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Abdominal Aortic Aneurism Inherited Real Risk: Quantum-Biophysical-Semeiotic Patho-Physiology, Symptomatology, Diagnosis and Therapy.

Abstract.
In the paper, for the first time the Author describes AAA. Inherited Real Risk, characterised by VV. Microcirculatory Remodelling, typical of all Inherited Real Risks, including CVD one. Notoriously, newborn-pathological, type I, subtype b), Endoarteriolar Blocking Devices slow down the flow-motion along VV. in CVD Inherited Real Risk, but especially in AAA. one. AAA. Inherited Real Risk quantum-biophysical-semeiotic patho-physiology, symptomatology, diagnosis, differential diagnosis and its Quantum Therapy are fully illustrated.

Introduction.

Notoriously aortosclerosis is a pathological thickening of aorta wall, while an abdominal aortic aneurysm is an enlarged area in the lower part of the aorta, occurring slowly, mainly after some decades from individual's birth.
Because from the aorta originate a flurry of other arteries, a diseased aorta, worse of all, the ruptured abdominal aortic aneurysm, represents a life-threatening condition (1-3).
Despite of both aorta wall thickening and the size and rate at which abdominal aortic aneurysm is growing, its initial stage is the till now un-known Aortosclerosis and AAA Inherited Real Risk, I am describing for the first time later on. Consequently, the best treatment of all is the same as that at the base of every IRR therapy. Thus, in the future Medicine physicians will not await the overt diseased aorta for beginning therapy, neither they shall need any emergency surgery, as now happens.
As a matter of fact, once an abdominal aortic aneurysm is found, casually in the majority of cases, doctors will closely monitor it so that surgery can be planned if it's necessary. Emergency surgery for a ruptured abdominal aortic aneurysm can be risky.
In next future, thanks to QBS Primary Prevention, based on Aortosclerosis and AAA Inherited Real Risk, physicians will are able to avoid such a procedure, that shall not be necessary at all, thanks to AAA Primary Prevention.

Abdominal Aortic Aneurism Traditional Causes.

Most aortic aneurysms occur in the part of aorta that's in the abdomen (1-3). Although the exact cause of abdominal aortic aneurysms is unknown, a number of factors may play an important role, as tobacco use. Cigarette smoking and other forms of tobacco use appear to increase the risk of aortic aneurysms. In addition to the damaging effects that smoking causes directly to the arteries, smoking contributes to the build-up of fatty plaques in arteries [involved by CVD Inherited Real Risk (IRR) !] and high blood pressure. Smoking can also cause an aneurysm to grow faster by further damaging the aorta. Atherosclerosis occurs when fat and other substances build up on the lining of a blood vessel, increasing the risk of an aneurysm [according to the old Outside-In Theories of Atherosclerosis: See later on!].
Aneurysms can develop anywhere along the aorta. When they occur in the upper part of the aorta, they are called thoracic aortic aneurysms. More commonly, aneurysms form in the lower part of the aorta and are called abdominal aortic aneurysms. These aneurysms may also be referred to as AAA.

Abdominal Aortic Aneurism Traditional Risk Factors

There is now a general agreement among Authors, who do not know Quantum Biophysical Semeiotics, that abdominal aortic aneurysm risk factors are: Age, since abdominal aortic aneurysms occur most often in people age 65 and older [because the misery of traditional semeiotics!]; Tobacco use, that is a strong risk factor for the development of an abdominal aortic aneurysm [but at the condition that AAA. IRR is present locally!]; the longer you've smoked or chewed tobacco, the greater your risk [see above-cited remarks!]; Atherosclerosis, which increases the risk of an aneurysm; Male sex, because men develop abdominal aortic aneurysms much more often than women do; Family history, since people who have a [mother!] family history of abdominal aortic aneurysm are at increased risk of having the condition. In fact, people who have a family history of aneurysms tend to develop aneurysms at a younger age and are at higher risk of rupture.

Abdominal Aortic Aneurism Traditional Physical Semeiotic Symptoms.

