferably in high dependency unit. The goal of treatment is to correct the high blood sugar level with insulin. Another goal is to replace fluids lost through urination and vomiting. Most of the time, one needs to go to the hospital, where the following will be done:

- Insulin replacement
- Fluid and electrolyte replacement
- The cause of the condition (such as infection) will be found and treated

Prognosis

Acidosis can lead to severe illness or death. Improved therapy for young people with diabetes has decreased the death rate from this condition. However, it remains a big risk in the elderly, and in people who fall into a coma when treatment has been delayed.

Complications

Diabetic ketoacidosis is treated with fluids, electrolytes such as sodium, potassium and chloride and insulin. Perhaps surprisingly, the most common complications of diabetic ketoacidosis are related to this lifesaving treatment:

- Cerebral oedema
  - Cerebral oedema causing symptoms is relatively uncommon in adults during diabetic ketoacidosis (DKA), although asymptomatic cerebral oedema may be common.
  - Cerebral oedema usually occurs within a few hours of the initiation of treatment. It presents in the first 24 hours with headache, behavioral changes and urinary incontinence, progressing to abrupt neurological deterioration and coma.
  - Cerebral oedema associated with DKA is more common in children than in adults.
- Pulmonary oedema
  - Pulmonary oedema has only been rarely reported in DKA. Pulmonary oedema usually occurs within a few hours of the initiation of treatment.
  - Elderly patients and those with impaired cardiac function are at particular risk, and monitoring of central venous pressure should be considered.
- Iatrogenic hypoglycaemia: severe hypoglycaemia is also associated with cardiac arrhythmias, acute brain injury and death.
- Iatrogenic hypokalaemia.
- Cardiac dysrhythmia due to electrolyte disturbance (particularly K^+) or metabolic acidosis.
- Myocardial suppression due to metabolic acidosis.
- Venous thromboembolism.
- Myocardial infarction.
- Diabetic retinopathic changes may be seen prior to or after therapy for DKA.
- Hypophosphataemia - rarely has significant clinical effects. Although there is a large loss of total body phosphate in DKA, there is no evidence of benefit of phosphate replacement but phosphate measurement and replacement should be considered in the presence of respiratory and skeletal muscle weakness.
- Adult respiratory distress syndrome.

If untreated, the risks are much greater & diabetic ketoacidosis can lead to loss of consciousness. Eventually, Diabetic ketoacidosis can be fatal.

Prevention

People with diabetes should learn to recognize the early warning signs and symptoms of ketoacidosis. In people with infections or who are on insulin pump therapy, measuring urine ketones can give more information than glucose measurements alone. Insulin pump users need to check often to see that insulin is still flowing through the tubing, and that there are no blockages, kinks, or disconnections.

Actions a person with diabetes can take to prevent diabetic ketoacidosis include:

- Close monitoring and control of blood sugars, especially during times of infection, stress, trauma, or other serious illness.
- Taking extra insulin injections or other diabetes medications on time as directed by the health care practitioner.

References:
1. www.Patient.co.UK
2. www.Pub Med
3. Davidson's Principle & Practice of Medicine, 21th edition
4. Williams Textbook of Endocrinology, 11th edition

Vesicocervical fistula: An uncommon presentation

Introduction

Vesicocervial fistula is a disease that is rare in developed world but still prevailing in developing countries. It is mostly due to a result of obstetrical cause, sometimes a gynaecological operation may lead to the condition. This consequence of child delivery, secondary to a lesion in the bladder, was followed with a distressing, long lasting urinary incontinence non reactive to conservative treatment. The case details, clinical features, possible reasons and treatment modality are discussed.

Case report

Mrs. Jobaida is a 44 year old woman, para 4+0, 2 living; hailing from Narayanpur, Bhairav, presented in Ad din Hospital with urinary incontinence for 22 years, since her last twin birth. She is married for 30 years. Her first and second children were delivered at home by vaginal route. The second child died at the age of its 5 months due to diarrhoea. Her next pregnancy was twin and the both of the twins presented as breech which were delivered by vaginal route with the help of a traditional Dai. The first of the twins delivered at midnight as a still birth followed by the later twin who was delivered almost 17-18 hours later, at afternoon; also a stillbirth. She experienced with the symptoms of continuous urinary leakage and dribbling of urine from that period of time. Her last childbirth took place by normal vaginal route at home of Bhairav 20 years ago without any difficulty. As she did not know that the condition she is having any cure or treatment, she never seek for any medical attention then. After hearing the treatment facility of the problem is available at Ad din Hospital from a rural neighborhood, she got herself admitted on April 05, 2010. On examination, her general condition was good, and she had frank leakage of urine through the vagina. She is non asthmatic, nonhypertensive and nondiabetic.

The dye test revealed that the urine was exiting through the cervical canal and an old big tear was found on anterior wall of the cervix and vagina through which the dye came out. Intravenous urography performed later showing normal study. She was diagnosed as a case of vesicocervical fistula. She received antibiotic tablet Cefuroxime 500 mg for the treatment of urinary tract infection, tablet Vitamin-B complex and Vitamin-C, Tablet Iron and Anti-helminthic drug prior operation.

Necessary preoperative assessments were done, including ultrasonography of whole abdomen and Injection Tetanus Toxoid. She underwent an operation of Total Abdominal Hysterectomy with repair of Bladder on April 21, 2010. The postoperative period was uneventful. She was treated with Infusion Hartmann's Solution and 5% DNS, Injection Ciprofloxacin 500 mg, Injection Metronidazole 400 mg, Injection Pethedine, Injection Ranitidin and Injection Prochlorperazine for three days. From 4th POD, the antibiotic drugs were given orally with Tablet Nitrofurantoin 100 mg, Vitamin-C, Calcium, Tiemonium and Benzodiazepum, and Syrup Lactulose. The ureteric catheter was removed on 7th POD and the perurethral catheter was removed on 21st POD. The patient was discharged on 24th POD.

