

SYNDROMES OF INTRACAVITARY AND INTRAORGAN HYPERTENSION IN SURGICAL PRACTICE

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ABSTRACT

The significance of intracavitary, intraorgan, hypertension syndrome identification as a novel uniform (model) pattern of a pathological process is substantiated in the article based on currently available literature sources and clinical practice experience. A conformity analysis of the considered pathological syndromes and model pathological processes criteria is presented with reference to data on etiology, pathogenesis, anatomical and some ontogenetic suppositions and stipulations. Criteria for the studied pathological conditions differentiation in clinical practice are proposed.

UDC CLASSIFICATION & KEYWORDS

■ 616.12-008.331.1 ■ Uniform Pathological Process ■ Intracavitary ■ Intraorgan ■ Intratissular ■ Hypertension ■ Syndrome ■ Excessive Pressure ■ Perfusion Pressure ■

INTRODUCTION

Pathological conditions caused by excessive pressure, i.e. hypertension (compression) in the cavities and organs of the human body, has increasingly been an object of intense study. Considerable progress has been made in the study of intraabdominal hypertension, compartment syndrome in cases of limb fracture, intracranial hypertension.

The implication of intraabdominal hypertension (IAH) was substantially considered on the discovery of an association between hypertension and multiorgan insufficiency development, as well as an exclusively high mortality rate in this group of patients (24, 25, 28, 32, 39,42).

According to the conducted trials, about 30% patients with acute abdominal pathology in a critical condition present with IAH and approximately 4-5% of intensive care patients reveal abdominal compartment syndrome (ACS) to be the main cause of death. A number of Russian researchers have demonstrated the contribution of intracavitary and intraorgan hypertension to the development of abdominal (296 patients) and thoracic (316 patients) compartment syndromes, Mallory-Weiss (336 patients) syndrome, complicated diverticular colon disease (524 patients), acute intestinal obstruction (213 patients) [12-19]. Mortality rate with intracranial pressure >20 mm Hg makes up to 45%, with ≥ 60 mm Hg – 100% (J.L. Miller, 1983, quotation from V.Ye. Grushevski [2]).

Acute cholecystitis is known to develop concurrently with gall bladder neck obturation, most commonly by calculi, the outcome mainly depending on bile outflow restoration and intravesical pressure reduction. If bile hypertension is not corrected and resolved in a timely manner, the consequent irreversible changes of the gallbladder wall, i.e. vascular thrombosis and destruction [3, 20,30], occur.

Compression syndrome and hypertension have proved to be significant factors in heart trauma cases: cardiac tamponade [up to 11.1%] has been shown one of the common mortality causes (up to 50% [22]). Pericardial tamponade occurs with 300-700 ml blood trapped in the pericardial cavity.

In shin-bone fracture, compartment syndrome develops in 35% cases, high intertissular pressure in the osseo-fascial compartments resulting in circulatory failure and tissue revitalization threat [1, 9, 36, 37, 44]. With ischemia progression, irreversible (necrotic) changes in the muscular tissue, vessels, nerves [6, 7, 10, 11] take place. Intratissular (subfascial) pressure of 30 mm Hg has been found an indication for a fasciotomy procedure [4]; an increase in the pressure value >50 mm Hg leads to a complete cessation of tissue perfusion, with marked ischemic features presenting in the tissues in 4-8 hours and irreversible changes in the muscles and nerves in 12 hours [38, 43, 44].

Patients and methods

We have examined 1955 patients by means of integrated investigation methods (clinical, laboratory, biochemical, endoscopic, including laparoscopy, thoracoscopy, ultrasonography, computer tomography and magnetic resonance tomography and others) and measurements; monitoring of intracavitary, intraorgan, intratissular pressure. Pressure measurements were taken by "Spiegelberg" company unit (Germany), "Тритон" (Russia) – in the abdominal, thoracic and cranial cavities, organs. The subfascial pressure on the shin-bone was measured by means of "Stryker" monitoring device (USA). Intraabdominal pressure was evaluated by the pressure in the urinary bladder or in the stomach due to lack of their use possibility as applied to the pressure level in v.femoralis. Transducers for intracranial pressure measurements were placed after trepanation in craniocerebral injury.

