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Acquired Coenzyme Q_{10} Deficiency Leading to Mitochondrial Dysfunction is A Shared Biochemical Feature of Thyroiditis as Well as of Heart Failure with Reduced Ejection Fraction

Roy Moncayo* **and Helga Moncayo**

Abstract

Unless a disease is either congenital or genetically determined, it arises as an acquired alteration occurring during life. Thyroid and heart clinical practice guidelines address the situation of patients once disease has developed, thus leaving out the identification of any causal event. Early and current thyroid practice guidelines have been only oriented towards the use of thyroxine to keep the blood level of TSH within an apparent ideal range. This approach has resulted in patients who present so-called residual symptoms who are even put into a hodgepodge called medically unexplained symptoms. This suggests that thyroxine and TSH are not the only players to be evaluated. Careful biochemical examination of blood levels of magnesium, selenium, CoQ_{10} and ferritin can reveal single or complex deficiency states which affect mitochondrial function. The interpretation of the laboratory tests is based on defined thresholds: 0.85 mmol/L for magnesium, 100 ng/ml for ferritin, 10 µg/dl for selenium, and 2.5 mg/L or higher for CoQ_{10}. Blood levels of NT-proBNP should not exceed 125 ng/ml. Successful restoration of adequate blood levels of Mg, Se, and CoQ_{10} will result in restoration of normal morphology and perfusion of the thyroid. Iron deficiency can be present in patients with chronic tiredness requiring even iron infusions. Once patients with pre-heart failure have been detected, administration of CoQ_{10} should be considered to restore normal blood levels of NT-proBNP. A clinical evaluation will find improved quality of life as well as improved physical exercise capacity. Beneficial effects of CoQ_{10} administration emphasize the need to consider a determination of CoQ_{10} levels together with NT-proBNP in settings of preventive or initial screening. This approach has helped us to resolve the situation of residual symptoms in treated hypothyroid patients by which the nonsense term “medically unexplained symptoms” turns meaningless. We include the description of our practical diagnostic approach.

Keywords: Coenzyme Q_{10}, magnesium, selenium, ferritin, mitochondria, 3D power Doppler thyroid ultrasound.

Preamble

Through our clinical research over the past 20 years, we have discovered that benign thyroid disease presenting as thyroiditis is caused by acquired coenzyme Q_{10} (CoQ_{10}) deficiency. A typical sign is thyroid hypervascularization as seen in 3D power Doppler sonography and glycolysis as detected by 18F-FDG. Adequate correction of this deficiency, i.e., raising blood levels to > 2.5mg/L, leads to normalization of thyroid morphology and perfusion thus confirming causality. We will extend the applicability of this approach to primary diagnosis

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of pre-heart failure and delineate a causal evaluation and treatment.

Cardiotoxicity after Doxorubicin Administration – The Prototype of CoQ10 Deficiency

The pioneering work from Folkers delineated the key relation between CoQ_{10} and heart function half a century ago. The main findings of a 1970 publication were that low levels of CoQ_{10} correlated with higher classes of heart failure [1] thus delineating the picture of heart failure with reduced ejection fraction (HFrEF). The key consequence of CoQ_{10} deficiency is the induction of hypoxia [2)]which in turn is the trigger for the release of NT-proBNP [3-6]. Lack of CoQ_{10} induces a situation of HFrEF [7]. Further interactions arising from hypoxia are the inactivation of thyroid hormones [8] leading to the low-T3 syndrome and the release of IL-6 [9]. Two of the hypoxia-responsive elements related, i.e. NT-proBNP and IL-6, show a positive correlation (Figure 1) [10]. Figure 1 depicts our model of altered mitochondrial dysfunction following doxorubicin administration. Doxorubicin can displace CoQ_{10} altering the function of respiratory complex I.