Operation procedure

With all asceptic precaution, the abdomen was opened. Total hysterectomy was done after retraction of the urinary bladder. The bladder was repaired in three layers using 3-0 vicryl sutures on the inner layer in a continuous fashion, beginning at the apex and extending through the full muscle layers. The bladder was imbricated with a second layer using interrupted 1-0 vicryl sutures. Transabdominal ureteral and urethral catheterizations were performed.

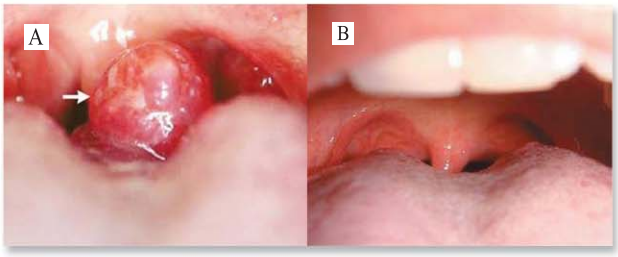
Conclusion

In this case, abdominal hysterectomy was done due to surrounding area of the tear is distorted and fibrosed and the patient was already amenorrheic due to repeated infection. In their work describing the complications of obstructed labor, Arrowsmith, Hamlin, and Wall drew attention to that "obstructed labor is one of the greatest unaddressed healthcare needs for the women of this planet. It should not be allowed to remain so. One must realize the historical context of the obstetric fistula and the societal difficulties surrounding the prevailing problem, and the surgical principles that govern fistula repair. Successful closure of vesicocervical fistula requires accurate diagnostic evaluation, appropriate repair using techniques that utilize basic surgical principles, and the careful application of interposing tissue flaps. With a better understanding of the issue along with experience, we can begin to combat the problem.

Reference: BMJ Vol: 40, No. 1, January 2011

Lingual thyroid

A 45 year old woman presented with long standing dyspnea, especially when lying down, and dysphagia in association with solid food. She had been diagnosed with hypothyroidism at 43 years of age, at which time the blood level of thyrotropin was 12.8 μIU per milliliter (normal range 0.25 to 4.0) and free thyroxine (T4) was 0.4 ng per deciliter (5.1 pmol per liter; normal range 0.6 to 1.8 ng per deciliter [7.7 to 23.2 pmol per liter]) and levothyroxine replacement therapy was initiated. At the current presentation, oropharyngeal examination revealed a solid, hyperemic, spherical mass at the base of the tongue

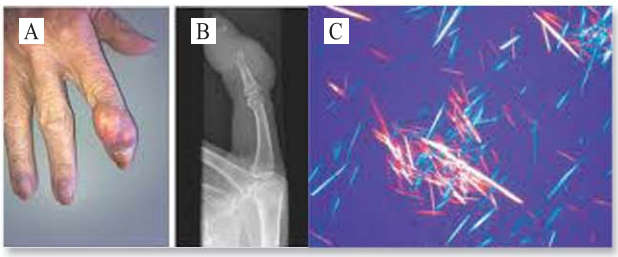


(Panel A). Laboratory studies, performed while the patient was receiving daily treatment with 100 μg of levothyroxine, revealed a thyrotropin level of 2.2 μIU per milliliter, a total (T4) level of 11.9 μg per deciliter (153.2 nmol per liter; normal range, 6.1 to 11.8 μg per deciliter [78.5 to 151.9 nmol per liter]), and a total triiodothyronine level of 111 ng per deciliter (1.7 nmol per liter; normal range 60 to 190 ng per deciliter [0.9 to 2.9 nmol per liter]). A thyroid scan performed after the administration of radioiodine (^{131}I) showed focal uptake only at the base of the tongue, a feature consistent with lingual thyroid (Panel A, arrow). The patient was treated with 7 mCi of ^{131}I and, 10 months later, with 10 mCi of ^{131}I. The ectopic thyroid mass receded over a period of 24 months after the initiation of treatment, in association with marked improvement in swallowing and resolution of airway compromise (Panel B).

Reference: N. Engl. J. Med. March 8, 2012, Vol. 366 (10): e15

Tophaceous gout

A 74 year old woman with chronic renal failure was admitted for diarrhea and functional impairment. She was noted to have a tender, soft swelling of the medial and distal phalanx of the right index finger (Panel A). She had no history of joint inflammation or any recent use of diuretics but reported consuming a bottle of wine daily. Plain radiography showed substantial osteolysis of the distal phalanx and partial osteolysis



of the medial phalanx (Panel B). Needle aspiration yielded a white viscous liquid, with numerous urate crystals identified on polarized light microscopy (Panel C). Abdominal computed tomography did not identify any uric acid stones. Slightly elevated levels of serum uric acid (386 μmol per liter) were attributed to chronic renal failure and untreated hypothyroidism. Bone destruction was attributed to tophaceous gout. Treatment with allopurinol and colchicine was initiated, and the patient was referred to an orthopedist who performed an arthrodesis of the remainder of the medial and distal phalanx. The patient was discharged without further complications.

