October-December 2016
ISSN 2222-5188

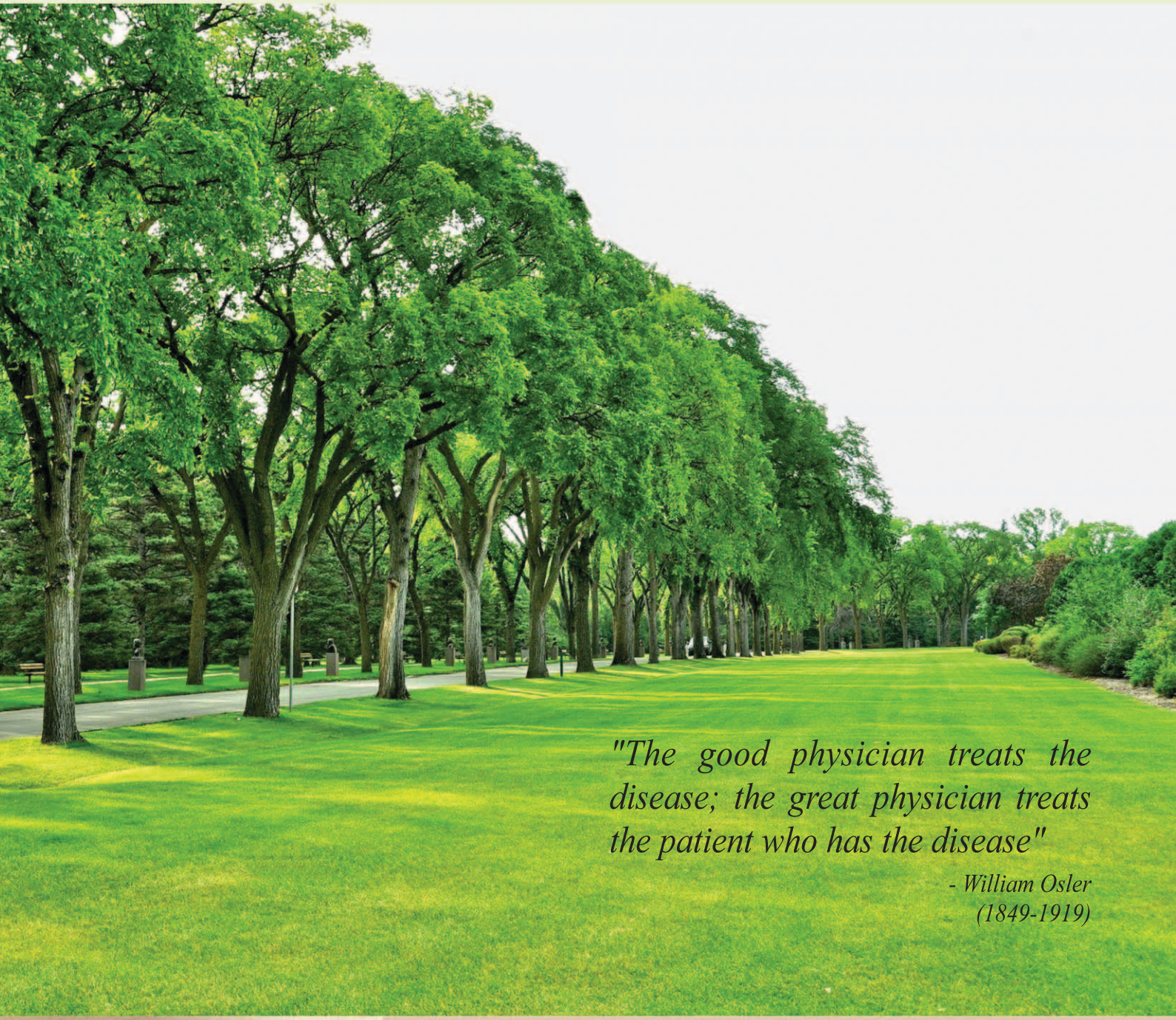
Volume 13 Issue 4

Info MEDICUS

The essence of medical practice

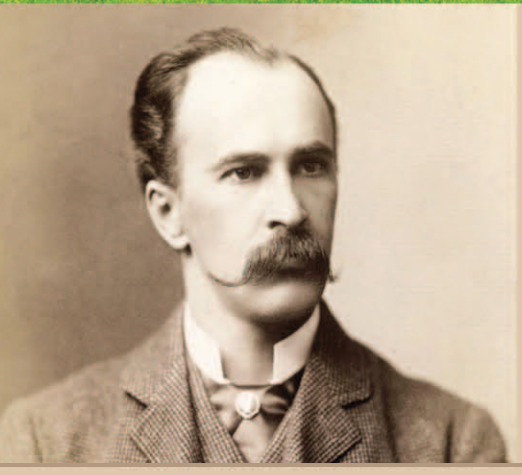
Review Article:
Gestational diabetes mellitus

Case Review:
Gas in the kidney often misdiagnosed as colonic gas



"The good physician treats the disease; the great physician treats the patient who has the disease"

- William Osler
(1849-1919)



Sir William Osler was a Canadian physician and one of the four founding professors of Johns Hopkins Hospital. Osler created the first residency program for specialty training of physicians, and he was the first to bring medical students out of the lecture hall for bedside clinical training. He has frequently been described as the "Father of Modern Medicine". His most famous work, 'The Principles and Practice of Medicine' quickly became a key text to students and clinicians.

CONTENTS

EDITORIAL

Dear Doctor,

On behalf of the entire editorial team, we welcome you to the fourth issue of the Info Medicus 2016. We express our sincere gratitude to all of you for your endless support to Info Medicus in the last 13 years of its journey.

The aim of this publication is to enable the physicians to provide the best possible care to their patients. That's why we always try to cover a broad range of topics relevant to the physicians. In the glossary of this issue, we have chosen "Gestational diabetes mellitus" as our review article. We believe that this informational article will be valuable in your daily clinical practice. Other sections are there as usual.

We appreciate your support and are so happy to have you as a reader of this publication. We will be honored if you share your comments regarding our publication on the Business Reply Post Card.

With warmest thanks,

(Dr. S. M. Saidur Rahman)
Deputy General Manager
Medical Services Department

(Dr. Rumana Dowla)
Manager
Medical Information & Research

Gestational diabetes mellitus

Gestational diabetes mellitus (GDM) is defined as a glucose intolerance resulting in hyperglycaemia of variable severity with onset during pregnancy. During normal pregnancy, insulin sensitivity declines with advancing gestation. These modifications are due to placental factors, progesterone and estrogen. In a physiological situation, a compensatory increase in insulin secretion maintains a normal glucose homeostasis. GDM occurs if pancreatic β cells are unable to face the increased insulin demand during pregnancy. GDM is most commonly a forerunner of type 2 diabetes (T2D) which is the most prevalent form of diabetes. These women share similar characteristics with predisposed subjects to T2D, insulin resistance before and after pregnancy, and carry more T2D risk alleles. Auto immune and monogenic diabetes are more rare aetiologies of GDM. Adverse pregnancy outcomes of GDM are mainly related to macrosomia caused by fetal hyperinsulinism in response to high glucose levels coming from maternal hyperglycaemia. Screening recommendations and diagnosis criteria of GDM have been recently updated. High risk patients should be screened as early as possible using fasting plasma glucose and if normal at 24 to 28 weeks of gestation then using 75g oral glucose tolerance test.

