Abdominal hypertension and venous insufficiency of lower limbs: is there a relationship?

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Domenico Parmeggiani, Marco Apperti, Vincenzo De Pascale, Nicola Avenia**, Pasquale Ambrosino, Imma Madonna, Roberto Peltrini, Sarah Apperti, Adelmo Gubitosi*, Umberto Parmeggiani*

Department of Gerontology, Geriatrics and Metabolic Disease, Second University of Naples, Naples, Italy
**Department of Anesthesiological, Surgical and Emergency Sciences, Second University of Naples, Naples, Italy*
***Department of General Surgery, University of Terni, Perugia, Italy*

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OBJECTIVE: *The purpose of our study is to understand the compression, by subcutaneous fat in obese patients, who present a chronic increase in abdominal pressure, and on the sapheno-femoral cross. Such increase would result in a chronic venous insufficiency (CVI) of lower limbs and, if possible, it will be quantified also according to the posture.*
MATERIAL AND METHODS: *We studied two different groups of patients with CVI: obese and non-obese. The severity of CVI was classified with the CEAP classification or by the standards of recent classifications. Abdominal pressure, by means of Kron's method, and anterior-sagittal diameter were measured in some of the non-obese patients (4 males and 4 females) and in all the obese patients. The diameter of the femoral vein of both groups of patients in supine, sitting and standing positions was also measured.*
RESULTS: We *found a higher incidence of CEAP 5-6 classes in the obese group than in the non-obese one; we also saw a higher incidence of classes 1-2 in the non-obese group than in the obese one. Moreover, Kron's method showed a significant difference in abdominal pressure in relation to the BMI and the increase in the femoral vein diameter appears to be related to the weight and to the position.*
DISCUSSION: It *was clear that the clinical manifestations of CVI are more severe in obese than in non-obese patients. Obesity in our study clearly acts with a continuous compressive action on the veins of the abdomen and thereby on the femoral vein, further contributing to a retrograde flow. So we tried to quantify an entity that until now could only be assumed.*

KEY WORDS: Abdominal Compartment Syndrome, Obesity complication,. Venous insufficiency of lower limbs

Introduction

Organs and organ systems are located in semirigid or rigid structures with pressure balances within, which are essential for the maintenance of the anatomic function and integrity. Life itself can be rapidly, irremediably and definitively extinguished by a sudden and uncontrolled rise in intracranial pressure (cerebral haemorrhage) or endothoracic pressure (hypertensive pneumothorax). There is clear evidence of such eventualities which have been known since remote past. From the 1960's on vascular surgeons and orthopaedists have described the compartment syndrome of the limbs, characterized by underfascial edema with possible evolution towards ischaemia and soft tissue necrosis, suggesting and successfully performing fasciotomy.
Only in the last decades has the clinicians' attention been drawn to the problems related to hypertensive conditions

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Correspondence to: Domenico Parmeggiani, MD,Via A. Falcone 290/A, 80128 Naples, Italy (E-mail: ground25@libero.it, d_parmeggiani@yahoo.it)

in the abdominal area. These problems are complex in the physiopathological field and insidious in the symptomatological one. This accounts for the delay in our knowledge of the subject, although some specific experimentations and clinical observations about that date back even to the 19th and the early 20th century.
Through a bibliographic research on PubMed 498 articles on this subject have been detected from 1966 to the present, 463 of which, equal to 92.9%, have been published in the last 10 years and as many as 135 (27.1%) in the last 24 months.
In spite of the growing interest, many doubts persist about the real incidence of the syndrome, which in literature varies from 6.1 to 39.4%, and about the therapeutic route that still today does not seem to be fully codified.
Today there is much discussion about the abdominal compartment syndrome and its possible implications; their real importance is not clear yet but they may prove to be of great theoretical and practical significance. The scientific community's interest is so great that the World Society of Abdominal Compartment Syndrome was founded in 2004 and whose mission is "to promote research, foster education and improve the survival of patients with IAH (intrabdominal hypertension)/ACS (abdominal compartment syndrome) by bringing together physicians, nurses and others from a variety of clinical discipline so to share informations on effective management strategies for reducing the significant of morbidity and mortality of IAH/ACS". In order to facilitate the achievement of the objectives a website (www.wsacs.org) has been created.
An increase in intra-abdominal hypertension (IAH) can have very serious effects. It occurs acutely when the content of the abdomen expands or the wall shrinks. IAH is different from abdominal compartment syndrome (ACS) which shows the advanced or the final result of an extreme abdominal hypertension state. Favouring factors are pregnancy, obesity and cirrhosis of the liver. Rare are the cases in which the syndrome is caused by pathologies with chronic evolution (bulky retroperitoneal or pelvic peritoneal neoplasms).
In 1996 Burch proposed the following classification comparing the abdominal hypertension grade (indirect measurement via the urinary bladder) with the severity of the clinical pathophysiological state.