Reference: N. Engl. J. Med. January 19, 2012, Vol. 366(3) : e6

Info Quiz Participants

- Have you selected the correct answer (s) You still have time to put your entry submission together for Info Quiz Prize
- The closing date for entries is 15 August 2012
- We look forward to receiving your winning entry

Info Quiz Answers
April-June 2012

1. d	2. c	3. a	4. c	5. a
6. c	7. d	8. d	9. c	10. d

The management of ingrowing toenails

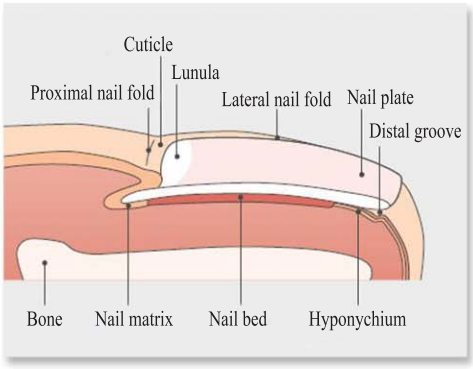


Fig 1: Anatomy of the nail and surrounding area. The nail plate inserts proximally into the proximal nail fold and consists of modified skin epithelium composed mainly of keratin. The cuticle is a thin membranous extension of the proximal nail fold. The nail bed matrix lies beneath the nail plate and is conventionally divided into the germinal matrix proximally and the sterile matrix distally; the germinal matrix is the regenerative part of the nail, whereas the sterile matrix adds thickness to the nail as the nail grows longitudinally along the nail bed. The hyponychium is the area under the free edge of the nail plate. The lunula is a white crescent shaped area seen in the posterior fifth of the nail plate, distal to the cuticle: it marks the distal part of the less vascular germinal centre

Ingrowing toenails are a common condition that causes pain and disability in the foot. The condition occurs when the nail plate traumatises the nail fold, giving rise to pain, inflammation, or infection (or a combination thereof). It commonly occurs in the great toe but can also affect the lesser toes. Patients with ingrowing toenails are usually male, between the ages of 15 and 40 years. The surgical treatments for ingrowing toenails include procedures on the nail plate, the nail bed (germinal matrix), and the surrounding soft tissues. Historically, a recurrence rate of 13-50% has been reported after surgical treatment, although more recent papers have reported recurrence rates of less than 5%, particularly with the use of wedge resection of the nail and phenol ablation of the nail matrix. A Cochrane review of nine randomised clinical trials of surgical treatments concluded that simple nail avulsion combined with phenol ablation was most effective in reducing symptomatic recurrence. It is important to recognise, however, that the presentation and disease process of ingrowing toenails covers a wide spectrum, and that management options will depend on the stage at which a patient presents. In this review the management of ingrowing toenails, focusing on the effectiveness of the procedures most commonly used.

How does an ingrowing toenail occur?

The term ingrowing toenail is used to describe a sharp spike of nail growing into an overlapping nail fold. This condition is caused by a combination of extrinsic and intrinsic factors, such as poorly fitting shoes, improperly trimmed nails, tight socks, excessive sweating, soft tissue abnormalities of the toe, and inherent nail deformity. Normal nails vary greatly in shape, and the nail walls are adaptable to marked curvature of the nails. Ingrowing toenails can occur in the context of normal nail shape or abnormal nail shape. In normal nails the nail plate is slightly convex from side to side; in people with normal nails, improper nail trimming can lead to a nail spike that traumatises soft tissue. This provides a port of entry for bacterial and fungal skin flora, resulting in tenderness, inflammation, and infection. Poorly fitting shoes can exacerbate the situation. Ingrowing toenails can also occur in people with abnormal nail shapes, such as incurvated nails or a wide nail plate. In this situation, the condition can occur congenitally or in adults, where increased pressure on the nail leads to increased transverse curvature that causes the edge of the nail to dig into the toe.

Clinical features

The clinical presentation of an ingrowing toenail has traditionally been divided into three stages

Stage I: Pain, swelling, and erythema

Stage II: Signs of inflammation together with active or acute infection

Stage III: Chronic infection leading to formation of hypergranulation tissue at the nail folds.

It can be difficult to determine the clinical stage, however, and it is simpler to consider the presentation as being on a spectrum of the disease process. The initial clinical presentation is of pain, swelling, erythema, and hyperhidrosis in the affected toe. After the initial inflammation and infection, a draining abscess causes further erythema, oedema, hyperhidrosis, and tenderness. Attempts at healing lead to the formation of hypertrophic granulation tissue, which is slowly covered by epithelium; this inhibits drainage and promotes oedema, leading to chronic infection and hypertrophy of the nail wall.

Treatment options for ingrowing toenails

Traditionally, management has been dictated by the clinical stage of presentation. With the recent evaluation of treatment methods such as partial nail avulsion and segmental phenol ablation, however, the management of this condition has changed, and it is simpler to classify ingrowing toenails into those

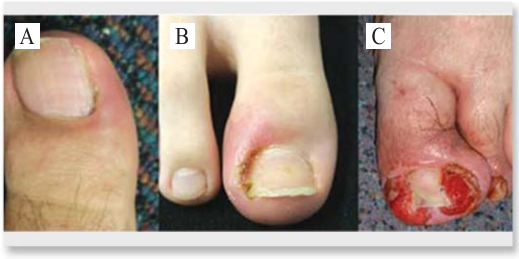


Fig 2: An ingrowing toenail at different stages of presentation. (A) Stage I with pain, swelling, and erythema; (B) stage II with signs of inflammation together with active or acute infection; (C) stage III with chronic infection leading to granulation tissue formation at the nail folds

that occur in normal nails and those occurring in abnormally wide or incurvated edge toenails.

Ingrowing toenails in normal nails tend to present in younger people and are usually a result of improper nail trimming of the lateral edge, which leaves a sharp nail spike that traumatises the nail fold. Evidence from observational studies indicates that the initial treatment should be conservative, with the patient given general instructions in foot care and footwear. The nail should be trimmed at right angles to the long axis of the toe and patients are able to carry this out at home. A chiropodist can gently retract the nail fold and trim the offending nail spike.

Ingrowing toenails most commonly develop in adults with abnormally wide toenails or those with an incurvated edge. Incurvated (or involuted) toenails can be caused by a bony malformation of the dorsum of the distal phalanx, or by secondary changes in the toenail as a result of irritation and pressure. There is no consensus on standard non-operative treatment of ingrowing toenails in abnormally shaped nails, but failure of conservative management should lead to consideration of surgical options. These patients are best offered partial nail avulsion with segmental phenol ablation. Phenol has potent antiseptic properties, so the procedure can be carried out even in the presence of infection without risk of wound infection. Patients with severe involuted nails on both the tibial and fibular sides (pincer nails) would be left with a too thin nail after wedge excision and may be better treated with a total nail avulsion.