In humans, normal glucose tolerance is maintained because of a balance between adequate insulin secretion and insulin sensitivity. The secretory response of the pancreatic β cells to glucose (particularly in the early phase) and the sensitivity of the glucose utilizing tissues to insulin, determine the ability of insulin to dispose carbohydrates. In individuals with the same degree of glucose tolerance, the product of insulin sensitivity and insulin secretion is constant and the relationship between the two variables follows an approximate hyperbola. This constant is termed the disposition index; it reflects the ability of the β cell to compensate for insulin resistance. It is now well recognized that abnormal glucose tolerance occurs when the pancreatic β cells output do not meet tissues insulin needs in response to changes in insulin resistance.

Mechanism of glucose regulation during pregnancy

Fasting blood glucose decreases at early pregnancy and continuously during gestation. Insulin sensitivity declines with advancing gestation to reach at late gestation (34 to 36 weeks). As a reflection of insulin resistance occurrence, fasting insulin concentrations increase. The changes in insulin sensitivity are inversely related to changes in maternal body fat mass. Hepatic glucose production increases during pregnancy suggest that the defect in insulin action also targets the liver. It is found in a study that there is a significant increase in basal endogenous glucose production at the end of gestation in spite of the important increase in fasting insulin concentration. Endogenous glucose production remained sensitive to insulin infusion throughout gestation.

Alterations in maternal physiology during pregnancy are mediated by placental factors, as evidenced by the significant increase in maternal insulin sensitivity that occurs within days after delivery. Alterations in maternal metabolism have generally been ascribed to placental hormones, such as human placental lactogen (hPL), progesterone and oestrogen. Prolactin, progesterone and oestrogens increase during pregnancy. The lipolytic effect of hPL allows the reorientation of maternal metabolism towards lipid rather than glucose utilization, favouring glucose sparing for the foetus. The consequent increase in free fatty acid levels may participate to insulin sensitivity changes occurring during pregnancy as is the case in non pregnant subjects. However, a direct effect of hPL on mother insulin sensitivity has not been demonstrated. In addition, changes in inflammatory circulating factors such as tumour necrosis factor alpha (TNFα) may also be involved in pregnancy associated insulin resistance. It is reported that the level of placental TNFα is the most important determinant of insulin resistance during pregnancy independently from fat mass changes. Meanwhile insulin secretion increases as a consequence to the development of insulin resistance. In that first and second phase insulin secretion increase by almost 300% throughout gestation. This insulin secretion adaptation is probably due to the rise of maternal hormones which coincides with the development of maternal insulin resistance. In conclusion, the robust plasticity of β cell function in the face of progressive insulin resistance is the hallmark of normal glucose regulation during pregnancy. Diabetes may occur if pancreatic β cells are unable to keep up with heightened insulin demand during pregnancy.

Pathogenesis of gestational diabetes

Insulin resistance during pregnancy stems from a variety of factors, including alterations in growth hormone and cortisol secretion (insulin antagonists), human placental lactogen secretion (which is produced by the placenta and affects fatty acids and glucose metabolism, promotes lipolysis, and decreases glucose uptake) and insulinase secretion (which is produced by the placenta and facilitates metabolism of insulin). In addition, estrogen and progesterone also contribute to a disruption of the glucose insulin balance. Increased maternal adipose deposition, decreased exercise, and increased caloric intake also contribute to this state of relative glucose intolerance.

Risk of gestational diabetes mellitus

- Older mothers, especially over the age of 30 years of age
- Women with a family history of type 2 diabetes
- Women who are overweight
- Women who have had gestational diabetes
- Women who have had large babies or obstetric complications
- Women who have had polycystic ovarian syndrome
- Women from certain ethnic backgrounds including:
  - South Asian
  - Vietnamese
  - Indigenous Australian
  - Chinese
  - Middle Eastern
  - Polynesian

Aetiology of gestational diabetes mellitus

Type 2 diabetes

GDM is most commonly a forerunner of T2D. Women with GDM have a sevenfold risk of T2D compared to women with normal glucose tolerance during pregnancy. There is a strong association between T2D risk alleles and a history of GDM. Study shows that among the susceptibility genes, variants of glucokinase (GCK) and TCF7L2 loci are associated with higher glucose levels during oral glucose tolerance tests in pregnant women.

Monogenic diabetes

Monogenic form of diabetes may also be revealed during pregnancy. It has also been shown that common variants in maturity onset diabetes of the young (MODY) genes contribute to GDM, like polymorphism of the promoter of GCK and polymorphism of hepatocyte nuclear factor 1a (HNF1a). MODY refers to any of the several forms of hereditary diabetes caused by mutations in an autosomal dominant gene influencing insulin production. One of these forms is MODY 2, which seems to be the most frequently associated with GDM, with a prevalence of around 10% of GDM. It is due to mutations of the GCK gene. Additionally, included women were insulin treated during at least one pregnancy and they had a history of T2D, GDM or fasting hyperglycaemia in a first degree relative. Few MODY 3 and MODY 4 have also been reported in GDM women.

Type 1 diabetes

Autoimmune diabetes may also be considered as aetiology of GDM. The prevalence of autoimmune markers of type 1 diabetes (T1D) is between 0.98 and 14.7% in women with GDM. It predicts later development of T1D in these women but not necessarily. Autoimmunity was associated with poor pregnancy outcomes (fetal death, preterm delivery and macrosomia).

Other factors

Some factors like ethnicity and race may be at the origin of GDM onset. GDM may result from interaction between genetic and environmental risk factors. Old age, obesity and high fat diet represent some important non genetic factors.

Screening and diagnosis of GDM

There is debate regarding the preferred screening protocol for GDM. Some experts recommend universal screening because not all women who develop GDM have risk factors. The american diabetes association (ADA) policy states that screening may be omitted in low risk women. A woman is considered low risk if all of the following factors are present: age younger than 25 years; BMI less than 25 before pregnancy; not of Hispanic, African American, American Indian, South or East Asian; no first degree relative with diabetes mellitus (DM); no history of abnormal glucose tolerance and no history of poor obstetric outcome. The American College of Obstetricians and Gynecologists (ACOG) practice bulletin, states that universal screening is the most sensitive and more practical approach, but it notes that low risk women may be excluded from screening per the ADA recommendation. The United States Preventive Services Task Force on Preventive Health Care concluded that there is not enough evidence to support or deny universal screening for GDM.

Screening

When the universal screening approach is employed, patients with no known risk factors should undergo a 1 hour glucose test at 24 to 28 weeks of gestation. Patients with known risk factors that indicate the possibility of glucose intolerance may be tested at the onset of prenatal care. If this initial screen is normal, then the test is repeated at the beginning of the third trimester (24 weeks).

For the glucose test, the patient receives 50 g of glucose. One hour later, blood is drawn for a plasma glucose determination. A glucose value above 130 to 140 mg/dl is considered abnormal and necessitates a second test, the 3 hour glucose tolerance test. To perform glucose tolerance testing (GTT), first draw a fasting glucose sample and then administer 100 g of glucose. Blood for glucose values is drawn at 1 hour, 2 hours and 3 hours. Although some perform a 75 g 2 hour GTT as both a screening test and a diagnostic test.

