

Quantum Biophysical Semeiotic Bedside Diagnosis of Tako-Tsubo Cardiomyopathy.

The central Role played by CAEMH-Dependent GERD in precipitating
the transient cardiac Dysfunction.

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Introduction

Tako-Tsubo cardiomyopathy, described in Japan in 1991 (1), also known as transient apical ballooning syndrome (2), apical ballooning cardiomyopathy, stress-induced cardiomyopathy, heart-broken syndrome, and simply stress cardiomyopathy, is a type of non-ischemic cardiomyopathy in which there is a sudden temporary weakening of the myocardium.

According to recent data published on *Giornale Italiano di Cardiologia*, 2012, Vol. 13, n1 p 59-66 (www.giornaledicardiologia.it), 72% of patients with stress-induced cardiomyopathy show normal coronary artery.

Tako-Tsubo cardiomyopathy mimics acute coronary syndrome and it is characterized by reversible left ventricular apical ballooning in the absence of angiographically significant coronary artery stenosis. In Japanese, “tako-tsubo” means “fishing pot for trapping octopus,” and the left ventricle of a patient diagnosed with this condition resembles that shape during acute episodes.

Tako-Tsubo cardiomyopathy, which is transient and typically precipitated by intense emotional stress, so that it is also known as “stress cardiomyopathy” or “broken-heart syndrome.” (3-10)

In spite of the etiology of Tako Tsubo cardiomyopathy, overlooking Quantum Biophysical Semeiotics (11-21), it is not fully understood, so that several action mechanisms have been proposed.

Regarding the aim of this article we have to remember that the left anterior descending artery (LADA) supplies the anterior wall of the left ventricle in the majority of patients. If this artery also wraps around the apex of the heart, it may be responsible for blood supply to the apex and the inferior wall of the heart. This correlation between Tako-Tsubo and this type of LADA has been sufficiently enlightened.

Other researchers have shown that this anatomical variant is not common enough to explain Tako-Tsubo cardiomyopathy (1-10). This theory would also not explain documented variants where the midventricular walls or base of the heart does not contract (akinesis).

Another theory suggests Transient Vasospasm: some of the original researchers of Tako-Tsubo emphasise that multiple simultaneous spasms of coronary arteries could cause enough lowering of blood flow to cause transient stunning of the myocardium (1). Unfortunately, this theory cannot explain the heritable microcirculatory remodelling, based on Congenital Acidotic Enzyme-Metabolic Histangiopathy (CAE-MH) (11).

Furthermore, other researchers have shown that vasospasm is much less common than initially thought. It has also been noted that when there are vasospasms, even in multiple arteries, that they do not correlate with the areas of myocardium that are not contracting (5, 7).

According to the 'Quantum Biophysical Semeiotics' (QBS) theory, we are emphasising, Tako-Tsubo cardiomyopathy is based on inherited, CAE-MH dependent, Microvascular Dysfunction, characterized by microvascular remodelling at level of left apical cardiac region. In such a area, there is a dysfunction of the coronary arteries at the level where they are no longer visible by coronary angiography, i.e., in heart tissue-microcirculatory-unit (22-29).

We have observed a microcirculatory dysactivation in the left apex region, in the sense that both vasomotility and vasomotion shows less intensity and duration of diastole, than the normal one (at rest, NN = 6 sec.). Under these microcirculatory conditions, the coronary provides insufficient material-information energy exclusively to apex myocardial cells, as illustrated later on.

The aim of this article is to describe the QBS Syndrome, which allows physicians to bedside diagnose Tako-Tsubo Cardiomyopathy for the first time, even outside the acute episode and better understand its pathophysiology, since the etiology of Tako-Tsubo cardiomyopathy is till now not fully understood, though several mechanisms have been proposed.

Interestingly, all our cases are, involved by GERD with jatal hernia, a disorder dependent of Congenital Acidosis Enzyme-Metabolic Histangiopathy.

(http://www.semeioticabiofisica.it/semeioticabiofisica/Documenti/Eng/Ernia%20Jatale_eng.doc).

Methods and Cases.

To bedside recognise Tako-Tsubo cardiomyopathy by means of QBS Syndrome (Table 1), doctor has to be skilled in Quantum Biophysical Semeiotics (11- 21).