Surgical options

The surgical options consist of procedures that are temporary or permanent.

Temporary procedures: A Cochrane review has shown that recurrence of symptoms is high after temporary measures, such as simple (or partial) nail avulsion without chemical or surgical ablation, and this may lead to low patient satisfaction. Therefore, we prefer to perform the procedure in selected patients only. However, removal of the nail spike is curative if followed by appropriate aftercare, as detailed above.

Permanent procedures: Historically, ablation of the germinal matrix centre (Zadik's procedure) or reductive procedures to the lateral nail fold (Winograd's procedure) were popular. In Zadik's (sometimes mistakenly called Zadek's) procedure, the nail forming part of the nail bed is removed and adequate skin cover is provided without shortening the distal phalanx. Winograd's technique of wedge excision involves partial removal of the nail plate and matrix, as well as removal of a wedge of the lateral nail fold. In essence, these procedures attempt to prevent nail recurrence by destroying the germinal matrix by surgical ablation, but are less commonly performed than procedures that combine partial nail avulsion with ablation of the nail matrix using electrocauterisation, laser surgery, or agents such as phenol or sodium hydroxide.

A Cochrane review of surgical treatments suggests that simple nail avulsion combined with phenol ablation should be the treatment of choice. A recent randomised clinical trial also showed lower rates of recurrence with partial nail avulsion and phenol ablation compared with partial avulsion with nail matricectomy. The success of phenol matricectomy depends on the use of good quality phenol and satisfactory haemostasis. Individually packed and sealed sterile containers of 90% liquid phenol with appropriately sized cotton tips are now available, and these are safer to use than phenol in brown bottles, which usually come from pharmaceutical suppliers. These individually packed containers also reduce the risk of spillage.

Partial nail avulsion with segmental phenol ablation
We use the following technique when performing this procedure (fig 3). The toe is cleaned with an appropriate skin preparation, such as povidone-iodine or chlorhexidine. A ring block with 1% plain lidocaine is injected at the base of the toe and a coloured ring tourniquet with a tag is applied-flesh coloured glove tourniquets are no longer used because of the risk of failing to remove them at the end of the procedure. Blunt dissection is carried out to separate the edge of the appropriate nail plate from the soft tissues.

A cut is made with a straight Beaver mini-blade to isolate an appropriate (usually 3-5 mm) section of the affected nail segment extending under the proximal nail fold, which is lifted off by grasping with an artery clip and using a central to lateral twisting motion to avulse the germinal centre.

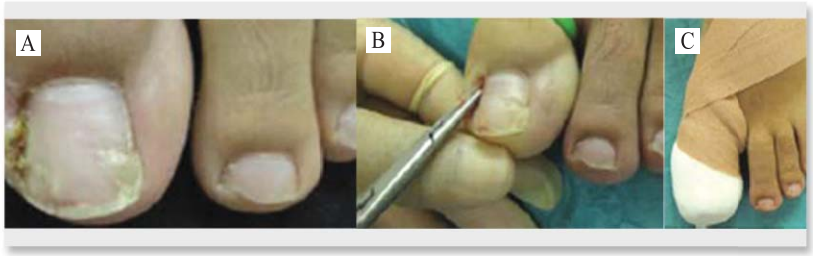


Fig 3: (A) An ingrowing toenail causing chronic infection; (B) partial nail avulsion with removal of a nail segment that extends under the eponychium; (C) postoperative dressing

Good haemostasis should be achieved before application of phenol because the presence of blood prevents a proper matricectomy. Denatured matrix looks white as opposed to the black colour of denatured blood. The surrounding skin is protected by application of paraffin jelly. A one minute application of phenol is usually performed twice, followed by a washout with normal saline. A washout with alcohol is commonly performed but is unnecessary. The chemical action of phenol is self limiting as a result of the process of cellular destruction, not the change in solvents after the application of alcohol. A postoperative dressing is applied and the patient is asked to remove the dressing in 48 hours and soak the foot in tepid salt baths daily. This is done to prevent debris from accumulating in the nail folds because this can lead to infection. Patients usually experience very little postoperative pain and can return to work the next day. Warn the patient that a serous discharge often occurs but usually settles within two weeks, although it can sometimes persist for several weeks. The rate of recurrence after phenol ablation is low and is usually treated by repeat application of phenol. An added advantage of this procedure is that it can be carried out even in the presence of acute infection. We advise against making an incision in the skin to remove the nail segment in the presence of acute infection.

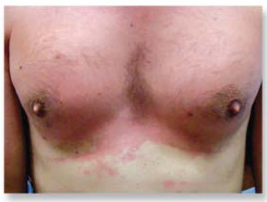
Conclusion

The treatment of ingrowing toenails has traditionally been blighted by high recurrence rates and poor patient satisfaction, but with the increasing use of chemical ablation of the nail matrix in combination with partial nail avulsion reported recurrence rates have decreased.

Reference: BMJ 7 April 2012 , Volume 344: 37-40

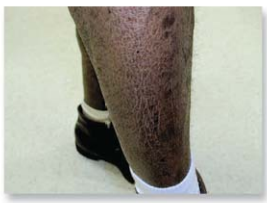
DERM DILEMMA

ANSWER



CASE 1

The diagnosis is scombroid poisoning, which is caused by ingestion of fish contaminated with high levels of histamine. It is commonly believed that fish from the Scombridae family are the most frequent cause; however, other fish such as mahi mahi and sardines have been implicated. Patients typically present with flushing, macular erythema, conjunctival injection, gastrointestinal upset, headache, and palpitations minutes to 1 hour after ingesting spoiled fish. They may also note a peppery taste. The condition is treated with antihistamines. Although the prognosis is very good, rare cardiovascular compromise is possible. Scombroid poisoning is not an allergic reaction, and corticosteroids should not be considered standard of care. The source of the contaminated fish should be identified to prevent or limit an outbreak.