Diagnosis

In the Carpenter or Coustan conversion method, diagnosis of GDM is based on the presence of 2 or more of the following factors:

- Fasting serum glucose concentration exceeding 95 mg/dl
- 1 hour serum glucose concentration exceeding 180 mg/dl
- 2 hour serum glucose concentration exceeding 155 mg/dl
- 3 hour serum glucose concentration exceeding 140 mg/dl

Alternatively, some employ the National Diabetes Data Group (NDDG) criteria, which are slightly more liberal. The abnormal values are as follows:

- Fasting serum glucose concentration exceeding 105 mg/dl
- 1 hour serum glucose concentration exceeding 190 mg/dl
- 2 hour serum glucose concentration exceeding 165 mg/dl
- 3 hour serum glucose concentration exceeding 145 mg/dl

Management

The treatment of GDM intends to decrease adverse pregnancy outcome. There is insufficient knowledge of clinical outcomes of both lifestyle as well as pharmacological interventions against GDM using the new criteria. Treatment is based on blood glucose targets independently from the aetiology of GDM. Diabetes phenotyping may be performed later on as it is usually done during pregnancy in routine diabetes care. Treatment evaluation is based on blood glucose self monitoring (pre and 1 hour or 2 hour post prandial according to recommendations) and not on A1c level. Blood glucose targets are shown in Table 1. Education is the cornerstone of GDM management. Trained nurses and dieticians are the most effective in this regard. The aim of dietary therapy is to avoid large meals and simple carbohydrate rich foods. Insulin therapy is added if targets are not obtained with lifestyle modifications alone. The efficacy and safety of insulin have made it the standard for treating GDM. Oral antidiabetic agent metformin and glyburide have shown efficacy with no evidence of harm to the foetus, although long term safety remains a concern. Trials have shown that glyburide does not cross the placental barrier or may cross it in low concentrations while metformin concentration is similar in the fetal and maternal circulation. Metformin in gestational diabetes trial, the largest study of metformin use in women with GDM compared to insulin therapy. There was no significant difference in the fetal outcome between the two groups and approximately half of the metformin treated mothers also required insulin in order to achieve target glucose levels. Nevertheless, metformin seems to be favourable with regards to weight gain and amount of insulin needed during pregnancy.

Table 1: Target blood glucose level for GDM patients	
Time of day	**Targets mg/dl (mmol/l)**
Preprandial or fasting	95 (5.3) or lower
1 hour after meal	140 (7.8) or lower
2 hour after meal	120 (6.7) or lower

Complication of GDM

Women with GDM experience twice the number of urinary tract infections than women who do not have GDM. This increased infection incidence is thought to be due to the increased amount of glucose in the urine beyond the normal glucosuria that is present in pregnancy. There is also an increased risk of pyelonephritis, asymptomatic bacteriuria and preeclampsia. There is a 10% risk of polyhydramnios that may increase the risk of abruption placenta and preterm labor as well as of postpartum uterine atony. Congenital anomalies do not occur at an increased rate in patients with GDM. There is reportedly an increased incidence of stillbirth when glucose control is poor.

There is also a 10% per year risk of developing type 2 diabetes after the pregnancy in which GDM occurred, with the greatest risk within the first 5 years following the index pregnancy. Macrosomia, if it occurs, typically becomes evident at 26 to 28 weeks of gestation. Complications associated with macrosomia include fetopelvic disproportion leading to operative delivery, shoulder dystocia and neonatal hypoglycemia. There is an increased incidence of hyperbilirubinemia, hypocalcemia, respiratory distress syndrome and polycythemia in the neonate. Long term complications can include obesity, diabetes during childhood, impaired motor function, and higher rates of inattention and hyperactivity.

Prevention

Development of diabetes mellitus (DM) after GDM is determined by some modifiable factors and some unmodifiable factors. Non modifiable risk factors for development of DM from GDM are ethnicity, age and family history. There is very little scope to target this. But targeting the modifiable risk factors like multiple pregnancy obesity or weight gain during or after GDM, dietary indiscretion, sedentary habit, hyperlipidaemia, hypertension and smoking is an achievable goal. The procedure which can be achieved are regular follow up and biochemical monitoring, regulation of diet, exercise, smoking, hypertension, lipid abnormality, avoidance of further pregnancy, and hormone replacement therapy in menopausal age group, all being supplemented by patient education and public awareness. Education programme delivered during pregnancy should also include the risk of recurrent GDM and future DM, hypertension and dyslipidaemia and thereby the need for regular follow up. They should also know that frequently diabetes develop without any symptoms and should also know the symptoms for self referral.

For ethnic groups with a high prevalence of DM, blood sugar estimation immediately after delivery, after 2 months and every year thereafter should be followed. Risk of developing DM is highest between 6 months to 5 years but becomes a plateau after ten years. High risk group during or after delivery should be monitored more frequently. Till now as for diabetes prevention lifestyle modification is best. Immunomodulatory treatment of women with GDM is another option for prevention of type 1 DM. One of the important causes of DM developing after GDM is discontinuity of care as neglected by young women after delivery. Current American Diabetes Association guidelines recommend blood sugar estimation after child birth, after 6 to 8 weeks and every 3 years thereafter. Women with high risk factors require more frequent testing.

Summary

GDM is a problem that affects a significant number of women during pregnancy. GDM can have lasting health impacts on both the mother and the fetus. In order to circumscribe and minimize potential complications to both mother and child, screening, diagnosis, and management of hyperglycemia are critical. There is still work to be done to gain a better sense of what screening protocols are most efficacious and cost effective, and when they should be administered. Future studies will provide guidance as to what the optimal management choices are. Insulin in addition to oral hypoglycemic therapy appears to be the focus of research in the near future.

References: *1. Eur. J. Endocrinol., February 2016; 174(2)*
2. Medicine Update 2005, P 842-8454
3. The National Diabetes Services Scheme (NDSS)
4. Reviews in Obstetrics & Gynecology, 2008; Vol. 1, No. 3

Aged muscles heal slowly

As we age, the function and regenerative abilities of skeletal muscles deteriorate, which means it is difficult for the elderly to recover their muscle from injury or surgery. Carnegie's Michelle Rozo, Liangji Li, and Chen Ming Fan from New work demonstrate that a protein called b1-integrin is crucial for muscle regeneration.

