Quantum-Biophysical-Semeiotic bedside Diagnosis of Frontal-Temporal Dementia, starting from its Inherited Real Risk.

By Sergio Stagnaro

Frontal-temporal dementia is a form of dementia that occurs when the frontal-temporal lobes of the brain are damaged genetically through mother impaired mitochondria. Experts estimate that it is responsible for 10%-15% of dementia cases (1-17).

Frontal-temporal dementia, based on its Inherited Real Risk I have recently discovered, has its own constellation of symptoms and is separate from Alzheimer's disease, because in AD typically there is not cerebral physiological Microcirculatory Activation, associated, type I, under the Insulinemic Acute Pick Test (18-20).

In addition, but less important from the quantum biophysical semeiotic differential diagnosis point of view, one difference between frontal-temporal dementia (FTD) and Alzheimer's disease is that, on average, frontal lobe dementia firstly presents itself significantly earlier in life. FTD symptoms usually appear between 45 and 65 years of age, while the majority of Alzheimer's cases occur in those over 65.

The frontal lobes are responsible for helping inhibition and behavior regulation, so people with frontal lobe dementia will often exhibit strange or unusual behaviors and personality changes. In fact, personality changes and behavior problems are hallmarks of the disorder, whose synptoms appear 4-5 decades after birth, if physician ignores the Inherited Real Risk of FTD, present at birth, diagnosed with a stethoscope and removed by inexpensive therapy (1-15).

Personality changes due to FTD include: Impulsiveness, Apathy and indifference, socially inappropriate behavior

People with frontal lobe dementia may suddenly struggle with binge eating and gambling compulsions because of the impulsiveness associated with frontal lobe atrophy

Frontal-temporal dementia does not cause memory loss, but it can other cognitive and neurological problems similar to those caused by Alzheimer's disease or stroke.

These symptoms can include: Difficulty with speech and language; Inability to concentrate, Inability to pla; Using the wrong object for the wrong task, or at the wrong time; Movement and balance difficulties.

However, nowadays physicians can perform differential diagnosis at the bedside clearly an quickly by means of characteristic quantum biophysical semeiotic signs (20, 21).

Fronto-temporal dementia and parkinsonism (FTDP) is a major neurodegenerative syndrome, particularly for those with symptoms beginning before age 65. This variant of FTD is characterized by the association of PD signs, especially the data of prolactin test (= the breast massage causes a gastric aspecific reflex, more persistent of 7 sec., physiological value) and the impairment also of the posterior third of parietal lobe, typically undamaged in FTD. (See later on)

Although Inherited Real Risk is always present in FTD, a spectrum of degenerative disorders since ever notoriously present as sporadic or familial FTD.

Mutations in the gene encoding the microtubule associated protein tau (*MAPT*) on chromosome 17 have been found in many kindreds with familial FTDP. Several other kindreds with FTDP had been linked to chromosome 17, but they had ubiquitin-positive inclusions rather than tauopathy pathology, and no mutations in *MAPT*. This conundrum was solved over this past year with the identification of mutations in the gene encoding progranulin (*PGRN*), which is only 1.7 Mb centromeric to *MAPT* on chromosome 17. Authors have compared and contrasted the demographic, clinical, radiologic, neuropathologic, genetic, and pathophysiologic features in patients with FTDP linked to mutations in *MAPT* and *PGRN*, highlighting the many similarities but also a few important differences (16, 17).

There is a general agreement that no one test is able to **diagnose Frontal-temporal Dementia**. Instead physicians are able to use the balance of evidence to diagnose frontal-temporal dementia based on their best judgment. Because there is no foolproof test and diagnosis depends on the physician's knowledge, judgment, and observation of the patient, FTD is notoriously difficult to diagnose in its early stages, if physician ignore Quantum Biophysical Semeiotic. Although as the disease progresses, it becomes easier to definitely distinguish it between other disorders (15-18).

Behavioral changes are the most common early signs of the variant GRN-related frontotemporal dementia. These include marked changes in personality, judgment, and insight. It may become difficult for affected individuals to interact with others in a socially appropriate manner. Affected people may also become easily distracted and unable to complete tasks. They increasingly require help with personal care and other activities of daily living.

Many people with GRN-related frontal-temporal dementia develop progressive problems with speech and language (FTDP). Affected individuals may have trouble speaking, remembering words and names, and understanding speech. Over time, they may completely lose the ability to communicate.

As all other numerous **Inherited Real Risks**, Frontal-temporal Dementia heritable Risk does really exist, bedside diagnosed from birth, and treated with Reconstructing Mitochondrial Quantum Therapy (20-25). Twenty years ago, I have discovered Constitution-Dependent, Inherited Real Risks of chronic degenerative disorders, as CVD, T2DM, Osteoporosis, Cancer and of a flurry of neurodegenerative disorders: www.semeioticabiofisica.it.

All mothers of patients affected by FTD are positive to their own Inherited Real Risk.

Recently I've discovered the Inherited Real Risk of Fronto-temporal Dementia, that I predict is also eliminated with inexpensive Reconstructive Mitochondrial Quantum Therapy, as all other risks, mentioned above, dependent on mitochondrial cytopathy (CAEMH, Congenital Acidosic Enzyme-Metabolic Histangiopathy) transmitted through the mother (22-27).

The **diagnostic procedure** aiming to bedside recognize such a risk, even in apparently normal individuals, starting from the birth, begins with the evaluation of Oculo-gastric aspecific reflex: in a subjects, lying down in supine position with closed eye, physician presses with moderate intensity (500 dyne / cm2) with a fingertip on the eyeball on one side and then the other (28).

In healthy, after a Latency Time of 8 sec., in the stomach appears dilation of both fundus and body, while antral region contracts, Gastric Aspecific Reflex. Reflex intensity is less than 1 cm., and the Duration < 3 sec. – 4 sec. <

On the contrary, in subject involved by FTD Inherited Real Risk, the Reflex Parametric Values, i.e., Latency Time, Reflex Duration and Intensity, are pathologically modified, in relation to the seriousness of underlying disorder (28). Soon thereafter, the physician assesses the Cerebro-gastric aspecific Reflex by pressing with medium intensity pressure by means of a fingertip (700 dyne / cm.2) on the skin projection area of brain frontal, parietal, temporal, occipital, and cerebellar lobes.

In FTD such a reflex shows pathological parameter values, i.e., Latency Time (NN = 8 sec.), reflex Duration (NN < 3 sec. – 4 sec.<) and intensity (NN less then 1 cm.), when are stimulated the trigger points of frontal lobe, anterior and middle temporal, and cerebellar lobes.

Interestingly, in FTD the posterior third of temporal lobe is normal: differential diagnosis. Exclusively in the FTDP also parietal lobes are pathologically modified

As in all forms of dementia, also in the FTD the Cerebellar-Gastric Aspecific Reflex shows pathological parametric values starting from birth, because the cerebellum is a sensor of dementia (29).

As it is known, above-referred parametric values are related to the severity of the underlying disease. Therefore, the described clinical evaluation plays a central role in therapeutic monitoring of Frontal-temporal Dementia under treatment with Reconstructing Mitochondrial Quantum Therapy (20, 24, 27).

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