

# **Male Central obesity**

*Essay*

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in **General surgery**

*By*

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## Introduction

Obesity has reached epidemic proportion globally, and the situation is likely to deteriorate. The obesity epidemic also increases the risk for many disorders that are associated with high mortality and morbidity (*Ioannidis, 2008*).

The prevalence of obesity is increasing worldwide at an alarming rate. In 2006 the number of overweight and obese people in the world exceeded the number of malnourished (*Anand, 2006*).

A significant gender difference is noted in the prevalence of abdominal obesity. This difference is broadly explained by the biological differences between males and females in their fat metabolism (*Armabepola et al., 2006*).

Men are more likely to have abdominal or upper-body obesity (android obesity), whereas women are more likely to have a gluteofemoral or lower-body pattern of fat distribution (*gynoid obesity*) (*Ioannidis 2008*).

Body fat can be divided into 2 main compartments: subcutaneous and intra-abdominal. Fat may also accumulate at ectopic sites such as muscle, including the heart, pancreas, and liver) (*Abramof, 2008*).

Abdominal obesity refers not to subcutaneous fat beneath the skin of the abdomen, but to visceral fat, which surrounds the

omentum of the bowel. The amount of visceral fat inside the abdomen is directly proportional to the waist circumference (*Haslam, 2004*).

The visceral adipose tissue (VAT) accounts for approximately 15% of total body fat in the lean subject and includes the intraperitoneal (mesenteric and omental) fat, which drains into the portal circulation, and retroperitoneal fat, which drains into the systemic circulation (*Abramof, 2008*).

Unfortunately for men, the android form is dangerous (*Haslam, 2004*) because the visceral fat (omental and mesenteric fat), which constitutes more than 80% of the intra-abdominal fat, has unique metabolic characteristics and anatomical localization. Visceral adipose tissue has a higher turnover rate than other adipose tissue depots in both men and women. The visceral fat is drained via the portal vein to the liver, in contrast to peripheral fat depots, which are drained by the systemic circulation. The increased lipolytic activity of visceral fat, combined with its anatomical location, exposes the liver to higher concentrations of free fatty acids (FFAs) than any other organ. Free Fatty Acids decrease insulin clearance by the liver and increase hepatic glucose output and the secretion of very low-density lipoprotein (VLDL). These effects of (FFAs) on the liver cause peripheral hyperinsulinaemia, hyperglycaemia and an elevated (VLDL) level in patients with visceral obesity (*Marin et al., 1998*).

Excessive accumulation of (VAT) is an important and significant risk factor for coronary artery disease or diabetes. Metabolic studies in human subjects have shown that an increase of (VAT) is associated with insulin resistance, glucose intolerance, dyslipidemia, and hypertension. These reports and the unique anatomical location of (VAT), which (FFA) and various adipokines into the portal circulation for direct transport to the liver, have led to the concept that (VAT) is largely responsible for the metabolic abnormalities associated with abdominal obesity (*Furukawa et al., 2009*).

Visceral adiposity is increasingly recognized as the main determinant of metabolic risk among prediabetic or diabetic individuals. (*ma et al.2012*).

A major complication of central abdominal obesity is the increase intra abdominal pressure. The condition leads to venous stasis, gastroesophageal reflux disease, stress urinary incontinence, obesity hypoventilation syndrome, nephritic syndrome, incisional and inguinal hernia and elevated pleural pressure that can markedly increase pulmonary artery and pulmonary capillary wedge pressure (*Eric, 2006*).

Anthropometric measurements such as skin folds, and circumferences measured at the waist and hip are often used to assess body fat distribution (*García et al., 2010*).

**Waist circumference** is an important consideration in determination of the health risks related to obesity (*Wang et al., 2005*).

It is well documented that most determinants of, abdominal obesity was defined as  $WC \geq 90$  cm among males and  $WC \geq 80$  cm among females (*Armabepola et al., 2006*).

The key to therapy is to generate a negative energy balance by having the patient eat fewer calories than are expended so that the endogenous fat stores will be consumed for fuel. Approximately 75% to 85% of weight that is lost by dieting is composed of fat, and 15% to 25% is fat free mass (*Klein, 2001*).

The medical tools available for weight management include diet therapy, a regimen of physical activity, behavior modification and pharmacotherapy (*Fernández, et al., 2012*).

These measures may be beneficial in mild or moderate obesity; however they offer little help in morbidly obese patients (*Buchwald, 2005*).

The national institute of health has concluded that surgical therapy offers the best chance of successfully treating male abdominal obesity (*Jacobs & Robinson, 2007*).