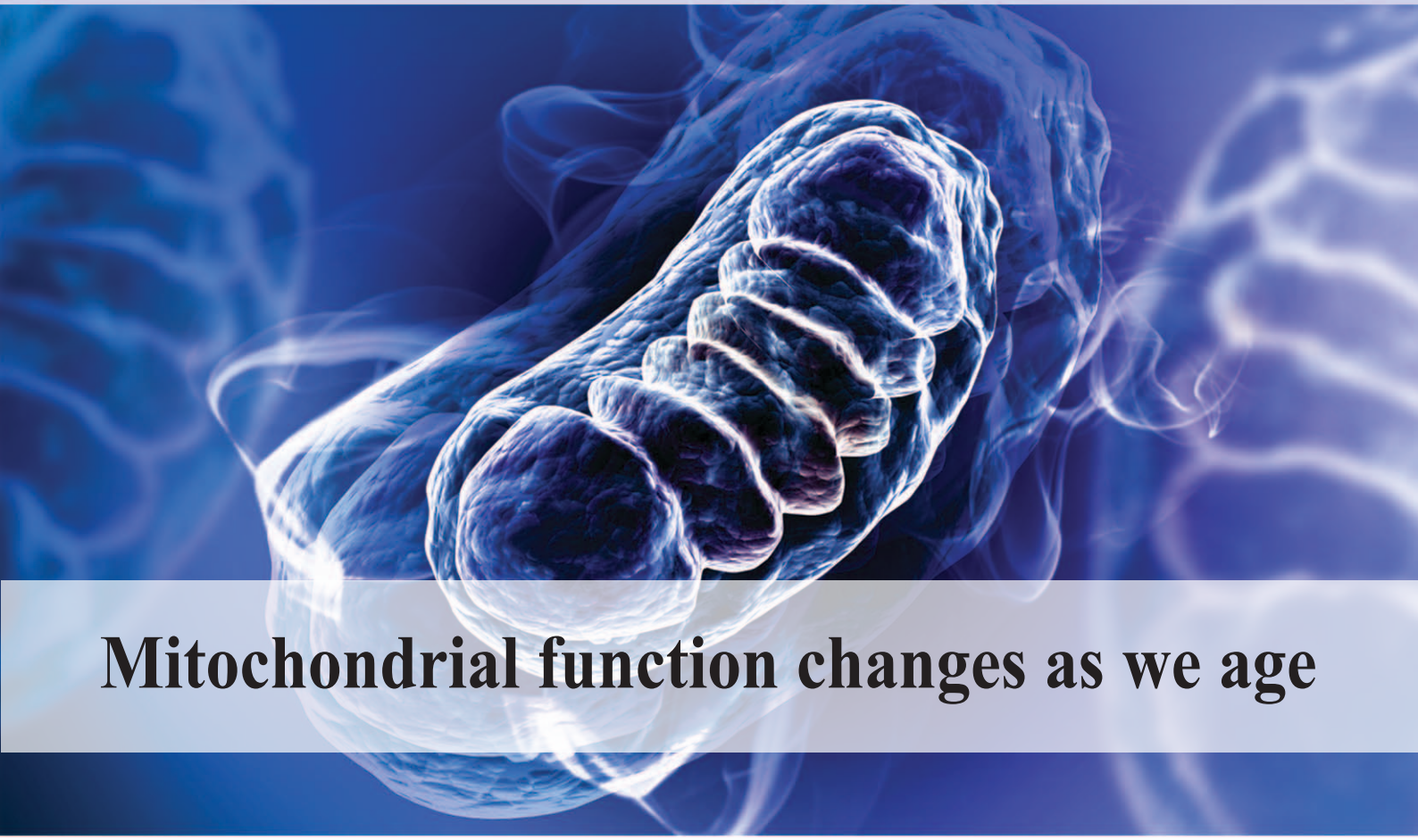
Muscle stem cells are the primary source of muscle regeneration after injury. These specialized adult stem cells lie dormant in the muscle tissue; off to the side of the individual muscle fibers, which is why they were originally dubbed satellite cells. When muscle fibers are damaged, they activate and proliferate. Most of the new cells go on to make new muscle fibers and restore muscle function. Some return to dormancy, which allows the muscle to keep repairing itself over and over again.

Rozo, the lead author, determined that the function of integrins (or, more specifically, the protein called b1-integrin) is absolutely crucial for maintaining the cycle of hibernation, activation, proliferation, and then return to hibernation, in muscle stem cells. Integrins are proteins that 'integrate' the outside to the inside of the cell, providing a connection to the immediate external environment, and without them, almost every stage of the regenerative process is disrupted.

The team theorized that defects in b1-integrin likely contribute to phenomena like aging, which is associated with reduced muscle stem cell function and decreased quantities of muscle stem cells. This means that healing of the muscle wound after injury or surgery is very slow, which can cause a long period of immobility and an accompanying loss of muscle mass. Rozo and Li determined that the function of b1-integrin is diminished in aged muscle stem cells. Furthermore, when they artificially activated integrin in mice with aged muscles, their regenerative abilities were restored to youthful levels. Importantly, improvement in regeneration, strength, and function were also seen when this treatment was applied to animals with muscular dystrophy, underscoring its potential importance for the treatment of muscle disorders.

Muscle stem cells use b1-integrin to interact with many other proteins in the muscle external environment. Among these many proteins, they found a clue that one protein called fibronectin might be most relevant. To connect b1-integrin to fibronectin, they teamed up with many other groups. They discovered that aged muscles contain substantially reduced levels of fibronectin compared to young muscles. Like b1-integrin, eliminating fibronectin from young muscles makes them appear as if they were old, and restoring fibronectin to aged muscle tissue restores muscle regeneration to youthful levels. Their joint efforts demonstrated a strong link between b1-integrin, fibronectin and muscle stem cell regeneration.

Reference: *www.medicalxpress.com*



Mitochondrial function changes as we age

A new study finds that age related onset of type 2 diabetes and impaired glucose tolerance may be due to the lowered ability of muscle mitochondria to switch from metabolizing fatty acids to metabolizing glucose in healthy elderly people compared to young people. People over the age of 65 years are more likely to develop type 2 diabetes or glucose intolerance. The reasons for this are largely unknown, but studies have shown that muscle insulin resistance, increased intra myocellular lipid content (IMCL), and decreased metabolic rates are related to aging. Because of this, scientists are interested in studying differences in the function of mitochondria located within muscle cells of elderly people compared to young people. However, singling out mitochondrial function in muscle cells has been difficult to do using typical indirect calorimetry method. In an effort to gain a more accurate picture of mitochondrial function in muscle cells, several scientist, developed a method for studying the flux of two key metabolic enzymes using liquid chromatography tandem mass spectrometry (LC-MS/MS) and 13C-labeled glucose.

During cell metabolism, pyruvate enters the mitochondria, undergoes a series of reactions that produce carbon dioxide, water and acetyl-CoA. In the first step of the process, the pyruvate reaction is catalyzed by an enzyme called pyruvate dehydrogenase. Acetyl-CoA converts to citrate through the enzyme citrate synthase. This study assessed the ratio of the flux of pyruvate dehydrogenase to citrate synthase within muscle mitochondria after a twenty four hour fast and after insulin stimulation in healthy, non smoking elderly subjects (average age was 69 years). They were matched with young subjects (average age was 27 years).

The flux of pyruvate dehydrogenase and citrate synthase in muscle cells was assessed by taking muscle biopsies before and after insulin stimulation. After fasting, fluxes of both enzymes were the same for the elderly subjects and the young subjects. This was different from the indirect calorimetry method that showed a higher respiratory quotient in the elderly group than the younger group, indicating that the elderly group had a lower metabolic rate. Their muscle specific technique, however, was able to single out muscle cells. Their studies showed that after fasting, both groups had a similar rate of pyruvate dehydrogenase to citrate synthase flux in muscle cells. After insulin stimulation, the ratio of the flux of pyruvate dehydrogenase to citrate synthase increased three fold in the younger subjects, while the rate did not change in the elderly.

These results indicate that after insulin stimulation, the younger group switched from lipid oxidation to glucose oxidation, while this switch did not occur in the elderly group. This points to a problem in insulin signaling in the muscle associated with aging and may be a contributing factor to the higher incidence of type 2 diabetes and impaired glucose intolerance in elderly people.

Reference: *www.medicalxpress.com*

Intraosseous catheter placement in children

Overview

Obtaining vascular access in acutely ill or injured children can be challenging. Intraosseous access provides a means for delivery of medications, crystalloid fluids, colloids, and blood products during pediatric resuscitation. In addition, when intraosseous access is achieved, blood samples can be obtained for laboratory analysis. An intraosseous catheter can be placed more rapidly than a central venous catheter and that this technique is more reliable than venous cut down.