Abdominal aortic aneurysms mainly grow slowly and usually without symptoms, making them difficult to detect them (4). Some aneurysms will never rupture. Many start small, and stay small, although many expand over time. Others expand quickly. Predicting how fast an abdominal aortic aneurysm may enlarge is difficult. As an abdominal aortic aneurysm enlarges, some people may notice a pulsating feeling near the navel; deep, constant pain in your abdomen or on its side of your abdomen and finally back pain.

Anyone age 60 and older who has risk factors for developing an abdominal aortic aneurysm, such as smoking or a [mother!] family history of abdominal aortic aneurysm, should consider regular screening for the condition. Because being male and smoking significantly increase the risk of abdominal aortic aneurysm, men ages 65 to 75 who have ever smoked cigarettes should have a one-time screening for abdominal aortic aneurysm using abdominal ultrasound.

If there is a [mother!] family history of abdominal aortic aneurysm, doctor may recommend an ultrasound exam to “screen” for the condition. There are no specific screening recommendations for women [in absence of AAA. IRR, of course!] .

Abdominal Aortic Aneurism Complications.

Aortic dissections are the main complications of abdominal aortic aneurysm. A ruptured aortic aneurysm can lead to life-threatening internal bleeding. In general, the larger the aneurysm, the greater the risk of rupture. Signs and symptoms that an aortic aneurysm is dangerous are sudden, intense and persistent abdominal or back pain, that radiating down to legs; sweatiness, clamminess, dizziness, nausea, vomiting, low blood pressure, fast pulse, loss of consciousness, shortness of breath.

Another complication of aortic aneurysms is the risk of blood clots. The aortic aneurysm can bring about small blood clots. If one of them breaks through from the inside wall of an aneurysm and blocks a blood vessel elsewhere in the patient’s body, it can cause pain or block the blood flow to the legs, toes, kidneys or abdominal organs.

AAA. Pathogenesis.

At this point, I emphasise an interesting pathogenetic aspect of AAA. Notoriously, the intercostal arteries supply Vasa Vasorum to thoracic aorta. On the contrary, in the descending aorta, vasa vasorum cease to supply the arterial walls with oxygenated blood at the level of the renal arteries (9). As a consequence, below this point, the aorta is dependent on diffusion for its metabolic needs,

and is necessarily markedly thinner. This leads to an increased likelihood of aneurysm at this location, especially in the presence of atherosclerotic plaques. Other species, such as dogs, do have vasa vasorum below their renal vasculature, and aneurysms at this location are substantially less likely (9). Cerebral blood vessels are devoid of vasa vasorum; however, these vessels have rete vasorum, which have similar function to vasa vasorum (10).
In rare cases, abdominal aortic aneurysm may be caused by an infection or inflammation, autoimmune in origin, that weakens a section of the aortic wall.
Fascinating studies have shown that increases in proteolytic activity are associated with abdominal aortic aneurysms. Herron GS. et al. have studied samples of the dilated aortic wall, taken during corrective surgery for AAAs, in terms of the number, type, and tissue location of connective tissue proteinases and their inhibitors (11).
Five distinct caseinolytic serine proteinases and six gelatinolytic metalloproteinases were resolved by molecular weight by use of sodium dodecyl sulfate-substrate gel electrophoresis (11). Isoforms of the Mr 92,000 neutrophil gelatinase were identified by immunoprecipitation of biosynthetically labeled organ culture media. About 50% of the total radiolabeled protein secreted by AAA organ cultures was identified as the Mr 30,000 glycoprotein, tissue inhibitor of metalloproteinase (TIMP), by immunoprecipitation. Both TIMP and gelatinase were localized to the vasa vasorum by immunoperoxidase staining (11). However, interstitial collagenase could not be detected by any method. These results suggest the involvement of the vasa vasorum in the maintenance and possibly the genesis of AAAs.
However, till now, the precise pathophysiology of AAA., a common disease among elderly individuals, remains unknown (12). In AAA, an intraluminal thrombus prevents luminal perfusion of oxygen, allowing only the adventitial vasa vasorum (VV) to deliver oxygen and nutrients to the aortic wall. Tanaka H. et al. recently examined changes in the adventitial VV wall in AAA. to clarify the histopathological mechanisms underlying AAA (12).
They found marked intimal hyperplasia of the adventitial VV in the AAA. sac, corroborating my previous researches, QBS Microcirculatory Theory is based on (13-21).
At this points, I already emphasised that marked intimal hyperplasia of the adventitial VV. occurs after years from the birth, while the first pathological alteration, i.e., the microcirculatory remodelling of VV. is heritable by mother (13-21).
Further, immunohistological studies revealed proliferation of smooth muscle cells, which caused luminal stenosis of the VV., but this occurs later. Tanaka H. et al. also found decreased HemeB signals in the aortic wall of the sac, as compared with those in the aortic wall of the neck region in AAA. The stenosis of adventitial VV. in the AAA sac and the malperfusion of the aortic wall observed in the present study are new aspects of AAA pathology that are expected to enhance our understanding of this disease (12).