Importantly, patients involved by stress-dependent disorders, as Tako-Tsubo cardiomyopathy, are positive for Congenital Acidotic Enzyme-Metabolic Histangiopathy, a mitochondrial cytopathy, condition sine qua non of the most common and serious disorders, as I have demonstrated in former article (30-34).

First of all, soon thereafter recognizing CAEMH, doctor must assess some paramount parameter values: latency time of heart-gastric aspecific reflex, informing on local pH, tissue oxygenation, Microcirculatory Functional Reserve, more precisely evaluated with the aid of Clinical Microangiology, illustrate in www.semeioticabiofisica.it (34-36).

Secondly, with the aid of Psychokinetic Diagnostics (37-54), doctor evaluates apex ballooning and local vasomotility, vasomotion, and tissue oxygenation.

By thought, physician applies digital pressure upon well-limited area of the heart, particularly heart apex: if the pressure intensity is small, doctor can assess the microcirculation by means of upper and lower ureteral reflexes (23-43) (www.semeioticabiofisica.it, Microangiology).

When the pressure, brought about by physician's thought, is of mean intensity, heart-gastric aspecific reflex occurs, informing on tissue oxygenation, Microcirculatory Functional Reserve, fractal dimension of microvessel fluctuation, as well known (14-29).

Finally, the mean-intense digital pressure, caused by thought, applied upon LV apex region brings about significant "in toto" ureteral-, choledocical, caecal, and gastric aspecific reflex, indicating apex ballooning, which disappears soon thereafter Tako-Tsubo episode is over.

One of us has observed 6 cases, three females (one has dead) and three males, alive: age between 60 and 93 when the episodes occurred.

Quantum-Biophysical-Semeiotics Syndrome
To Bedside Diagnose Tako-Tsubo Cardiomyopathy

Congenital Acidotic Enzyme-Metabolic Histangiopathy (CAE-MH)

Neuro-Vegetative System Dysfunction

GERD with jatal Hernia

Caotino's Sign Negative in about 72% of cases,
but positive (0,5 cm .) 100% during Tako-Tsubo acute episode!

Inherited Microcirculatory Remodelling of Left Apex Tissue Microcirculatory Unit,
showing small Microcirculatory Dysactivation

NORMAL Coronary Microcirculation in 72% of cases, with the exception in APEX region

Apex ballooning and intense Mircrocirculatory Dysactivation during acute episode

Brain Sensor Activation during acute episode; increasing trigger points in Left Apex Region

Table 1

Results

According to the data referred on the *Giornale Italiano di Cardiologia*, 2012, Vol 13, n1 p 59-66 (www.giornaledicardiologia.it) in 72% of patients involved by Tako-Tsubo Cardiomyopathy, as in the cases recently visited according with QBS.

On the contrary, during the acute episode Quantum Biophysical Semeiotics allows doctor to recognised quickly all signs of QBS Syndrome, summarised in Table 1.

CAEMH is unavoidably present, since it represents the condition sine qua non of macro- and microvascular dysfunction.

Interestingly all six cases suffered from GERD, in presence of jatal hernia, before and during the Tako-Tsubo acute episode

Caotino's sign (11-15) becomes positive, showing a disorder in the heart microvessels, located in the apex region, where is present microcirculatory dysactivation, causing local pH lowering, reduced oxygenation, lowered fractal dimension of microvessel fluctuation.

Finally, mean intense digital pressure, brought about according to Psychokinetic Diagnostic, causes an intense Cystic Syndrome: "in toto" ureteral-, gastric aspecific-,caecal, choledocic and gall-bladder-reflex (11).

What is until now known about Tako Tsubo Syndrome.

A preponderant of the syndrome occurs in elderly and especially more frequent in postmenopausal females. The onset is consequent to acute emotional stress or an acute medical condition. ST-segment elevation or depression, or T-wave changes, even in individual healthy before the episode. A prolonged QT interval can be present and the mild increase in cardiac enzymes, alarming physicians and patient. Typical akinesis of the apical and distal anterior wall together with hypercontraction of the basal wall van be observed. The occasional presence of transient

intracavitary pressure gradients in some patients Complete resolution of the apical wall motion abnormality and the depressed LVSF as soon as the episode is over.