Indication

Intraosseous cannulation is indicated when peripheral vascular access cannot be rapidly obtained in an infant or child in shock, respiratory failure, or respiratory or cardiac arrest. The most recent guidelines on pediatric advanced life support state that intraosseous access is useful as the initial vascular access in cases of cardiac arrest.

Anatomy

It is possible to introduce fluid and medications through the bones because of the connections between the marrow cavity and the systemic venous circulation (Figure 1). The cortex overlying the metaphysis of long bones is relatively thin and easy to penetrate. The administered substance passes through the underlying cancellous bone by means of venous sinusoids, which in turn drain into nutrient vessels and emissary veins that connect with systemic venous circulation. Cannulation anywhere within the non-collapsible medullary cavity provides a reliable means of infusing fluids or medications into the systemic venous circulation.

Site selection

Several anatomical sites may be used for intraosseous cannulation. The most commonly used sites that include the proximal tibia, the distal tibia, the proximal humerus, and the distal femur. The anterior superior iliac spine is used less commonly. The sternum and distal radius have been identified for use in adults but not in children.

The proximal tibia is the preferred site in children because palpation and identification of underlying bony landmarks are less likely to be obscured by large amounts of soft tissue. In addition, this location is remote from the head and chest, where airway management and chest compressions may be ongoing in an emergency situation. The prominence immediately below the lower pole of the patella is the tibial tuberosity. The broad, flat surface of tibia that is 1 to 2 cm inferior and medial to the tuberosity serves as the targeted insertion site.

The distal tibia has easily palpable landmarks in most children. It has less cortical thickening than the proximal tibia and is therefore preferred in older children. The intraosseous insertion site spans the flat portion of the tibia, 1 to 2 cm proximal to the superior margin of the malleolus.

When tibial sites are not available or when previous attempts of intraosseous cannulation at these sites have been unsuccessful, the proximal humerus can be used. Begin by palpating the mid portion of the humerus in the upper arm. Follow the bone proximally until the greater tubercle is appreciated, just anterior to the midline of the lateral shoulder and distal to the shoulder joint. This bony prominence is the insertion site.

The distal femur can be cannulated for intraosseous infusion, although abundant overlying soft tissue and muscle often make identification of bony landmarks challenging. Therefore, this site should be considered only when the use of tibial and humeral sites is contraindicated. The distal femoral insertion site is in the midline, 1 to 3 cm proximal to the palpable epicondyles of the distal femur.

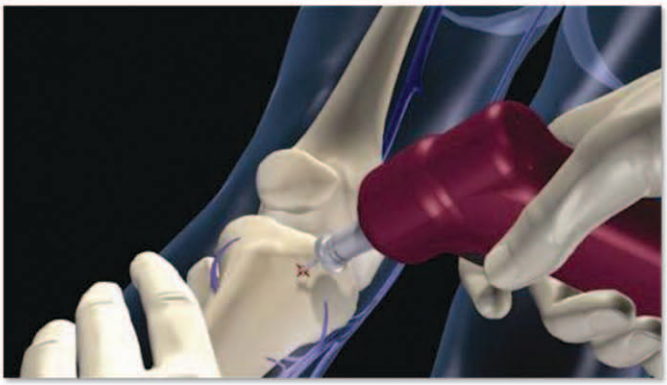


Figure 1: Infusion through the marrow cavity and emissary veins to the systemic venous circulation

Equipment

- Manual needles: It is designed specifically for intraosseous access and are available for use in children of all ages. These needles have cutting stylets to prevent soft tissue or bony spicules from obstructing the cannula, ergonomic handles to facilitate placement, and depth markings or an adjustable flange to guide depth of insertion
- Large bore spinal and butterfly needles: It may be used in infants if an intraosseous needle is not available
- A spring loaded device: It allows the single deployment of a needle to a preset depth of insertion, which is calculated on the basis of the patient's age
- Drill assisted device: It allows placement of needles of varying lengths suitable for children or adults
- The battery powered driver and cutting needle: It facilitates penetration of the bony cortex, and the operator can control the depth of insertion by increasing or decreasing the pressure applied and by changing the length of time the drill trigger is depressed

Procedure

Steps of procedure are:

Preparation

Place all equipment on an open surface that is readily accessible. Make sure that approved biohazard sharps receptacles have been placed nearby, since intraosseous cannulae are not equipped with retractable safety needles. Whenever possible, identify the patient with the use of two identifiers before beginning the procedure and whenever a procedural time out is initiated. Explain the procedure to the conscious patient and to any family members who are present. When no surrogate adult is available, this urgent life saving procedure can be performed without informed consent.

Position the patient so that the selected insertion site is easily accessible. When using the proximal tibia or distal femoral sites, placement of a rolled towel under the popliteal fossa may help to flex the knee and maintain stability of the leg. Wear protective eye wear and sterile gloves. Using aseptic technique, clean the chosen insertion site with chlorhexidine or povidone iodine solution. In patients who are awake and alert, infiltrate the area with lidocaine to provide local anesthesia.

Manual technique

When using manual insertion, remove the safety cap from the needle. Ensure that the stylet is appropriately placed within the needle and that the bevels are aligned. Using non dominant hand, stabilize the limb distal to the insertion site. This allows for counter pressure against the advancing needle and prevents unexpected patient movement. To decrease the risk of needle stick injury, make sure that no portion of the hand is behind the insertion site. Place the needle handle in the palm of the dominant hand, with thumb and forefinger positioned along the shaft of the needle for stabilization. Place the needle against the skin overlying the site.

Puncture the skin and continue through the soft tissue. Use firm, steady pressure and a rotating or coring motion to penetrate the bony cortex. Avoid continued pressure at this point, which could push the needle through the opposite side of the bone. Remove the needle cap and stylet. If the device has a supporting flange, adjust it so the surface is flush with the skin to stabilize the needle and to avoid inadvertent deeper insertion. Take care to avoid disrupting the needle while adjusting the flange.

Drill assisted technique

For drill assisted insertion, select the appropriate needle size on the basis of the patient's weight. The pediatric needle is designed for use in children weighing less than 40 kg, unless large amounts of overlying soft tissue indicate the need for a longer needle. Remove the needle from its protective container. Attach the needle set to the driver, allowing the magnetic pull to hold it in place. Turn the safety cap clockwise to remove it and pull it off the needle. Place the needle against the skin overlying the site. Be careful to insert the needle 1 to 2 cm away from the adjacent physis. To allow the needle to penetrate the soft tissue and then the bony cortex, apply gentle, steady pressure while engaging the trigger. Once resistance decreases, mark the entrance into the medullary cavity, release the trigger and allow the driver to stop spinning before pulling back or disconnecting the needle. Gently hold the needle and pull the drill directly backward and off the needle to disconnect. Turn the stylet counter clockwise to unscrew it, and gently remove it from the needle set.

Spring loaded technique

For spring assisted insertion, select the appropriate depth of penetration in accordance with the patient's age. Firmly position the device perpendicular to the insertion site, holding the device with the dominant hand. Pull out the safety latch with the non dominant hand. Trigger the device by pushing down with the palm of the hand while continuing to hold the bottom firmly against the skin. Slowly pull up the casing around the inserted needle. Remove the stylet and secure the needle with the safety latch. Place the stylet in a biohazard sharps receptacle.