CASE 2

The patient has ichthyosis, a condition that is characterized by xerosis and a fish scale appearance of the skin. Acquired ichthyosis usually occurs later in life and may be associated with systemic diseases such as hypothyroidism, cancer, and HIV infection. Ichthyosis vulgaris is transmitted as an autosomal dominant trait and manifests during the first year of life or shortly thereafter. Ichthyosis is presently without cure. Therapy is lifelong and consists of hydration and moisturization of the skin.

Reference: Eme. Med. Nov. 2011, Vol. 43, No. 11: 17-18

Transvaginal pudendal nerve blocking

Blocking the pudendal nerve with injection of local anesthetic is used for vaginal deliveries and for minor surgeries of the vagina and perineum. Use of this nerve block for vaginal delivery was reported as early as 1916. However, the procedure did not become popular until 1953-54, when Klink and Kohl implemented the modified technique.

The sensory and motor innervations of the perineum is derived from the pudendal nerve, which is composed of the anterior primary divisions of the second, third, and fourth sacral nerves. The pudendal nerve's 3 branches include the following:

- Dorsal nerve of clitoris, which innervates the clitoris
- Perineal branch, which innervates the muscles of the perineum, the skin of the labia majora and labia minora, and the vestibule
- Inferior hemorrhoidal nerve, which innervates the external anal sphincter and the perianal skin

A pudendal nerve block targets the pudendal nerve trunk as it enters the lesser sciatic foramen, about 1cm inferior and medial to the attachment of the sacrospinous ligament to the ischial spine. Here, the nerve is medial to the internal pudendal vessels. This nerve is accessed by 2 approaches, transvaginal and transcutaneous (or perineal). The former approach is more reliable and is used most often, except when an engaged head makes vaginal palpation more difficult. The anatomical basis for both approaches is to block the nerve proximal to its terminal branches.

Indications

While neuroaxial analgesia continues to replace pudendal nerve block as the technique of choice, there are and will always remain situations in which anesthesia service is unavailable. In this event, pudendal block provides a suitable alternative for the following:

- Analgesia for the second stage of labor
- Repair of an episiotomy or perineal laceration
- Outlet instrument delivery (to assist with pelvic floor relaxation)
- Used in the past as an alternative to neuroaxial analgesia in assisted twin and breech deliveries
- Minor surgeries of the lower vagina and perineum Contraindications
- Patient refusal
- Patient's inability to cooperate
- Patient sensitivity to local anesthetics
- Presence of infection in the ischiorectal space or the adjacent structures, including the vagina or perineum
- Coagulation disorders

Anesthesia

Lidocaine 1% is often used for pudendal nerve block. Agents that could be used instead include 2 chloroprocaine 2%, bupivacaine 0.25%, prilocaine 1%, or mepivacaine 1%. Because of its short duration of action, 2 chloroprocaine 2% is used less often. However, its rapid onset of action provides an advantage if the block is performed immediately before delivery. Another advantage of 2 chloroprocaine 2% comes from its rapid metabolism and short intravascular half-life, which decrease the risk of maternal and fetal toxicity. Although some obstetricians contend that the addition of epinephrine to the local anesthetic solution improves the quality of the block. The addition of epinephrine slightly prolonged the interval between the block administration and delivery. Maternal venous blood mepivacaine concentrations were slightly higher in the group without epinephrine, but no such differences were noted in umbilical cord blood samples.

Equipment

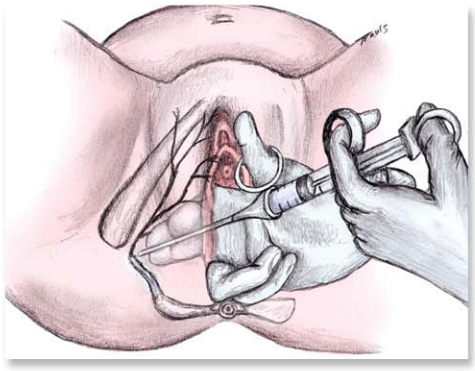
- Iowa trumpet or similar guide to facilitate the placement of the needle
- Needle, usually 6 in, 20-22 gauge
- Syringe with finger ring, 10 ml
- Local anesthetics (eg, lidocaine 1%)
- Sterile gloves
- Resuscitation equipment and medications in case an adverse reaction to the anesthetic is encountered

Positioning

The block is performed with the patient in the lithotomy position.

Technique

- Usually, no vaginal preparation is needed.



Technique of pundendal nurve blocking

- Palpate the ischial spine. This is usually done transvaginally but can also be done through the rectum.
- Be sure to use a needle with a guide (either the Iowa trumpet or the Kobak needle guide) to limit the depth of submucosal penetration and to prevent injury to the vagina and the fetus.
- To perform a left sided block, palpate the ischial spine with the index finger of the left hand, hold the syringe in the right hand, and guide the needle between the index and middle finger of the left hand toward the ischial spine.

The 3-injection technique

- Place the end of the guide beneath the tip of the ischial spine.
- Push the needle into the vaginal mucosa.
- Aspirate to ensure that the injection is not intravascular.
- Raise a mucosal wheal with 1 mL of local anesthetic.
- Advance the needle through the vaginal mucosa until it touches the sacrospinous ligament 1 cm medial and posterior to the ischial spine.
- Infiltrate the tissue with 3 mL of local anesthetic.
- Next, advance the needle further through the sacrospinous ligament for a distance of 1 cm until a loss of resistance is appreciated.
- The tip now lies in the area of the pudendal nerve. At this point, the pudendal vessels lie just lateral to the pudendal nerve, so care must be taken to avoid intravascular administration. Aspirate to confirm the needle placement is not intravascular prior to injecting lidocaine.
- Inject another 3 mL of local anesthetic solution into this region.
- Subsequently, withdraw the needle into the guide and move the tip of the guide to just above the ischial spine.
- At this new location, reinsert the needle though the mucosa and again inject 3 mL of local anesthetic.