Operations that are used to treat abdominal obesity are subjects of ongoing research & are being refined (*Jacobs & Robinson, 2007*). It improves abnormal lipid levels, controls

hypertension and reduces the risk of diabetes (*Greenway et al., 2002, Schauer et al., 2003 and Sugerman et al., 2003*).

Surgical procedures in management of morbid obesity fall into three broad categories: malabsorptive, gastric restrictive operations and operations with both malabsorptive and restrictive procedures. The available options include: Gastric bypass, vertical banded gastroplasty, silastic ring gastroplasty, stoma adjustable silicon gastric banding, biliopancreatic diversion and others. (*Buchwald, 2005*).

## **Aim of the Work**

The aim of the study is to review the different causes of male central obesity, as well as discussing the management and the possible treatment options for each kind of male central obesity.

## **Anatomy of the Male Abdomen**

### **Anatomy of the anterior abdominal wall**

The abdominal wall is a complex musculoaponeurotic structure that is attached to the ribs superiorly, the bones of the bony pelvis inferiorly and the vertebral column posteriorly.

The abdominal wall is developed from the lateral plate of the intraembryonic mesoderm, becomes segmented into proliferating somites and forms four folds (*cephalic, caudal, two lateral folds*). All of these segments and folds coalesce in the middle line at the umbilicus. So, the abdominal wall is derived embryonically in a segmental manner, which in turn is reflected in its blood supply and innervation (*Grevious et al., 2006*).

The major source of structural integrity and strength of the abdominal wall is provided by the musculofascial layer (*main paired abdominal muscles and their respected aponeuroses*). The integrity of the abdominal wall is essential in protecting the visceral structures, stabilizing the trunk and in aiding trunk movement and posture (*Townsend, 2004*).

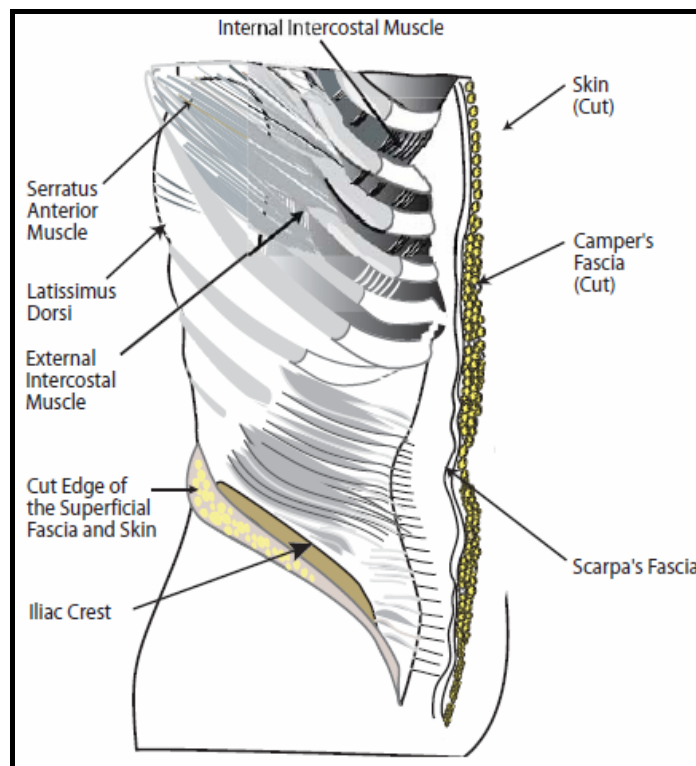
The surgical anatomy of skin, fascia, muscle, arterial and nerve supply should be taken into consideration while performing abdominal contour surgeries (*Kevin et al., 2001*).



## Structure of anterior abdominal wall

### Layers of the Abdominal Wall

The anterolateral abdominal wall consists, from the outside in, of the skin, superficial fascia, external and internal abdominal oblique, transverse abdominis and associated aponeuroses, rectus abdominis and pyramidalis, as well as the transversalis fascia (*Orhan, 2005*).



**Figure (1):** Layers of the anterior abdominal wall skin (*Orhan, 2005*).

**Skin:**

The skin is of average thickness, and loosely attaches to the underlying tissue. It exhibits certain surface markings such as the umbilicus, and McBurney's point. The umbilicus, a midline fibrous cicatrix covered by a folded area of skin, is an important anatomical landmark in the anterior abdomen that marks the original attachment of the fetal umbilical cord. (*Abuhamad et al., 2000*).