TABLE I - *Burch classification of IAH*

Grade	cmH_2O	mmHg
Grade I	10 - 15	7 - 11
Grade II	15 - 25	11 - 18
Grade III	25 - 35	18 - 26
Grade IV	> 35	> 26

Obese patients surely have a chronic increase in abdominal pressure. Such chronic increase can cause pressure alterations also in the vessels of the abdominal area or in the vessels related to this area (vessels of the lower limbs). The correlation between obesity, hence abdominal hypertension, and venous insufficiency of lower limbs needs verifying.
Few studies have been made to understand the mechanisms through which whether or not obesity causes venous insufficiency in lower limbs (IVC), although many researchers believe there is a relationship between obesity and IVC.
Epidemiological studies, such as Basel Study[1] and Jerusalem Community Report[2], show a relationship between obesity and IVC in females but not in males, whereas Stanhope[3], Hirai[4] and Malhotra[5] have not found any. For a Danish epidemiological study[6], instead, obesity is a risk factor in both sexes. Framingham Study[7], instead, has demonstrated a higher incidence in women, who are often more obese than men.
It is still doubtful, therefore, whether or not there is a relationship between obesity and IVC and whether obesity plays a causal role or whether it simply worsens the IVC clinical picture.
Intra-abdominal pressure is surely higher in obese people than in non-obese ones[7,8]. Sobolewski[9] has shown that the increases in intra-abdominal pressure occurring during laparoscopy are sufficient to prevent the venous return in the lower limbs. This is why we think that the same mechanism can happen in obese subjects causing an action on the sapheno-femoral cross. The panniculus adiposus can have the same compression effect on the pelvic veins as the inflatable dilatation. It is likely that this phenomenon has a different effect according to the posture, standing or supine position.
The purpose of this study will be to understand this compression mechanism and, if possible, to quantify it also according to the posture.

Material and Methods

We have studied two different groups of patients with IVC: obese and non-obese. Both groups were studied through anamnesis: IVC family history (positive or negative), previous surgical interventions or deep venous thrombosis.
Clinically the severity of CVI was classified in the majority of the cases with the CEAP classification or by the recent standards of classification (CVI)[10,11]: (0) asymptomatic, (1) light venous insufficiency with prominent varicose veins, (2) moderate venous insufficiency with edema or skin pigmentation, (3) severe venous insufficiency with chronic or active ulcers.
Also BMI was measured in all patients.
Abdominal pressure, by means of Kron's method, and anterior-sagittal diameter were measured in some of the

non-obese patients (4 males and 4 females) and in all the obese patients. The technique used for monitoring the abdominal pressure was the measuring of vesical pressure. The bladder is emptied using a three-way Foley catheter and 100 ml of sterile physiological saline solution are instilled. The Foley catheter is closed with a clamp, the needle is connected to the tube of the water barometer and the height of the water column is measured. The pubic symphysis is considered as zero reference point, with the patient in a strictly horizontal dorsal decubitus position.
We wanted to measure this pressure also in sitting and standing positions but it was difficult to find the zero reference point so we tried to mime these positions by elevating the head from the bed of 30 and 45 degrees. Also the diameter of the femoral vein of both groups of patients in supine, sitting and standing positions was measured.

Results

The patients recruited in the study were 80 in total: 45 non-obese (21 males and 24 females) and 35 obese (20 males and 15 females). The mean BMI of the obese was 38; of the non-obese (calculated for 8 patients, 4 males and 4 females) it was 26.