- Many practitioners use a single 10 mL injection instead of the 3-injection technique. The single injection is done after the needle is introduced nearly 1 cm through the sacrospinous ligament medial and posterior to the ischial spine.
- To block the right side of the pelvis, repeat these steps using the right hand to hold the needle and needle guide. Pudendal block, transvaginal approach.
- This block could be attempted under ultrasonographic, CT, or fluoroscopic guidance. Although imaging helps delineate the anatomic landmark for needle placement, it is rarely used by obstetricians and gynecologists.

Additional anesthesia

Even in the best hands, pudendal anesthesia is less than 100% reliable. The patient must be checked bilaterally for loss of anal wink reflex before proceeding with the surgical procedure. If mild stimulus does not elicit a reflex response, a pinch confirms the effectiveness of bilateral anesthesia. A smaller repeat dose can be used if an adequate block is not seen, but care must be taken to avoid toxic serum levels. Appropriate monitoring of the patient and the fetus is mandatory, and intravenous access should be readily available. Provisions should be made for rapid resuscitation should toxicity or adverse reactions occur. Keep in mind that the pudendal block provides inadequate anesthesia for mid-forceps delivery, deliveries that require uterine manipulation, postpartum examination and repair of the upper vagina and cervix, and manual exploration of the uterine cavity. Under these circumstances, the addition of intravenous narcotics may provide appreciable, though not total, relief from pain. With such an approach, caution must be exercised to avoid dosages or combinations of narcotics and sedatives that might obtund the patient and cause possible airway obstruction or aspiration.

Timing of procedure

Timing of block placement is important, since at least 5-10 minutes are required for the infiltration to take effect. Most obstetricians perform pudendal

block immediately prior to delivery. This practice reflects the concern that perineal anesthesia may prolong the second stage of labor. Anesthesia may last 20-60 minutes, depending on the agent used with or without epinephrine. In patients without neuroaxial analgesia, the pudendal block can be performed when the patient reports vaginal or perineal pain. With early pudendal nerve block, the obstetrician may repeat the block, if necessary, so long as the maximum dose of local anesthetic is not exceeded.

Efficacy

Efficacy varies depending on the experience of the obstetrician. Unilateral or bilateral failure is common, with success rates of only 50% with the transvaginal route and approximately 25% with the transperineal route. Obstetricians typically perform simultaneous perineal infiltration, especially if the block was not placed until just prior to delivery. If delivery occurs before the pudendal block has become effective, an episiotomy can still be made without pain. By the time of the repair, the pudendal block usually has become effective.

Pearls

- Pudendal nerve block does not abolish sensation to the anterior part of the perineum, as the perineum is supplied by branches of the ilioinguinal and genitofemoral nerves.
- Failure to wait a sufficient time after injection is a common reason for block failure.
- Pudendal block does not abolish the pain of uterine contractions and cervical dilatation; this sensation is transmitted by the sympathetic fibers derived from the spinal levels of T10-L2.
- This block does not help relax the uterus.
- The obstetrician should be alert to the total dose of local anesthetic given, especially in cases of repetitive pudendal nerve blocks or pudendal nerve block in association with perineal infiltration. When lidocaine without epinephrine is used, the dose recommended is 4.5 mg/kg. When lidocaine with epinephrine is used, the recommended dose is 7 mg/kg. The maximum dose of lidocaine used should not exceed 300 mg. When bupivacaine without epinephrine is used, the recommended dose is 2.5 mg/kg, with the maximum dose not to exceed 175 mg. When used with epinephrine, the maximum dose of bupivacaine that could be used is up to 225 mg.
- Be sure to use a needle with a guide (either the Iowa trumpet or the Kobak needle guide) to limit the depth of submucosal penetration and to prevent injury to the vagina and the fetus.

Complications

Potential complications should be explained to the patient prior to getting informed consent. These complications are uncommon but may be serious. Direct intravascular injections or systemic absorption of an excessive dose of local anesthetic may result in systemic toxicities. Other risks include the following:

- Laceration of the vaginal mucosa is a potential complication.
- The second stage of labor may be prolonged due to a loss of the bearing-down reflex, particularly when local anesthetics are combined with epinephrine (does not affect incidence of instrument delivery).
- Systemic anesthetic complications, though rare and usually transient, may include palpitation, tinnitus, dysarthria, drowsiness, confusion, loss of consciousness, convulsions, hypotension, and bradycardia.
- Hematomas (vaginal, retroperitoneal, and ischiorectal) from injury to the pudendal artery can be a complication of pudendal block, particularly with defective coagulation.
- Infection (retro psoas and subgluteal abscess) has occasionally been reported, spreading superiorly along the psoas muscle or laterally along the obturator internus. Infrequent occurrence and diagnostic difficulties make these abscesses especially dangerous.
- Ischial region paresthesia on the first postpartum day, or sacral neuropathy, may occur.
- Needle stick injury with associated risk of exposure to HIV and other blood borne diseases may result, as the needle guide does not uniformly protect the physician and the procedure requires multiple blind needle punctures.

References: www.Pub Med

Myocardial Infarction (MI)

MI is almost always due to the formation of occlusive thrombus at the site of rupture or erosioz of an atheromatous plaque in a coronary artery. Most patients present when it is still possible to salvage myocardium and improve outcome.

Clinical features

- In some patients breathlessness is the only symptom, while in others (particularly older or diabetic patients) the infarct may go unrecognised (silent MI).
- Syncope may occur due to an arrhythmia or profound hypotension.
- Vomiting and sinus bradycardia resulting from vagal stimulation are particularly common with inferior MI.
- Sudden death, from VF or asystole, frequently occurs within the first hour.