**1- Fascia of the anterior abdominal wall:**

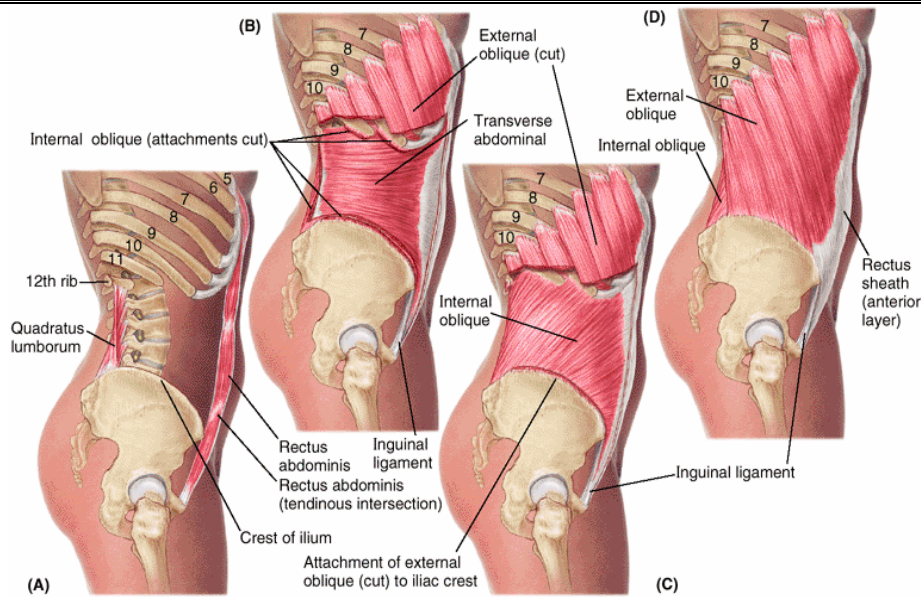
The superficial fascia consists of one layer above the umbilicus but below the umbilicus, it is divided into two layers: a superficial fatty layer (*Camper's fascia*) and a deep membranous layer (*Scarpa's fascia*). Camper's fascia is continuous with the superficial fat over the rest of the body and may be extremely thick (*3 inches or more*) in obese patients (*Cunningham et al., 2004*).

The deep fascia (Scarpa's fascia) is a thin, tough layer that surrounds and is adherent to the underlying abdominal muscles. Each abdominal muscle has aponeurotic component that contributes to the deep fascia. Scarpa's fascia is thin and fades out laterally and above, where it becomes continuous with the superficial fascia of the back and thorax respectively. Inferiorly, the membranous layer passes on to the front of the thigh, where it fuses with the deep fascia one finger breadth below the inguinal ligament (*Fong et al., 2004*).

## **2- Muscles of the anterior abdominal wall:**

The muscles of the anterior abdominal wall consist of five paired muscles: Three flat muscles (*The external oblique, internal oblique and the transverses abdominis*) and two vertical muscles (*The rectus abdominis and pyramidalis muscles*). The three-layered structure combined with extensive aponeuroses, works to protect the abdominal viscera and increase abdominal pressure facilitating defecation, micturation and parturition. Fusion of the fascial layers of these muscles forms three distinct fascial lines: The linea Alba and two semilunar lines. The linea alba is formed by the fusion of both rectus sheaths at the midline, while the semilunar lines are formed by the union of the internal oblique, transversus abdominis and external oblique as they join the rectus sheath (*Grevious et al.,2006*).

As the aponeuroses of the three sheets pass forward, they enclose the rectus abdominis to form the rectus sheath. The lower part of the rectus sheath might contain a small muscle called the pyramidalis (*Snell, 2004*).



**Figure (2):** Muscles of the anterolateral abdominal wall (*from Snell, 2004*).

**a) External oblique muscle:**

The external oblique muscle (*paired right and left*) is the largest and the thickest of the flat abdominal wall muscles. It is a broad thin muscular sheet that arises from the outer surface of the lower eight ribs and fans out to be inserted into the xiphoid process, linea alba, the pubic tubercle and the anterior half of the iliac crest. Most of fibers are inserted by means of a broad aponeurosis which is relatively thin superiorly and very strong inferiorly. Note that the most posterior fibers passing down to the iliac crest form a posterior free border. A triangular shaped defect in the external oblique aponeurosis lies immediately above and medial to the pubic tubercle. This is known as the superficial inguinal ring. The spermatic cord (*or round ligament of the uterus*) passes through this opening and carries the