Abdominal Aortic Aneurism Quantum-Biophysical-Semeiotic Inherited Real Risk.

Because of a flurry number of heritable predispositions to human disorders, including CVD IRR, i.e., Atherosclerosis Constitution-Dependent, Inherited Real Risk (19), I have before conjectured as Hypothesis 0, the existence of a analogous related heritable risk, as those I have discovered and described in previous articles (13, 15 19, 24, 25), it is not surprising the existence of the Inherited Real Risk of AAA, beside that of aortosclerosis.
To recognise such a paramount heritable risk, the physicians have to know the Psychokinetic Diagnostic (14). Moreover, to understand in details what follows, doctors have to read previous papers on the Inherited Real Risks (IRR), as well as Quantum Biophysical Semeiotic Microcirculatory Theory of Atherosclerosis(15-24).

In daily practice, doctor has to follow this procedure, described here below:

1) first of all, to ascertain if Antognetti's Sign is positive, and than localise precisely the IRR; in case of overt AAA. this sign is particularly intense: ≥ 3 cm. or more;
2) to assess VV. density, in the site of IRR;
3) to evaluate properly all parameter values of artery-gastric aspecific reflex, recognizing the level of tissue acidosis;
4) to recognise local compliance impairment;
5) to observe the presence of inflammation sign, recognising its autoimmune nature, by stimulating the precise site of the risk by means of nail pressure.

In health, Antognetti's Sign is negative (18): *intense* digital pressure, applied upon every point of a large artery, e.g., the femoral artery at the groin, *simultaneously* does not bring about Gastric Aspecific Reflex, that appears physiologically after 16 sec., as in Artery Preconditioning. (15-23). On the contrary, in individuals involved by Inherited Real Risk of CVD, including aortosclerosis and AAA, since their beginning as IRR, under identical experimental condition, described above, *simultaneously* appears such a reflex, whose intensity parallels the seriousness of underlying predisposition to CVD.
Interestingly, only in presence of autoimmune inflammation, after gastric aspecific reflex it follows tonic Gastric Contraction, indicating intense tissue acidosis.
Soon thereafter, by means of numerous quantum biophysical semeiotic signs (19), doctors can exactly localise the site of CVD IRR, for instance in a precise tract of abdominal aorta.

2) The above-mentioned procedure is unavoidable in bedside assessing the local VV. density, with a small digital pressure (19). At this point, once recognised the predisposition to Atherosclerosis, physicians must apply *possibly* small digital pressure upon every tract of the abdominal aorta, or "by thought", according to Psychokinetic Diagnostic (14): a small digital pressure is applied on every tract of the thoracic and abdominal aorta. When digital pressure upon an artery is small, only VV. are stimulated.
In health, for 3 sec. appears a small first upper ureteral reflex, followed by a second reflex of the same intensity, that appears to be less than 0,5 cm., paralleling VV. density, as clinical evidence demonstrates. I think that probably VV. firstly disappear under small digital pressure, but then re-appear due to the activation of venous-arteriolar reflex, brought about by hypoxia.
This evaluation is really important to assess VV. density, that is lowered only in the first year in the IRR of ATS! Initiating from the second year, VV. density augments in relation to the CVD IRR seriousness: ureteral Reflex intensity is about 1 cm.
On the contrary, in individuals involved by AAA Inherited Real Risk, from birth, VV. density is significantly raised so that ureteral reflex results more than 1 cm. (19). However, the function of newborn-pathological microvessels is impaired, so that there is locally worsening acidosis, as described as follows.