Out of the total number, 296 patients had severe abdominal pathologies (generalized peritonitis, infected pancreatic necrosis, acute disturbance of mesenteric circulation), 70 patients had craniocerebral injuries, 316 patients had pulmonary diseases (cystic and bullous lung disease, emphysematous lesions), 336 patients had Mallory-Weiss syndrome, 524 patients had complicated diverticular colon disease, 213 patients had acute intestinal obstruction, 200 patients had severe forms of compound shin-bone fractures.

Statistical analysis was conducted by means of a standard Software package (Statistica for Windows).

Results

An analysis of intracranial pressure indices in 70 critical state patients with combined craniocerebral trauma and multiorgan insufficiency has demonstrated pathophysiological interrelations between intracranial, cerebral perfusion types of pressure and their response to the level of average arterial pressure. The obtained absolute indices of intracranial, cerebral perfusion types of pressure can ultimately be applied as substantial monitoring and prognosis parameters to this group of patients.

Observations made in our clinic (200 patients) of patients with severe shin-bone fractures also confirm the significance of subfascial pressure values, integrated diagnostic procedures and choice of treatment strategy for the given localization traumas. An analysis of the literature, as well as our own data, has enabled us to consider hypertension syndromes in the cavities, organs and organ tissues, respectively, as a uniform (model) pathological process characterized by a number of common etiological, pathophysiological and other factors.

Model pathological processes are defined by the following features [21]:

- I. They are caused by various etiological factors (dialectical emphasis on their representative features).
- II. They are not dependent on the localization and the type of the organism.
- III. They are specified as being similar in development mechanism (pathogenetic uniformity).
- IV. They differ in manifestation similarity (symptomatic uniformity).

According to the specified features, there can be distinguished a variety of hypertension syndrome types.

Intraabdominal hypertension syndrome can be caused by various reasons, the latter being divided into the following groups [32]:

- I. Postoperative: haemorrhage, intraoperative abdominal wall suture, peritonitis, pneumoperitoneum during laparoscopy and after it, dynamic intestinal obstruction.
- II. Posttraumatic: intraabdominal hemorrhage and retroperitoneal hematomas, edema of internal organs due to closed abdominal injuries, pneumoperitoneum in rupture of a hollow organ, pelvic bones fracture, abdominal wall deformations due to burns.
- III. Complications to the underlying disease: sepsis, peritonitis, cirrhosis with concurrent ascites, intestinal obstruction, abdominal aortic aneurysm, renal insufficiency with peritoneal dialysis, tumors.
- IV. Predisposing factors: systemic inflammatory response syndrome, acidosis ($\text{pH} < 7.2$), coagulopathy, massive hemotransfusions, hypothermia.

Among the observed 296 patients with severe acute abdominal pathology, intraabdominal hypertension syndrome was most commonly caused by acute obstructive intestinal obstruction (25%), gastroduodenal ulceration-induced complications (15.6%), abdominal cavity organs injuries (13.2%), acute mesenteric circulation disorder (12%), acute commissural intestinal obstruction (12.1%), peritonitis (11.8%), other pathologies (9.7%). Among 316 patients with signs of intrathoracic hypertension undergoing surgical treatment, the rate of nonspecific lung diseases was higher, with 112 out of their number presenting with chronic obstructive pulmonary disease. In the vast majority of cases, cystic and bullous changes were identified. Intracranial hypertension syndrome ($n=68$) was most frequently caused by acute cerebral blood circulation disturbances (hemorrhagic stroke) with intracranial hematomas formation, aneurysm rupture, traumatic intracerebral hematomas, brain tumors, etc.

Pronounced manifestations of hypertension syndrome were detected with hematoma volume of >30 -40 ml. According to the recently available publications, intracranial hypertension can be due to severe craniocerebral injury, intracranial and subarachnoid hemorrhage, hydrocephaly, brain edema, in massive strokes, hypoxic brain damage, CNS infections, encephalopathy in hepatic insufficiency [29,35,40].