Table II

	Non Obese Patients	Obese Patients
Males	21	20
Females	24	15
Positive family history of IVC	53%	55%
Previous surgery	25%	22%
Positive DVT	13%	19%
BMI	26	38

Table III

CEAP CLASS	Non Obese	Obese
2	48%	25%
3-4	32%	38%
5-6	20%	39%

Table IV

	Non Obese (8)	Obese
BMI	26	38
SAD (cm)	18	28
IAP (cm H_20)	3	16

Table V

	Non Obese AP (cm H_20)	Obese AP (cm H_20)
Supine Position	3	16
AT 30 degrees	7	21
AT 45 degrees	11	29

Table VI

	Non Obese FV diam (mm)	Obese FV diam (mm)
Supine	6.7	8.1
Sitting	8.6	10.7
Standing	10.2	13.3

There was no difference in the anamnesis as to family history, positive DVT, or surgery (Table II).
Instead, a higher CEAP 5-6 incidence in the obese group (35%) than in the non-obese one (20%) was found; also, a higher incidence of classes 1-2 in the group of the non-obese than in the obese one was observed (Table III).
The abdominal P, by means of Kron's method, and the anterior-sagittal diameter were then measured in both groups (all the obese patients and the 8 non-obese ones) with a clear difference in the results as shown in Table IV, also in relation to the BMI.
The measurement was also performed by elevating the head from the bed of 30 and 45 degrees with a significant increase in AP as shown in Table V.
Finally we measured the diameter of the femoral vein of both groups in supine, sitting and standing positions with an increase in the diameter appearing to be related to the weight and the position (Table VI).

Discussion

The Abdominal Compartment Syndrome (ACS) is defined as an increased abdominal hypertension (IAH), which causes, in a confined anatomical space, a decreased blood flow, leading to ischaemia and may lead to permanent impairment of function with clinical signs of multiorganic dysfunction[12,13]. The degree of the abdominal hypetension depends not only on the size of the abdomen, but also on the density of the material in it.[12].It has been classified from the World Society of the Abdominal Compartment Syndrome (WSACS) as primary, secondary and recurrent [13].
In patients with an increased intra-abdominal pressure, over 20-25 mmHg, an ACS triggers with altered functions in some organs, arriving at Multiple Organ Dysfuncion Syndrome (MODS). The intestine normally covers 58% of the abdominal volume but when there is ileus distention, intestinal pneumatosis develops which

Table VII

	Non Obese AP (cm H_2O)	Non Obese FV diam (mm)	OBESE AP (cm H_2O)	OBESE FV diam (mm)
Supine Position	3	6,7	16	8.1
30 degrees - sitting	7	8.6	21	10.7
45 degrees -standing	11	10.2	29	13.3

can occupy up to 90% of the entire cavity. At this moment, Gastro-intestinal failure can appear, which is a specific independent risk factor of mortality[14].

From the results of our study we had the impression that the severity of IVC and its complications are more frequent in the obese patient rather than in the non-obese one.

The first epidemiological studies to show this are the ones on the Caucasian race of Danish, Swiss, Israeli and German communities[1,2,10]. Also the New Zealand community seems to have the same epidemiological characteristics[15]

As for obesity as a risk factor for IVC, we think that a distinction should be made between the possible effects of obesity on the determinism of the pathology and the effects of obesity on the severity of the reflux and on the complications. Bonn study on obesity comes to the conclusion that obesity is not a risk factor for IVC but it is for edema and hyperpigmentation in IVC[16]

In our study it was clear that the clinical manifestations of IVC are more severe in obese than in non-obese individuals. Instead, the anamnesis, the family history, the DVT frequency and the surgical interventions do not seem to be different in the two groups. This might be related to the different pathogenetic mechanism of IVC between obese and non-obese patients dealt with by some authors. Padberg has published a work in which in 62% of the cases the echo-colour doppler of the lower limbs of obese patients was within normal limits[17]

Obesity causes a relative obstruction of the lower limb veins as it happens for patients suffering from ascites.

Although several of these patients also have a cardiac insufficiency, the combined effect of the request added to the reduced venous return cause a central arteriovenous damage.

A lymphatic occlusion may be another mechanism of damage. Recurrent episodes of cellulitis may cause cicatricial damage to lymphatic canals.

What may be even more relevant is the damage of the lymphatic flow caused by adverse intra-abdominal hemodynamic pressures: combined effect of lympho-venous hypertension[18]

The most significant evidence obtained with our work is surely the clear relationship between BMI and abdominal pressure and anterior sagittal diameter. There is a clear increase in the intra abdominal pressure and the anterior sagittal diameter because of the increase in BMI. This is the result we were more interested in when we started our study. This result was still more evident with the measurement of the abdominal pressure by elevating the head from the bed of 30 and 45 degrees: the pressure increased in proportion and this makes us think that our abdomen works as a real hydraulic pump.

We tried to measure the intra-abdominal pressure in standing position but it was difficult to find the zero point, so the results were not trustworthy.