A syringe can be attached directly to the needle hub. Alternatively, attach extension tubing to the hub to avoid further needle movement. Use wide bore tubing when available. The marrow specimen obtained on aspiration can be used for bedside glucose testing and sent for culture and determination of blood type, electrolyte concentrations, drug levels, and pH and partial pressure of carbon dioxide ($PaCO_2$). Samples should not be sent for a complete blood count because immature cell forms from the marrow space will not accurately represent findings from the peripheral circulation. If fluids or medications have been previously infused, samples should not be sent for laboratory analysis after more than 5 minutes of resuscitation. Once blood samples have been sent, a 10 ml saline flush is recommended to open venous sinusoids for further infusion of crystalloid, colloid, or medications.

In conscious children, pretreatment with a single dose of lidocaine (0.5 mg per kilogram of body weight) through the intraosseous catheter can be effective in preventing the visceral pain that results from the increased intra medullary pressure caused by the infusion of fluids.

Confirmation of proper placement

A number of methods can be used to confirm correct catheter placement. First, the needle should stand on its own, because of the lateral support provided by the bony cortex. Aspiration of bone marrow contents also signifies that the catheter is in the appropriate cavity. However, it is important to note that blood return may not occur even when an intraosseous catheter is placed properly. Absence of local swelling at the insertion site on infusion with a saline flush also indicates correct placement. Because the bone marrow cavity is not distensible, it is normal to sense resistance during manual infusion into the intraosseous cannula. Fluoroscopy and ultrasonography have been used to confirm placement, but their use is not routine.

Removal of the catheter

The intraosseous catheter should be removed as soon as more definitive access is obtained. To remove an intraosseous needle, loosen and remove any tape or dressing securing the cannula and extension tubing to the skin. Rotate the syringe and catheter clockwise while gently pulling the needle from the extremity. Place a sterile occlusive dressing over the insertion site.

Contraindication

Intraosseous access should not be attempted in any bone with a suspected or known fracture near or proximal to the insertion site or in a bone in which a previous attempt was unsuccessful. In these circumstances, infused medications or fluids may not reach the central circulation and extravasation during fluid administration may lead to compartment syndrome.

Indwelling hardware from a previous orthopedic procedure is a relative contraindication, since it may prevent successful cannulation in that bone. The presence of infection in the skin or soft tissue overlying the insertion site is also a relative contraindication, since passing a needle through the infected area may introduce bacteria into the bone or the systemic circulation. Avoid intraosseous cannulation in patients with underlying bone diseases, such as osteogenesis imperfecta or osteopenia, whose bone may not tolerate or support cannulation. In patients with osteopetrosis, a condition characterized by very dense bone, penetration through the thickened bony cortex may not be possible.

Summary

Appropriate placement of an intraosseous catheter is a reliable means of obtaining urgent vascular access in children and is associated with low rates of reported complications. Both manual and power assisted placement techniques can be used to deliver fluids and medications rapidly during pediatric resuscitation.

Reference: *N. Eng. J. Med. February 24, 2011; 364;8:e14*

Gas in the kidney often misdiagnosed as colonic gas

Introduction

Gas in the kidney or emphysematous pyelonephritis (EPN) is defined as a severe, necrotizing renal parenchymal infection that is characterized by the bacterial production of gas within the kidney parenchyma. Schultz and Klorfein first used the term 'EPN' in 1962, but the condition might have already been described by Kelly and Mac Cullem as far back as the end of the previous century. EPN involves a spectrum of disease processes that result in the production of gas in the renal parenchyma; the gas can be focal or diffuse, and can spread to the collecting system or track into the perinephric and paranephric spaces. EPN can be classified into many ways and most of them are based on their CT findings. Middle aged females with diabetes comprise the majority of patients with EPN.

Case report

A 40 years old female diabetic (insulin dependent) was admitted into hospital through emergency room (ER) to the medical ward with complaints of left sided abdominal pain, tightness of chest and decreased urine output for 7 days. There was no history of fever, cough or dysuria. She was admitted in a primary level hospital as a case of acute abdomen and treated conservatively for 3 days. As there was no improvement she was referred to a higher centre. In the medical ward she was treated as a patient of urosepsis with acute kidney injury. On the third day of admission she was diagnosed as left renal stone. She did a plain X-ray of kidney and urinary bladder (KUB) outside the hospital which was reported as normal except distended colonic gas shadow. By looking to the X-ray of KUB region with the diagnosis emphysematous pyelonephritis, she was transferred to urology department. Clinical evaluation revealed a co-operative but very ill patient. The temperature was 98^0F, pulse rate was 88 beats per minute, blood pressure was 120/70 mm of Hg and the respiratory rate was 18 breaths per minute. Cardiovascular and respiratory systems were within normal limits. Abdominal examination revealed severe tenderness at the left upper abdomen and the left renal angle. There was no mass palpable, and the remainder of the physical examination was normal.

Laboratory investigations on admission showed a haemoglobin of 10.6 gm/dl (normal range: 11.5 gm/dl to 16.5 gm/dl), total leukocyte count of 11.5 k/µl (normal 4.0 k/µl to 11 k/µl) with 88 % neutrophils (normal 40 % to 75 %). The platelet count was 99 k/µl (normal 150 k/µl to 400 k/µl). The serum creatinine was 2 mg/dl

(normal range: 0.4 mg/dl to 1.4 mg/dl) and blood urea was 116 mg/dl (normal range : 35 mg/dl to 40 mg/dl). The random blood sugar was 15 mmol/l (normal range 3.90 to 6.10 mmol/l). The serum electrolyte was sodium 130 mmol/l , potassium 4.8 mmol/l (normal range : Sodium 135 to 145 mmol/l and potassium 3.5 to 4.5 mmol/l). Urine microscopic examination revealed plenty pus cells, sugar 3+ with trace amount of blood and ketones. Urine and blood cultures were sent and both showed no growth of bacteria possibly due to taking of antibiotic prior to admission. Blood group was B positive. CRP was 236.9 mg/dl (normal range <5 mg/dl).

A plain X-ray of the abdomen revealed kidney shaped gas in the left renal area. Computerised tomography of the KUB region confirmed the presence of gas in the renal and perirenal area with extensive renal parenchymal destruction and pus tracking towards the left iliac fossa. The diagnosis was type II EPN. The patient was initially treated with meropenem 500 mg 8 hourly. An emergency PCN (percutaneous nephrostomy) was performed which immediately drained 100ml pus. As the patients condition was not improving expectedly left nephrectomy was done with left subcostal incision on the next day. Post operative period was otherwise uneventful except one day stay in ICU for delayed extubation. She was discharged on the 8th POD with IV antibiotics for another 7 days. 6 months follow up was excellent.

Discussion

EPN is a rare renal infection. However due to modern imaging techniques and practice of routine ultrasound more cases are reported now a days. EPN predominantly affects females. The female to male ratio is 3:1. The left kidney is more frequently involved than the right (60% Vs 35 %). Both the kidneys are involved in about 5% of the reported series. 90% of the reported cases have occurred in diabetic patients. EPN has also been reported in debilitated (alcoholic) and immunocompromised patients. EPN can be classified into many ways and most of them are based on their CT findings. One of the classifications of EPN by Huang and Tseng (based on CT) is Class I: Gas in collecting system only. Class II: Parenchymal gas only. Class IIIa: Extension of gas into perinephric space. Class IIIb: Extension of gas into pararenal space. Class IV: EPN in solitary kidney, or bilateral disease. Another simple classification of EPN by Wan et al 3 (based on CT) Type I: Renal necrosis with presence of gas but no fluid. Type II: Parenchymal gas associated with fluid in renal parenchyma, perinephric space or collecting system.