Motor coordination disorder, pathologic segmentation of certain large intestine regions (more frequently sigmoid colon) associated with autonomic innervations disorders and/or low-residue diet are responsible for intrainestinal hypertension in diverticular disease.

Mallory-Weiss syndrome develops as a result of an abrupt elevation of intraventricular, intraesophageal pressure (intraventricular, intraesophageal hypertension syndrome) most commonly occurring after vomiting, physical exertion and being accompanied by gastric and esophageal wall layers rupture [34].

Most commonly, causation between compound fractures, anaerobic infection (nonclostridial myonecrosis, necrotic fasciitis), compression syndrome, positional compression syndrome, acute thrombosis of lower limb veins, aneurysm rupture, etc. and compartment syndrome development is identified.

Thus, intracavitary, intraorgan hypertension syndromes can be caused by various etiological factors.

By the aforementioned, the syndromes are not localization bound, i.e. intracavitary hypertension syndromes can develop in anatomically confined body cavities causing pathophysiological changes in other cavities and subsequently leading to organ dysfunction and multiorgan insufficiency by reference to systemic disorders.

In excessive hypertension impact implementation, intraorgan hypertension initially manifests in the form of organ function disturbances and, moreover, henceforth results in general disorders of the whole body.

Virtually comparable pathological processes are described in other kinds of living organisms. Thus, gastrointestinal obstruction and obturation in livestock animals are predominantly referred to alimentary factors, physico-chemical, mechanical impacts, infectious and parasitic diseases [23]. Pathogenesis is characterized by disorders of food motility,

vermicular peristalsis disturbances (spasm, paresis, paralysis), disorders of blood and lymphatic circulation, pathological reactions of the central and peripheral nervous system. In waterfowl without ingluvies, acute extension (ectasia) of the hollow organ along the whole circular length. Estasia of a tubular organ is preceded by compensatory hypertrophy of its muscles and ultimately proceeds into atrophy, the organ declining into a state of functional insufficiency.

In acute equine stomach extension, stomach wall rupture in the area of larger curvature, in some cases the diaphragm rupture followed by a prolapse of stomach portion, liver part, intestinal loops into the thoracic cavity are observed.

Similar pathogenetic mechanisms underlie the pathological processes development of intracavitary and intraorgan hypertension (ICH and IOH) syndromes.

Increased hypertension pathological effects implementation in bodily cavities is related to pathological processes inside the organs confined in the cavities. Therefore, pathological effects implementation can be described by a phase-by-phase scheme:

- I. Intracavitary hypertension → intraorgan hypertension → intratissular hypertension;
- II. Intraorgan hypertension → intratissular hypertension.

The most significant component of intratissular hypertension is the impact of excessive pressure on blood and lymphatic vessels, as well as nerve structures. In routine cases of hypertension pathological syndromes, cells of organs and tissues (intracellular hypertension) are considered as impact endpoints.

In intracavitary hypertension development, its pathological effect is not limited to a single cavity.

Similar processes simultaneously take place in the organs of the adjacent and other cavities. Thus, in intraabdominal hypertension syndrome, a pronounced extension and displacement of the diaphragm into the thoracic cavity and respiratory disturbances have proved to be the initial signs of intraabdominal hypertension. Subsequently, intracranial hypertension manifestations follow. Thus, in intracavitary hypertension syndrome, several organs of the considered cavity, as well as those of the neighbouring and other cavities are found to be involved in the pathological process, which results in an ordinary outcome, i.e. multiorgan insufficiency involvement. In intraorgan hypertension, pathological process is initially organ-confined (organ dysfunction), and through complications involvement as a result of excessive pressure implementation, other organs and organ systems become involved into the pathological process.

Synchronously, the consequences and complications of intraorgan hypertension syndrome may show to be extremely severe and within a short term cause a critical condition in a patient. Specifically, in Mallory-Weiss syndrome, profuse hemorrhage due to ruptures of the mucous and submucous membranes with hemorrhagic shock; pneumothorax, pneumomediastinum, mediastinitis in complete tear involving all layers (stage III of the syndrome); peritonitis in perforation of esophagus abdominal region frequently occur.