Investigations

ECG: Shows a characteristic series of changes (Fig. 1):

- The earliest change is usually ST elevation followed by diminution in the size of the R wave, and development of a Q wave (indicating full-thickness infarction).
- Subsequently, the T wave becomes inverted and this change persists after the ST segment has returned to normal.
- ECG changes are best seen in the leads that 'face' the infarcted area.
- With anteroseptal infarction, abnormalities are found in one or more leads from V1 to V4.
- Anterolateral infarction produces changes from V4 to V6, in aVL and lead I.
- Inferior infarction is best shown in leads II, III and aVF.
- Infarction of the posterior wall of the left ventricle does not cause ST elevation or Q waves in the standard leads, but can be diagnosed by the presence of reciprocal changes (ST depression and a tall R wave in leads V1-V4).
- Occasionally, new onset LBBB is the only ECG change. Plasma biochemical markers (Fig. 2):

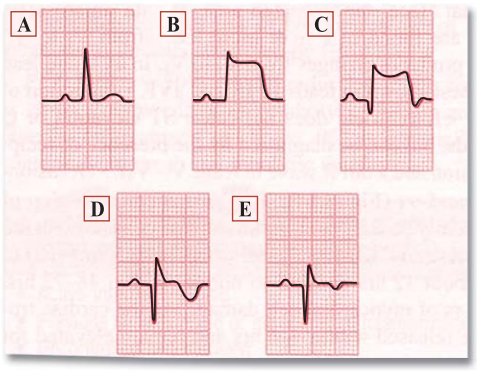


Fig. 1 **The serial evolution of ECG changes in full-thickness MI**. A. Normal ECG complex. B. (Minutes) Acute ST elevation. C. (Hours) Progressive loss of the R wave, developing Q wave, resolution of the ST elevation and terminal T-wave inversion. D. (Days) Deep Q wave and T-wave inversion. E. (Weeks or months) Old or established infarct pattern; the Q wave tends to persist but the T-wave changes become less marked

The plasma concentration of enzymes and proteins normally concentrated within cardiac cells is increased in MI. Creatine kinase (CK) and CK-MB (a cardiospecific isoform) start to rise at 4-6 hrs, peak at about 12 hrs and fall to normal within 48-72 hrs. The most sensitive markers of myocardial cell damage are the cardiac tro-ponins T and I, which are released within 4-6 hrs and remain elevated for up to 2 wks.

Echocardiography: Can be performed at the bedside. This is a very useful technique for assessing LV and RV function and for detecting important complications such as mural thrombus, cardiac rupture, ventricular septal defect, mitral regurgitation and pericardial effusion.

Early management

Immediate measures include:

- High-flow oxygen.
- Analgesia (IV opiates).
- IV antiemetic (metoclo-pramide).
- Oral aspirin (300 mg). Analgesia is essential to relieve distress, and also to lower adrenergic drive and susceptibility to arrhythmias.

Acute reperfusion therapy

Thrombolysis: Helps to restore coronary patency, preserves LV function and reduces the mortality of MI by 25-50%. Successful thrombolysis leads to reperfusion with relief of pain and resolution of acute ST elevation. It is indicated only in patients presenting within 12 hrs of the onset of symptoms and with ECG changes of LBBB or ST segment elevation of >1 mm in the limb leads or 2 mm in the chest leads. The benefit is greatest when treatment is given within the first few hours.

- Streptokinase (1.5 million U in 100ml normal saline IV over 1 hr) is widely used but may cause hypotension and, due to its antigenicity, serious allergic manifestations. Circulating neutralising antibodies are formed following treatment and this can render subsequent infusions of Streptokinase ineffective.
- Alteplase (human tissue plasminogen activator (tPA) 15 mg bolus then 0.75 mg/kg up to 50 mg over 30 mins, then 0.5 mg/kg up to 35 mg over 60 mins) is not antigenic, seldom causes hypotension and may produce better survival rates than Streptokinase,

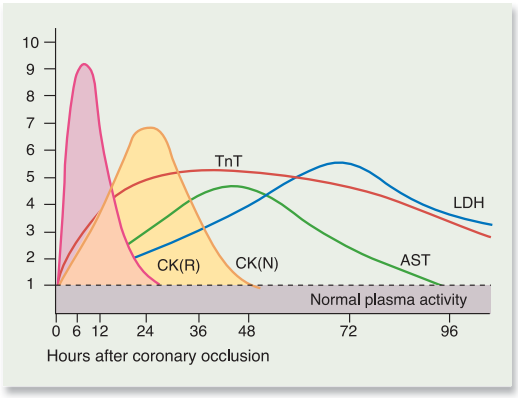


Fig. 2 **Changes in plasma enzyme concentrations after MI**. Creatine kinase (CK) and troponin T (TnT) are the first to rise, followed by aspartate aminotransferase (AST) and then lactate (hydroxybutyrate) dehydrogenase (LDH). In patients treated with a thrombolytic agent, reperfusion is usually accompanied by a rapid rise in plasma creatine kinase (curve CK(R)) due to a washout effect; if there is no reperfusion, the rise is less rapid but the area under the curve is often greater (curve CK(N))

particularly among high risk patients (e.g. large anterior infarct), but with a slightly higher risk of intracerebral bleeding.

- Newer generation analogues of tPA such as tenecteplase and reteplase have equivalent efficacy to alteplase but have a longer plasma half life and can be given by bolus administration. The major hazard of thrombolytic therapy is bleeding, particularly cerebral haemorrhage. Serious risk of bleeding is one of several relative contraindications to thrombolytic therapy.

PCI: Offers a 50% greater reduction in the risk of death, recurrent MI or stroke compared with thrombolytic therapy and, when available, is the reperfusion treatment of choice. PCI is especially useful in patients with contraindications to thrombolytic therapy.