The clear difference of abdominal pressure in the two groups of patients is undoubtedly at the basis of the different clinical characteristics of the groups.

In Lambert's work on obese patients IVC was diagnosed only if the chronic oedema was associated with pigmented dermatitis, ulcers or asymmetric oedema with a positive DVT anamnesis. The increase in AP was statistically significant only for patients with BPCO and IVC[19].

Other authors, instead, think that the effects of obesity on the venous system are related to the chronic increase in AP. They have shown such increase using Kron's technique (mean AP 19cm H_20) to corroborate this theory. Also, as an indirect proof, they describe how the patients who had a significant loss of weight with a gastric bypass had a reduction of IVC symptoms (oedema and ulcers).

Another datum we want to analyze is also the measurement of the femoral vein diameter. This value increases with the increase in abdominal pressure and is related to the position.

This summary table clearly shows how an increase in the FV diameter corresponds to an increase in AP, almost as a proof of what we have said and have read in literature.

In our study obesity clearly acts with a continuous compressive action on the veins of the abdomen and thereby on the femoral vein. We have tried in this way to quantify an entity that could only be assumed.

In one of his studies Erdogmus[20] says IVC occurs more frequently in patients with BPCO, suggesting that the pathogenesis can be really caused by an increase in AP. Arfvidsson[21], instead, has invasively shown the increase in iliofemoral venous pressure in obese patients leaving it to others how to understand the mechanisms that can contribute to the growth of lower limbs IVC.

We can conclude that a higher intra-abdominal pressure in obese patients could force more rapidly the venous reflux even extending, as we have shown, the veins, fur-

ther contributing to a retrograde flow. A minor extension of the veins in obese patients could also be the result of that, as shown by other AAs[22].

Riassunto

Le problematiche correlate a condizioni ipertensive instauratesi nel distretto addominale sono state approfondite solo negli ultimi decenni, fatta eccezione per alcuni riferimenti risalenti al XIX e XX secolo. Inoltre, circa il 93% degli articoli relativi all'argomento, dal 1966 ad oggi, sono stati pubblicati solo negli ultimi 10 anni. La Sindrome Compartimentale Addominale, la cui incidenza e trattamento non appaiono ancora compiutamente codificati, esprime l'esito avanzato o finale di una condizione di ipertensione addominale estrema. La classificazione di Burch correla la gravità clinica con il grado di ipertensione. Dal momento che i pazienti obesi mostrano un aumento della pressione addominale, essa può riflettersi anche sui vasi, sia quelli contenuti in addome sia quelli degli arti inferiori, determinando insufficienza venosa cronica (IVC). Alcuni studi testimoniano la correlazione tra obesità ed IVC, in altri essa è meno evidente. Resta inoltre da capire se l'obesità abbia un ruolo causale o rappresenti una condizione di peggioramento del quadro clinico.

Lo studio si propone di valutare e quantizzare in base alla postura del paziente l'azione compressiva esercitata dal tessuto adiposo a livello safeno-femorale, in ragione del fatto che anche la dilatazione pneumatica in corso di laparoscopia è sufficiente a ridurre il ritorno venoso degli arti inferiori. A tal proposito, sono stati studiati due gruppi di pazienti: obesi (35 pazienti) e non obesi (45 pazienti), entrambi con IVC opportunamente classificata, per i quali è stato calcolato il BMI; a tutti quelli del primo gruppo e solo ad otto del secondo, è stata misurata la pressione addominale (mediante la metodica di Kron), il diametro antero-sagittale e quello della vena femorale variando la posizione: eretta, supina, seduta. Il dato certamente più interessante che scaturisce dal nostro lavoro è la netta correlazione tra BMI, pressione addominale e diametro antero-sagitale: con l'aumento del BMI vi è una netta crescita della pressione addominale e del diametro antero-sagitale. Inoltre il diametro della vena femorale incrementa con l'aumento della pressione addominale e risente della postura. L'obesità in questo nostro studio agisce chiaramente con un'azione compressiva continua sulle vene dell'addome e di conseguenza sulla vena femorale. Abbiamo cercato di quantizzare in questo modo una entità che fino ad ora si poteva solamente supporre.

Questo studio suggerisce che severità e frequenza della IVC sono maggiori nei pazienti obesi a causa di un'ostruzione relativa delle vene degli arti inferiori e, verosimilmente, anche per una compromissione del flusso linfatico; tali alterazioni emodinamiche sono da riferire ad un aumento della pressione addominale.

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