Hypertension syndrome is a pathological process with its physical pressure parameters being of utmost significance in essence description. Hypertension is defined as excessive pressure inside a cavity, or an organ, the thresholds of which exceed the possibilities of physiological compensation of the produced pathological effects. It should be noted however that quantitative measures of excessive pressure (degree of hypertension) may vary but must be authentic and inevitably comparable to the mean measures of venous and arterial pressure. At the onset, ICH and IOH syndromes present with measurements approximation in relation to intracavitary, intraorgan pressure and their counterpart values of venous circulation in the organ, while in the full-fledged stage – comparable to the values of arterial blood flow in the organ. The extreme manifestation of hypertension syndrome (stages III-IV of ICH and IOH syndromes), i.e. compartment syndrome, can be identified in cases of critical level values of perfusion arterial pressure inside an organ, or a cavity, counteracting the microvasculature circulation.

Based on the data from currently available sources and those obtained from personal practice, excessive (pathological) pressure inside the abdominal cavity has proved to be >10 mmHg, abdominal compartment syndrome to be identified with intraabdominal pressure exceeding 35mmHg.

Discussion

As designated before, one of the main pathogenetic components of organ dysfunction is microcirculation disturbance, the efficiency of which is determined by adequate perfusion conditions. Perfusion pressure of the abdominal cavity (the so called abdominal perfusion pressure - APP), calculated as a difference between mean arterial pressure (MAP) and intracavitary (intraabdominal) pressure (IAP) ($APP = MAP - IAP$), is to be ≥ 50 -60 mmHg [26, 27, 33] for appropriate blood supply preservation in the abdominal organs. With abdominal perfusion pressure <40 mmHg, blood circulation stops at the level of microcirculation.

The normal level of intracranial pressure in a human depends on age and body position, amounting to 7-15 mmHg in adults in recumbent posture, the average of 10 mmHg in the upright position; it should not exceed 15 mmHg [29]. Intracranial pressure >25 mm Hg is an indication for aggressive intensive therapy, the condition being considered critical [40]. Intracranial hypertension syndrome is accompanied by a significant reduction of cerebral perfusion pressure (CPP), the measurements of <70-80 mmHg being critical. According to W.J. Gray, M.J. Rosner [31], a decline of cerebral perfusion pressure to <80 mmHg is referred to a 20% mortality rate increase with every 10mmHg decrease. Numerous prospective trials, with the leading purpose of therapeutic actions being maintaining CPP >70 mmHg, confirmed the effectiveness of this approach to damage outcome improvement.

The mean measure of intratissular pressure in an intact shin makes up 8-10 mmHg [1, 5, 41]. With a two-fold increase in intratissular pressure (ITP), hypertension syndrome symptoms manifest, requiring dynamic monitoring, while fasciotomy is recommended in cases of $ITP > 30$ mmHg [4].

An increase in ITP to >50 mmHg results in an incomplete tissue perfusion ceasation, and in 4-8 hours marked signs of ischemia are produced, in 12 hours irreversible damage to the muscle and nerve supply occur [38, 43, 44].

The physical basis for such pathological processes as hypertension syndromes - ICH and IOH syndromes, intratissular hypertension – is considered a disparity between the content and the cavity volumes. The intensity and severity of this pathological type can be assessed by a conventional coefficient (k) defined as a ratio of content volume (V_c) and cavity volume (V_n): $k = V_c/V_n$. Therefore, hypertension degree and pathological process severity rise with an increase in k value.

It should also be taken into consideration that k value is dependent on a number of predisposing and producing factors. The ratio V_c/V_n for different cavities, organs, tissues varies and is contingent on the anatomical, functional characteristics and peculiarities, physiological condition prior to pathological processes development. In our point of view, the following factors are to be taken into consideration:

Phylogenetic specifications-dependent biomechanical properties (criteria) of cavities, organs, tissues. The distinct structures differ in the durability degree, protective membrane and tissue distensibility. Conventionally, they can be of three safeguard types (degrees):