Relative Contraindications To Thrombolytic Therapy (Potential Candidates For Primary Angioplasty)

- Active internal bleeding
- Previous subarachnoid or intracerebral haemorrhage
- Uncontrolled hypertension
- Recent surgery (within 1 month)
- Recent trauma (including traumatic resuscitation)
- High probability of active peptic ulcer
- Pregnancy

Maintaining vessel patency

Antlplatelet therapy: Oral administration of aspirin (300 mg initially then 75 mg daily) improves survival (30% reduction in mortality) on its own, and complements the effect of thrombolytic therapy. In combination with aspirin, the early (within 12 hrs) use of clopidogrel (75 mg daily) confers a further 10% reduction in mortality.

Unfractionated or low molecular weight heparin: May prevent reinfarc-tion after successful thrombolysis and reduce the risk of thromboembolic complications.

Adjunctive therapy

- I.V. β-blockers relieve pain, reduce arrhythmias and improve short-term mortality in patients who present within 12 hrs of the onset of symptoms, but should be avoided if there is heart failure, AV block or bradycardia.
- I.V. nitrates are useful for the treatment of LV failure and the relief of recurrent or persistent ischaemic pain.

Complications of infarction

Arrhythmias: Nearly all patients with acute MI have some form of arrhythmia; pain relief, rest and the correction of hypokalaemia are important preventative measures.

- VF occurs in about 5-10% of patients who reach hospital. Prompt defibrillation usually restores sinus rhythm. Early VF (within the first 48 hrs) does not adversely alter long-term prognosis, provided patients are promptly resuscitated.
- AV block complicating inferior infarction is usually temporary and often resolves following reperfusion.
- AV block complicating anterior infarction carries a risk of asystole and a prophylactic temporary pacemaker should be inserted.
- Other common peri-infarct arrhythmias include sinus tachycardia, sinus bradycardia, AF, VT and idioventricular rhythm.

Acute circulatory failure: Acute circulatory failure usually reflects extensive myocardial damage and indicates a bad prognosis. Hypotension, oliguria, confusion and cold, clammy peripheries are the manifestations of a low cardiac output, whereas breathlessness, hypoxia, cyanosis and inspiratory crackles at the lung bases are typical features of pulmonary oedema. If necessary, a Swan-Ganz catheter can be used to measure the pulmonary artery wedge pressure: an indirect measure of left atrial pressure that guides fluid replacement. Inotropic agents may be required to augment cardiac output and insertion of an intra-aortic balloon pump can be very beneficial. Since viable myocardium surrounding a fresh infarct may contract poorly for a few days and then recover (myocardial stunning), it is often worth treating cardiogenic shock energetically in the hope that cardiac function will improve.

Reference: Davidson's Essentials of Medicine

Jog your memory

Please select the correct answer by (√) against a, b, c, d & e of each questions in the Business Reply Card and send it through our colleagues or mail within 15 August 2012; this will ensure eligibility for the Raffle Draw and the lucky winners will get attractive prizes!

1. **Post-traumatic epilepsy:**
   a. It usually follows head trauma within a month
   b. The CT reveals the causative abnormalities
   c. It requires surgical therapy in most cases
   d. It responds poorly to standard anticonvulsive therapy
   e. The EEG reveals its characteristic change

2. **In case of congenital hypertrophic pyloric stenosis:**
   a. It is more frequent in females
   b. There is an increased likelihood for any offspring to be similarly afflicted
   c. Any vomit almost never contains bile
   d. The pyloric ring is rarely palpable
   e. The majority of patients require surgical therapy

3. **Digitalis therapy:**
   a. Is contraindicated in atrial tachycardia
   b. Elongates the effective refractory period of the AV node
   c. Is likely to cause intoxication with a concomitant hyperkalemia
   d. Is contraindicated in cor pulmonale
   e. Is effective in hypertrophic obstructive cardiomyopathy

4. **In Paget's disease of the bone (osteitis deformans)**
   a. The serum alkaline phosphatase activity is normal unless the patient has had a recent fracture
   b. The serum phosphate concentration is typically low
   c. There is a high risk of renal stone formation
   d. Adequate therapy includes the administration of a high dose of steroids
   e. There is a periosteal thickening

5. **Osteoporosis:**
   a. Causes an elevation of the serum calcium concentration
   b. Typically causes elevation of the alkaline phosphatase activity
   c. Causes pain in the bones
   d. Improves during bed rest
   e. The response to calcium substitution therapy is usually positive

6. **Carcinoma of the gallbladder:**
   a. The prevalence is higher in males
   b. Associated with cholelithiasis in 90% of cases
   c. Virtually never causes hepatomegaly
   d. The formation of distant metastases is very rare
   e. Commonly develops from an adenomatous polyp

7. **In infectious endocarditis:**
   a. The presence of bacteria within the kidney can usually be demonstrated
   b. Any renal abnormalities are due to immune complex glomerulonephritis
   c. A real hematuria, if present, is not related to the disease
   d. Any renal involvement unfavourably alters the outcome of the disease
   e. A persistent hypocomplementemia is always present

8. **Symptoms of a ventricular septal defect include:**
   a. An elevated jugular vein pressure, even in the absence of cardiac failure
   b. A pansystolic murmur over the apex
   c. A systolic ejection murmur
   d. A Graham-Steel sound, which is a very common finding following the development of pulmonary hypertension
   e. A fixed, widely split second heart sound

9. **Klebsiella-pneumonia:**
   a. Is usually mild
   b. Is frequently associated with a collapse of the upper lobe
   c. Frequently causes lung abscesses
   d. Despite therapy the mortality is 50%
   e. Most commonly develops in youngsters

10. **In pyloric stenosis of infancy:**
   a. There is an autosomal dominant imheritance pattern
   b. Vomiting occurs during the first week of life
   c. The vomit is frequently tinged with bile
   d. An abdominal tumor is nearly always palpable
   e. If left untreated complications during adulthood commonly develop

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