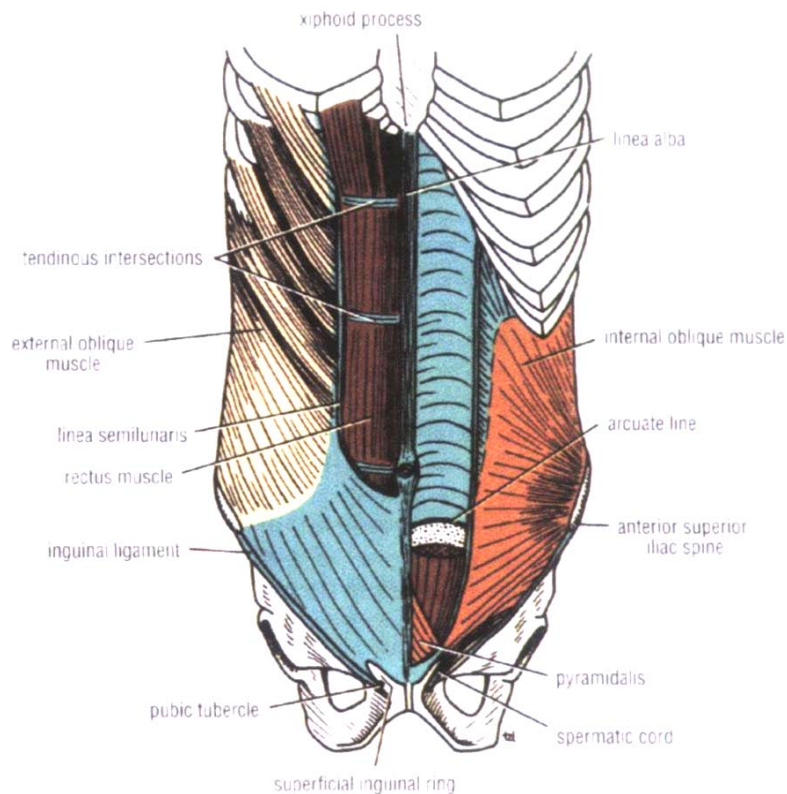
external spermatic fascia (*or the external covering of the round ligament of the uterus*) through the margins of the ring (**Stanley, 1995**).

**b) Internal oblique muscle:**

The internal oblique muscle is also a broad, thin muscular sheet that lies deep to the external oblique muscle, most of its fibers run at right angles to those of the external oblique. It arises from the lumbar fascia, the anterior two thirds of the iliac crest and the lateral two thirds of the inguinal ligament. The muscle fibers radiate as they pass upward and forward. The muscle is inserted into the lower border of the lower three ribs and their costal cartilages, the xiphoid process, the linea Alba and the symphysis pubis (**Moore et al., 1999**).

**c) Transversus abdominis muscle:**

The transversus muscle is a thin sheet of muscle that lies deep to the internal oblique and its fibers run horizontally forward. It arises from the deep surface of lower six costal cartilages (*Interdigitating with the diaphragm*), the lumbar fascia, the anterior two thirds of the iliac crest and the lateral third of the inguinal ligament. It is inserted into the xiphoid process, the linea alba and the symphysis pubis. The lowest tendinous fibers join similar fibers from the internal oblique to form the conjoint tendon, which is fixed to the pubic crest and the pectineal line (**Moore et al., 1999**).



**Figure (3):** Anterior view of rectus abdominis muscle and rectus sheath. Left: The anterior wall of the rectus sheath has been partly removed, showing the rectus muscle with its tendinous intersections. Right: The posterior wall of the rectus sheath is shown. The edge of the arcuate line is shown at the level of the anterior iliac spine (*from Snell, 2004*).

### c) Rectus abdominis muscle:

The rectus abdominis is a long strap muscle that extends along the whole length of the anterior abdominal wall. It is broader above and lies close to the midline being separated from its fellow by the linea Alba. The rectus abdominis muscle arises by two heads from the front of the symphysis pubis and the pubic crest. It is inserted into the 5th, 6th and 7th costal cartilages and the xiphoid process. When it contracts, its lateral margin forms a

curved ridge that can be palpated and often seen and is termed the linea semilunaris. This extends from the tip of the 9th costal cartilage to the pubic tubercle. It is divided into distinct segments by three transverse tendinous intersections, one at the level of the xiphoid process, one at the level of the umbilicus and one halfway between these two. These intersections are strongly attached to the anterior wall of the rectus sheath. The rectus abdominis is enclosed between aponeuroses of the external oblique, internal oblique and transversus abdominis, which form the rectus sheath (*Snell, 2004*).

**d) Pyramidalis muscle:**

The pyramidalis muscle is a small triangular muscle located in front of the lower part of the rectus abdominis that is absent in about 20% of population. If present, it arises by its base from the anterior surface of the pubis and is inserted into the linea alba (*Snell, 2004*).

**Action of the abdominal muscles**

Anatomically, there is a natural propensity to lower abdominal bulging because of the absence of the posterior rectus fascia below the arcuate line of Douglas and a genetic tendency toward fat accumulation in this region (*Matarasso and Belsley, 2005*).