- Those devoid of V_n volume changing possibility in vivo (for instance, cranial cavity in an adult, intramedullary canal), to a greater degree of confined space, some of them containing a complementary protective structure, e.g. additional firm medullary membrane in the cranial cavity. For this cavity types, V_n is a constant value ($V_n = \text{const}$) and k may vary within a small range. Therefore, the effects of compensatory mechanisms are dramatically limited in hypertension syndrome development; clinical cases even at the onset of manifestations are urgent presenting with intense signs by minor changes of V_c volume and insignificant factors of excessive pressure within a short term.
- Those with limited possibilities of V_n volume changing. Thoracic cavity, pericardial cavity, parenchymatous organs within the capsules, membranes, osteofascial spaces of limb segments can be referred to this type. Thus, V_n volume change of the thoracic cavity occurs due to the diaphragm shift and extension, as well as the intercostals muscles. In such cavity type, k coefficient is higher than that of the first type (a) and lower than the third type (c).
- Those with major possibilities of V_n volume changing. Abdominal cavity, hollow organs can be referred to this type, the k values significantly varying ($k > 2.0$).

Thus, the values of k are contingent of:

- extensibility, elasticity of the cavity, organ, tissue membranes (framework);
- value of intracavitary, intraorgan, intratissular pressures;
- phylogenetically eligible V_c/V_n ratio, i.e. originally stipulated reserve capacity (volume), determined by the formula: $V^A = V_n - V_c$. Undoubtedly, V^A measures for "b" and "c" type cavities, organs are markedly higher, providing compensatory mechanisms in pathological conditions within a relatively extended period of time;
- surgical corrective procedures (decompressive laparotomy, hemicraniectomy), pharmacological intervention (muscle relaxation, neural blockage by epidural anesthesia, edema decongestion, etc.) can substantially effect k coefficient values.

From the above data it should be noted that the level of intracavitary, intraorgan, intratissular pressure has been noted to be an amply steady value, amounting to about 10 mmHg, and thus can be considered constant for the human body (P_c). In a pathological condition, the clinical manifestations of excessive pressure are identified by an approximate 1.5-fold increase of P_c and, likewise, pronounced signs of a pathological process with a full-scale clinical picture are produced with at least a 2-fold rise in physiological pressure. The latter statement is primarily contingent of the types, cavity structures, organs, tissues, as well as hypertension syndrome-causing etiological factors. Thus, with cavities devoid of V_n volume changing possibility ("a" type), a 2-fold P_c pressure rise causes a severe intracavitary hypertension syndrome, yet with cavities of major V_n volume changing possibilities ("c" type), compartment syndrome develops by at least a 3-fold increase in P_c pressure. It is also necessary to emphasize that the level of intracavitary, intratissular hypertension exceeding 25 mmHg has been observed to be the threshold for the human body and, furthermore, reaching this value, pronounced signs of pathological hypertension syndrome, or compartment syndrome, are produced.

Therefore, intracavitary, intraorgan, intratissular hypertension syndromes present with multiple-factor etiology, similar pathogenesis mechanisms, organ dysfunction or multiple organ insufficiency and are capable of developing inside human and animal cavities, organs. The statistically significant criteria of these pathological syndromes are excessive intracavitary, intraorgan, intratissular pressure; a decrease in cavitory and organ perfusion pressure with ischemia development, severe disturbance and even microcirculation cessation; organ dysfunction or multiple organ insufficiency.

Intracavitary, intraorgan and intratissular hypertension syndromes can be defined as a uniform (model) pathological process induced by factors of traumatic, infection contagious and vascular nature, moreover, conditioned by a disparity between the content volume and cavity (organ) volume capacity, excessive pressure development inside of them and, consequently, a pronounced disturbance of intraorgan, intratissular perfusion, being accompanied by organ dysfunction or multiple organ insufficiency.

The significance of intracavitary, intraorgan, hypertension syndrome identification as a novel uniform (model) pattern of a pathological process is substantiated in the in the article based on currently available literature sources and clinical practice experience. A conformity analysis of the considered pathological syndromes and model pathological processes criteria is presented with reference to data on etiology, pathogenesis, anatomical and some ontogenetic suppositions and stipulations. Criteria for the studied pathological conditions differentiation in clinical practice are proposed.

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