3) Artery-Gastric aspecific Reflex gives doctor a lot of precious information: Latency Time (NN = 8 sec.) remains physiological as far as the reflex duration is lesser than 6 sec. (normal duration: > 3 sec. < 4 sec.), informing on tissue oxygen level, i.e., tissue acidosis. Interestingly, only when reflex duration is more than 6 cm., latency time begins to lower. In addition, when Latency Time decreases below 6 sec., it is followed by Tonic Gastric Contraction, indicating a severe tissue acidosis, i.e., lowered istangic pH.
From the above remarks, at the bedside doctors can assess the acidity level at the arterial site, wherein IRR is located.
The Second Latency Time, also termed as Differential Latency Time, before the second Reflex, lasts > 3 sec. > 4 sec., paralleling fractal Dimension of the local Vasomotion.

Finally, when the Oxygenation Recovery Time is normal (= reflex disappearing soon after digital pressure is quickly interrupted; O_2RT: NN < 2 sec.), compliance impairment, and inflammation, including autoimmune inflammation, are absent, as I refer to, later on (15-23):
Interestingly, the bedside evaluation of parameter values of Aorta-Gastric Aspecific Reflex inform on Latency Time (NN = 8 sec., informing on endocellular energy level), Duration (NN > 3 sec. < 4 sec.), related to Microcirculatory Functional Reserve, and thus tissue pH; the Second Latency Time, i.e., the interval between the end of the first microvessel oscillation and the following one, (NN > 3 sec. < 4 sec.), indicating the fractal Dimension of microvessel dynamics; Oxygen Recovery Tyme (NN < 2 sec.) parameter value really important to evaluate the flow-motion (19).

4) Aortic Compliance Evaluation.

To bedside assess aorta compliance, which plays an important role in recognizing the exact site of AAA. Inherited Real Risk, doctor enlarges "by thought" a precise point of the aorta, assessing the intensity of Gastric aspecific Reflex (26, 27)
In health, such a gastric aspecific reflex shows an intensity of about 2 cm. Diverse stress tests, Insulin Secretion Acute Pick Test as (28-31), Adiponectin test (27), Valsalva's Manoeuvre, increase significantly the reflex intensity.
On the contrary, in AAA. Inherited Real Risk, under above-mentioned experimental condition, the intensity of aorta-gastric aspecific reflex is initially less than 2 seconds and increases slightly during stress tests.

5) Inflammation and Autoimmune Inflammation Signs.

The pressure by means of a nail, applied upon an inflamed tissue, despite its seriousness, causes gastric aspecific reflex, after a latency time less than the normal 8 sec. Such a latency time is inversely related to the severity of underlying inflammation: 3-5 sec.
As above-referred, exclusively in autoimmune inflammation occurs the characteristic tonic Gastric Contraction, after the aspecific Gastric Reflex.
In health, from the individual's birth, under above-mentioned experimental condition, such a reflex appears after a Latency Time of $\geq$ 8 sec.
In presence of Aortosclerosis Inherited Real Risk, the latency time results $\geq$ 5 sec.
On the contrary, in presence of AAA. IRR the latency time is 3-4 sec.
From the above remarks it appears of extremely importance the role played by artery wall inflammation, autoimmune in nature, in the pathogenesis of AAA.

At this point, I illustrate briefly the differential diagnosis between aortosclerosis IRR and AAA. IRR, starting from the first days of life. In both cases, Antognetti's Sign is clear-cut positive, showing the existence of CVD Inherited Real Risk in the circulatory three. In a few words, in my long well-established experience, this sign proved to be reliable, useful, though it is not specific.
Interestingly, reflex intensity is generally about 0,5 cm., paralleling the risk seriousness of predisposition to CVD: in AAA IRR is characteristically about 1cm.
Finally, only in AAA. Inherited Real Risk, the sign of inflammation is positive and intense, showing a typical small latency time (3-4 sec.), from the birth and it is growing on in subsequent following months and years.

As in all other IRRs, the Quantum Therapy proved to be really efficacious in removing AAA. Inherited Real Risk (32, 33).

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