Until now, ignoring Quantum Biophysical Semeiotics, the exact pathogenesis of Tako Tsubo cardiomyopathy remained unclear, so that various mechanisms have been proposed (1-10).

Some Authors suggested coronary vasospasm as the pathogenic mechanism; however, induction of coronary vasospasm by acetylcholine or ergonovine has yielded mixed results. In some series, vasospasm in at least one epicardial coronary artery was present in most patients, whereas other colleagues found no coronary vasospasm in patients who underwent an acetylcholine challenge. Multivessel coronary spasm would be required to account for the apical wall motion abnormality seen in this syndrome. Similarly, the duration of wall motion abnormality in Tako Tsubo cardiomyopathy typically is longer than would be expected in conventional cases of coronary vasospasm.

Really, the possibility of myocardial injury due to microvascular spasm has also been suggested. Ako and coworkers (3-5), by the use of an intracoronary Doppler wire technique, demonstrated microcirculation impairments in instances of transient LV hypocontraction. Although this is an interesting explanation, several factors challenge its causative potential.

First, microscopic findings in some patients who had LV apical ballooning were different from those in patients who had myocardial ischemia. The most common pathologic finding in Tako Tsubo cardiomyopathy is not typically seen in patients with myocardial infarction. Second, in several cases, coronary angiography failed to reveal the slow-flow phenomenon, even in the presence of ST-segment elevation. Finally, impaired microcirculation during the acute phase is not direct evidence of causation, because microcirculatory impairment can result from a primary myocardial injury.

In our opinion, no method, other than Quantum Biophysical Semeiotics, is able to assess microcirculatory fluctuations, i.e., vasomotility and vasomotion, accurately evaluated, during and soon thereafter Tako Tsubo cardiomyopathy in individual under severe psychological stress.

Another putative mechanism is neurogenic stunned myocardium. Enhanced sympathetic activity appears to play a very important role in the pathophysiology of Tako Tsubo cardiomyopathy, but in subjects involved by CAEMH. Triggering factors, such as intense emotional stress, are frequently seen in patients with this syndrome.

Excessive levels of catecholamines have been observed in patients with Tako Tsubo cardiomyopathy. Catecholamines have been shown to induce myocardial damage, and excessive stimulation of cardiac adrenergic receptors has led to transient LV hypocontraction in animal models (55). There is a clinical evidence on strong relationship between stress-induced cardiomyopathy and increased plasma catecholamine levels, suggesting that exaggerated sympathetic activation may be important in the development of the cardiomyopathy (56).

There is an intriguing question surrounding Tako Tsubo cardiomyopathy, namely why the apical wall is affected but the base is spared. Several explanations have been proposed. The apex is structurally vulnerable because it does not have a 3-layered myocardial configuration, it has a limited elasticity reserve, it can easily become ischemic as a consequence of its relatively limited coronary circulation, and it is more responsive to adrenergic stimulation. All of these factors might make the apex more sensitive to the catecholamine-induced surge frequently observed in Tako Tsubo cardiomyopathy (57). Really, some Authors observed the interesting relation between the use of sympatomimetic drugs and the onset of acute episode of Tako Tsubo cardiomyopathy onset (62).

Quantum Biophysical Semeiotic Clinical Microangiology of Tako Tsubo Syndrome

Quantum Biophysical Semeiotic Clinical Microangiology has finally enlightened patho-physiology of Tako Tsubo cardiomyopathy. To understand completely it is unavoidable that doctor learn to

bedside analyze the different aspects of microcirculation in healthy and diseased subjects, at rest as well as under diverse stress test, we have describe in a previous paper (59).

Our data allows us to state that, even in healthy young, microcirculation of left apex region, but not of left, probably due to above-referred anatomical reasons, is significantly less efficient than that in a every other one, due to the local microcirculatory remodelling, characterized by the presence of type I, physiological, Endoarteriolar Blocking Devices, absent in every other myocardial area.

In health, at rest, heart–gastric aspecific reflex shows notoriously a latency time of 8 sec. and duration < 3 sec. > 4 sec. After heart preconditioning (15, 16), the latency time raises to 16 sec.