The organisms commonly responsible for causing EPN are *Escherichia coli, Klebsiella pneumoniae, Proteus mirabilis, Pseudomonas aeruginosa,* Citrobacter and rarely yeast. If left untreated the condition is uniformly fatal. The exact mechanism of gas formation in EPN is not known. Gas formation is believed to be due to pathogenic bacteria capable of mixed acid fermentation acting in a hyperglycaemic environment on tissues that are ischaemic. This results in tissue destruction, and encourages purulent infection and inhibition of the removal of locally produced gas.

The clinical presentation of emphysematous pyelonephritis was similar to that of upper urinary tract infection with fever, nausea and vomiting, lethargy, confusion, dyspnoea and shock. Laboratory data showed high glycosylate haemoglobin, leukocytosis, thrombocytopenia and pyuria.

Diagnosis of EPN rests on the clinical awareness and confirming it by appropriate investigations. The triad of symptoms of fever, flank pain and pyuria especially in diabetic patients who do not respond promptly to antibiotic treatment must raise the possibility of EPN. These patients require to be investigated and treated aggressively. The diagnosis of EPN is classically made by demonstrating gas in the renal or perirenal tissue by plain abdominal X-ray. However gas can be demonstrated only in 33% of plain abdominal radiographs in patients with EPN. Even by abdominal ultrasonography it may be technically difficult to distinguish the renal gas filled area from gas in the bowel. On the other hand, CT scan can not only confirm the diagnosis, but also show the extent of the disease. Abdominal CT scan is recommended for all patients in whom EPN is suspected.

Management of patients with emphysematous pyelonephritis has been a subject of controversy. Huang and Tseng reviewed the management of 48 patients with emphysematous pyelonephritis. They concluded that for localized emphysematous pyelonephritis (class I and II) according to CT scan, percutaneous drainage with antibiotic treatment can provide a good outcome. For extensive emphysematous pyelonephritis (class III and IV) with more benign manifestations, when saving the kidney is possible, percutaneous drainage combined with antibiotic treatment may be attempted because of its high success rate. However, nephrectomy can provide the best management outcome and should promptly be attempted for extensive emphysematous pyelonephritis with a fulminant course. The rapidly deteriorating general condition of the patient and the onset of septicaemic shock prompted us to go ahead with nephrectomy rather than adopt a more conservative line of management.

Conclusion

EPN is a severe and often life threatening infection. CT Scan is the investigation of choice for not only making a proper diagnosis but also in planning the treatment option. Renal preservation must be the aim of treatment, but this must not be at the cost of patient's life. One should not hesitate to resort to nephrectomy as and when indicated.

Reference: *J. Bang. Coll. Phys. Surg. 2015;33:91-94*

Inguinal hernia - at a glance

Inguinal hernias are one of the most common reasons where a patient may need surgical intervention. The history and physical examination are usually sufficient to make the diagnosis. Symptomatic patients often have groin pain, which can sometimes be severe. Inguinal hernias may cause a burning, gurgling, or aching sensation in the groin, and a heavy or dragging sensation may worsen towards the end of the day and after prolonged activity. An abdominal bulge may disappear when the patient is in the prone position. Examination involves feeling for a bulge or impulse while the patient coughs or strains. Although imaging is rarely warranted, ultrasonography or magnetic resonance imaging can help to diagnose a hernia in an athlete without a palpable impulse or bulge on physical examination. Ultrasonography may also be indicated with a recurrent hernia or suspected hydrocele, when the diagnosis is uncertain, or if there are surgical complications. Although most hernias are repaired, surgical intervention is not always necessary, such as with a small, minimally symptomatic hernia. If repair is necessary, the patient should be counseled about whether an open or laparoscopic technique is best. Surgical complications and hernia recurrences are uncommon. However, a patient with a recurrent hernia should be referred to the surgeon.

Anatomy of inguinal canal

- The inguinal canal is a passage in the anterior abdominal wall which conveys the spermatic cord in men and in women the round ligament
- Extends from the deep inguinal ring (fascia transversalis) to the superficial inguinal ring and parallel and above the medial half of inguinal ligament
- Deep inguinal ring: Midway between anterior superior iliac spine and the symphysis pubis, lateral to the inferior epigastric vessels and margins of ring gives origin to the internal spermatic fascia
- Superficial inguinal ring: Triangular defect in the aponeurosis of the external oblique immediately above and medial to the pubic tubercle and margins give origin to the external spermatic fascia
- A horizontal line stretching from anterior iliac spine to lateral margin of rectus abdominis

- The inguinal canal is larger and more prominent in men
- Walls of inguinal canal: Anterior, posterior, superior and inferior
- Structures passing through the inguinal canal:
  - Male: Spermatic cord, ilioinguinal nerve
  - Female: Round ligament, ilioinguinal nerve, lymph vessels

Inguinal hernia

An inguinal hernia happens when contents of the abdomen, usually fat or part of the small intestine bulge through a weak area in the lower abdominal wall. The area of the lower abdominal wall is also called the inguinal or groin region. There are two major types of inguinal hernia: indirect inguinal hernia and direct inguinal hernia.

Indirect inguinal hernias: It is caused by a defect in the abdominal wall that is congenital, or present at birth.

- 20 times more common in males
- One third are bilateral
- More common on the right
- Congenital in origin
- Hernial sac is the remains of the processus vaginalis
- The sac enters the inguinal canal through the deep inguinal ring lateral to the inferior epigastric vessels

Direct inguinal hernias: It usually occurs only in male adults and are caused by a weakness in the muscles of the abdominal wall that develops over time.

- Majority is bilateral
- The sac bulges directly anteriorly through the posterior wall of the inguinal canal
- Medial to the inferior epigastric vessels
- A disease of aged people with weak abdominal muscles

Risk factors

Factors that contribute to developing an inguinal hernia include:

- Being male: Men are eight times more likely to develop an inguinal hernia than are women
- Being older: Muscles weaken as age
- Family history
- Chronic cough
- Chronic constipation: Constipation causes straining during bowel movements
- Pregnancy: Being pregnant can weaken the abdominal muscles and cause increased pressure inside the abdomen
- Premature birth and low birth weight
- Previous inguinal hernia or hernia repair

Clinical feature

The first sign of an inguinal hernia is a small bulge on one or rarely, on both sides of the groin (the area just above the groin crease between the lower abdomen and the thigh). The bulge may increase in size over time and usually disappears when lying down. Other signs and symptoms can include:

- Discomfort or pain in the groin especially when straining, lifting, coughing, or exercising that improves when resting
- Feelings such as weakness, heaviness, burning, or aching in the groin
- A swollen or an enlarged scrotum in men or boys

Diagnosis

An inguinal hernia can be diagnosed by:

- A medical and family history
- A physical examination
- Imaging tests, including
  - Abdominal X-ray
  - Computerized tomography (CT) scan
  - Abdominal ultrasound