Interestingly, exclusively in left apex region, basal heart–gastric aspecific reflex is still 8 sec., but its duration is 4 sec., a slight pathological datum, revealing that apex Microcirculation Functional Reserve (MFR) is slightly compromised, due to the local microcirculatory remodelling, illustrated above, according to Angiobiopathy theory (11-19).

In addition, during heart preconditioning, latency time of left apex-gastric aspecific reflex arises physiologically almost to 16 sec., but duration appears raised from 4 to 6 sec. These value are related to the underlying disorder seriousness.

Finally, under stress tests (insulin, catecholamine, adiponectin tests, Valsalva's manouvre) latency time behaviour of left apex region microcirculation lasts among normal range values, but the duration results more prolonged 6-7 sec.. demonstrating a particular sensitivity and responsiveness of the microcirculation of such a region.

Interestingly, even away from acute episode of Tako Tsubo cardiomyopathy, the diastole of "peripheral heart" is slower than normal: dilation of small artery and arterioles, according to Hammersen, i.e., vasomotility, happens in 3 sec., ore more, *versus* normal 1-2 sec.

Quantum Biophysical Semeiotics and Clinical Microangiology highlight Tako Tsubo cardiomyopathy patho-physiology, thanks to the parameter values of apical microcirculation during acute episode, as well as immediately before and after.

We emphasise that CAEMH represents the *conditio sine qua non* of Tako Tsubo cardiomyopathy, as well as of all other CVD (11-17).

In addition, neuro-vegetative dystonia plays a central role in the patho-physiology of Tako Tsubo cardiomyopathy, suggesting that the sympathetic regulatory effect of microcirculatory wall dynamics is of primary importance, as demonstrates the experimental evidence by means of stress tests (33).

In fact, as referred above, Amariles P. et al. observed that Tako Tsubo cardiomyopathy is a syndrome of transient cardiac dysfunction precipitated by intense emotional or physical stress. Excessive sympathetic stimulation is believed to be central to the pathogenesis of this condition, thus drugs with sympathetic effect could precipitate TCM.

Regarding the relation between GERD and the Tako Tsubo syndrome acute episode, we emphasise the presence of LES dysfunction in all our 6 patients involved by jatal hernia. Interestingly, both disorders are based on CAEMH.

In addition, Quantum Biophysical Semeiotics have allowed one of us to demonstrate that the stimulation of jatal hernia trigger-points worsens coronary microcirculation (63).

Furthermore, in a fascinating paper (64) Elikowski et al. have described the interesting case of a woman, involved by GERD before the onset of ventricular arytmia and Tako Tsubo cardiomyopathy occurrence.

Among other case we remember that described by Mohammad Khalil, et al (65), regarding 8-years-old female with past medical history of gastroesophageal reflux disease (GERD), hypertension, hyperlipidaemia and diabetes presented with chest pain, radiated to left arm. All these disorders are CAEMH-dependent.

Caotino's sign negative at rest in our 6 patients with Tako Tsubo cardiomyopathy, away from the acute episode, shows the integrity of coronary artery (11-15, 60).

On the contrary, under stress tests, as well as during acute episodes, and immediately before and after, Caotino's Sign is positive due to tissue reduced oxygenation at apex level, and Brain Sensor Activation is increase by stimulating exclusively apical trigger-points (61).

As regards the tissue-microcirculatory-unit microcirculatory remodelling due to the presence of Endoarteriolar Blocking Devices, type I, physiological exclusively in left apex region (in all other walls of the heart these type I physiological DEB are absent in healthy subjects) physicians are invited to read former papers (1, 15-17, 22, 59)

Conclusion

Tako Tsubo cardiomyopathy, more frequent than generally admitted since physician ignore Quantum Biophysical Semeiotics and Clinical Microangiology, has important implications, because its clinical presentation mimics that of an acute coronary syndrome, causing both Psychological Jatrogenetic Terrorism, and hospital admittance, followed by numerous and expensive sophisticated investigations, as in the case recently observed. Increased awareness of Tako Tsubo cardiomyopathy will likely result in its being diagnosed more frequently, especially if doctor knows Quantum-Biophysical-Semeiotics, that has contributed to elucidate the specific pathophysiologic mechanisms responsible for this singular cardiomyopathy.

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