Treatment

Repair of an inguinal hernia via surgery is the only treatment for inguinal hernias and can prevent incarceration and strangulation. Research suggests that men with hernias that cause few or no symptoms may be able to safely delay surgery until their symptoms increase. Men who delay surgery should watch for symptoms check up regularly. Increase of hernia surgery is recommended for infants and children to prevent incarceration. Emergent or immediate, surgery is necessary for incarcerated or strangulated hernias. Hernia surgery is also called herniorrhaphy. The two main types of surgery for hernias are:

- Open hernia repair
- Laparoscopic hernia repair

Complication

Inguinal hernias can cause the following complications:

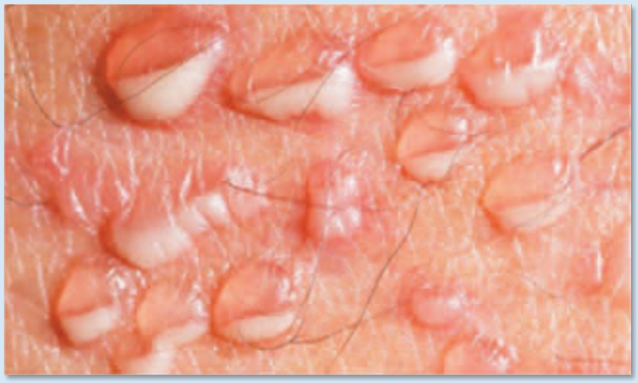
Incarceration: An incarcerated hernia happens when part of the fat or small intestine from inside the abdomen becomes stuck in the groin or scrotum and cannot go back into the abdomen.

Strangulation: When an incarcerated hernia is not treated, the blood supply to the small intestine may become obstructed, causing "strangulation" of the small intestine. This lack of blood supply is an emergency situation and can cause the section of the intestine to die.

References: *1. National Institute of Diabetes and Digestive and Kidney Diseases, May 2014*
2. Am. Fam. Physician, 2013;87(12):844-848
3. Dow University Health Sciences
4. www.mayoclinic.org

"Half-Half" blisters

A 66 year old man presented with an acute pustular eruption. On examination, an annular rash with multiple blisters was seen on the patient's trunk; the blisters contained both clear and yellow fluid. He had no associated cutaneous or systemic symptoms, and there was no facial or mucosal involvement. Examination of a biopsy sample taken from a representative blister revealed a subcorneal vesicle and other features characteristic of a subcorneal pustular dermatosis. Direct immunofluorescent staining showed IgA deposition in the superficial epidermis. Subcorneal pustular dermatosis is a chronic pustular dermatosis that frequently affects the flexures of the trunk and limbs. The lesions typically have the appearance of "half-half" blisters, with half of each blister containing pus and half containing clear fluid. The blisters eventually coalesce, giving rise to annular and serpiginous arrangements. It is usually idiopathic, but in a subgroup of patients IgA deposition can be seen with the use of direct immunofluorescent and it is thought that this phenomenon represents an overlap with IgA pemphigus. An association with IgA monoclonal gammopathy and myeloma has been reported.

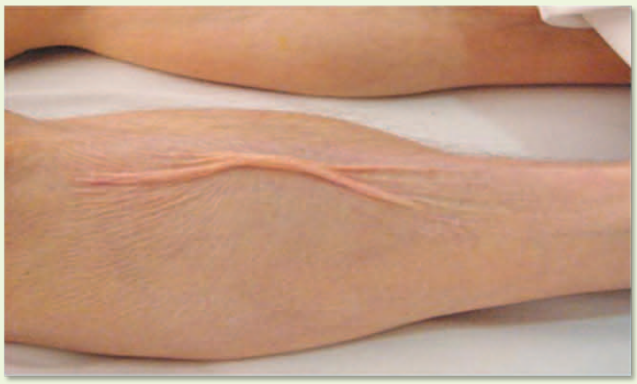


In this patient there was no evidence of an associated dyscrasia. Once the diagnosis is made, the treatment of choice is dapsone, but in this patient hemolytic anemia developed after its introduction. Although eruptions of this type do not always respond to glucocorticoids, there was a good therapeutic response in our patient.

Reference: *N. Eng. J. Med. May 17, 2012; 366;20:e31*

Decreased skin turgor

A 61 year old man with a vasoactive intestinal polypeptide secreting neuroendocrine tumor and diarrheal fluid losses exceeding 10 liters per day was transferred to the intensive care unit with severe volume depletion. On physical examination, the blood pressure was 54/30 mm of Hg and there was sinus tachycardia of 156 beats per minute. His total enteric fluid loss was 17 liters in the first 2 days after admission. The patient was also noted to have severely decreased skin turgor. Skin that was pinched over his anterior leg continued to be tented even after 10 minutes . In patients with a normal fluid volume, the skin would be expected to return to its usual contour almost immediately. Skin elasticity is known to decrease with age and there is no widely accepted threshold value distinguishing normal from abnormal skin turgor. Nevertheless, the skin turgor in this patient was dramatically decreased, clearly illustrating the loss of normal skin elasticity that can occur with severe volume depletion. The tenting



resolved with volume resuscitation. After the intravenous administration of octreotide, the secretory diarrhea ceased within 12 hours. A pancreatic tumor measuring 44 by 30 mm was subsequently identified on computed tomography and was resected. The patient had a full recovery.

Reference: *N. Eng. J. Med. January 27, 2011; 364;4:e6*

Keep a healthy life, so we won't be apart...

HEALTH MYTH

Please select the correct answer by (✓) against a, b, c, d, e of each question in the Business Reply Post Card and sent it through our colleagues or mail within 25 October 2016; this will ensure eligibility for the Raffle Draw and the lucky winners will get attractive prizes!

1 A 37 year old woman with immune thrombocytopenia failed to respond to corticosteroid therapy. Splenectomy was planned. What is the optimum time for pneumococcal vaccination?

a) 1 month after surgery

b) 1 month before surgery

c) 1 week after surgery

d) 1 week before surgery

e) Perioperatively

2 A 65 year old woman presented with a 12 hour history of the sudden onset of gait unsteadiness, vomiting and headache followed by increasing drowsiness. What is the most likely diagnosis?

a) Acute cerebellar haemorrhage

b) Acute subdural haemorrhage

c) Frontal subdural empyema

d) Herpes simplex encephalitis

e) Pituitary apoplexy

3 A 24 year old man complained of muscle fatigue on vigorous exercising. He said that his muscles were not getting enough oxygen. What is most likely to improve the delivery of oxygen to the tissues?

a) Decreased 2,3 diphosphoglycerate

b) Decreased temperature

c) Increased glucose

d) Increased PCO_2

e) Increased pH

4 A 46 year old man presented within 1 hour of ingesting 40 tablets of slow release theophylline. What is the most appropriate initial management?

a) Activated charcoal

b) Alkaline diuresis

c) Gastric lavage

d) Observation only

e) Whole bowel irrigation

5 The half life of a novel antiobesity drug exhibiting first order kinetics was calculated to be 4 hours. What percentage of the drug will be eliminated 20 hours after ingestion?

a) 75%

b) 80%

c) 90%

d) 97%

e) 100%

Published by
Medical Services Department, ACI Limited
Novo Tower, 9th Floor, 270 Tejgaon Industrial Area, Dhaka-1208

Designed by
Creative Communication Ltd., Road # 123, House # 18A
Gulshan 1, Dhaka 1212

ACI ADVANCING POSSIBILITIES