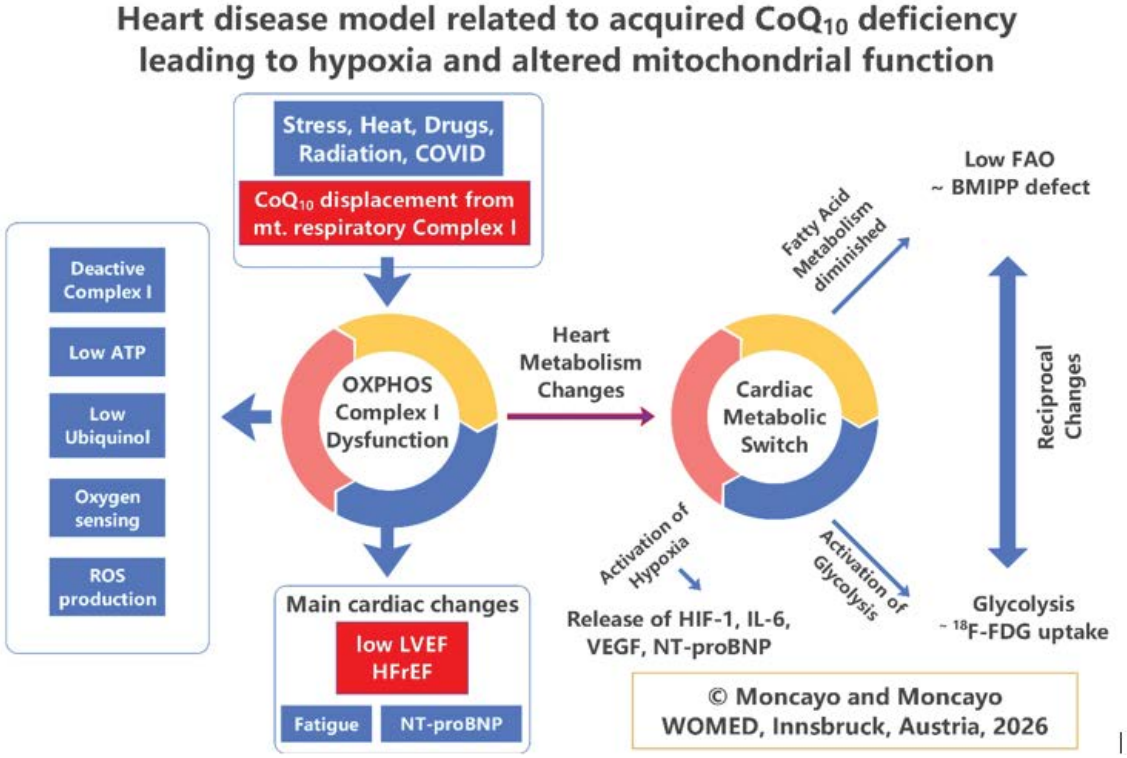


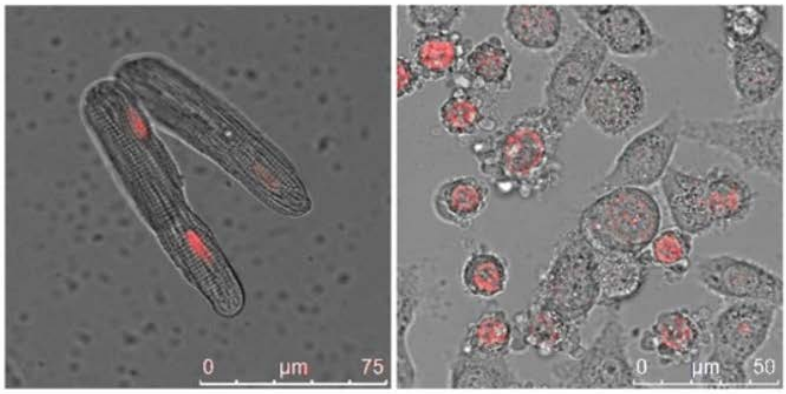
Figure 1: Integrative description of mitochondrial changes induced by doxorubicin.

The putative assumption that late cardiotoxicity arises from a historical doxorubicin exposure cannot be reconciled with simple, basic pharmacokinetics of the drug. Under ideal conditions, the half-life time of the alpha-phase is 3-5 min and the terminal half-life time is 20-30 h [11]. General principles of pharmacokinetics describe almost full disappearance of a drug by 5 t½ (12), i.e., 150 hours. Five years after administration of the drug corresponds to 1460 times the t½ cycle. This simple pharmacokinetic fact invalidates the putative, albeit failed, idea of late doxorubicin cardiotoxicity. Additional pharmacokinetic data on doxorubicin was published by Krischke et al. in 2016. Thirty hours after drug administration the plasma concentration was close to zero (Figure 2) [13]. Mohmaed Ali and coworkers have recently analyzed the pharmacokinetics of doxorubicin showing a clear diminution of drug concentration within 30 hours (Figure 1) [14]. When doxorubicin is no longer found in the body, proof of causality for this putative heart disease mechanism turns irreal [15-17]. A symbolic representation based on models of causality [18] would read: Symbols: Z: doxorubicin concentration, W: heart disease. Z causes W: $\mathbf{Z > W}$; When Z is zero, there is no effect on W: $\mathbf{Z_0 \neq W}$. McGowan and coworkers evaluated cardiotoxicity after chemotherapy with anthracycline. They could demonstrate the sequestration of doxorubicin into myocardial cells as seen in an image provided by Dr. Izabela Pitrowska (Figure 3) [19] (Figure 2 below).

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Fig. 2



Doxorubicin staining shows sequestration in (L) cardiomyocytes and (R) malignant cervical cancer cells (courtesy Dr. I Piotrowska)

Figure 2: The original Figure 2 from McGowan JV, Chung R, Maulik A, Piotrowska I, Walker JM, Yellon DM: **Anthracycline Chemotherapy and Cardiotoxicity**. *Cardiovasc Drugs Ther* 2017, **31**(1):63-75. PMID: 28185035. https://doi.org/10.1007/s10557-016-6711-0. CC BY license.

Characterization of high energy phosphates using ^{31}P cardiac spectrum has disclosed lower levels of ATP following treatment with adriamycin [20]. Experimental data from Maslow et al. showed also that high energy phosphates were altered before cardiac function changed [21]. Gamble et al. described alterations of both cardiac and skeletal muscle after anthracycline administration in breast cancer patients [22]. In 1999 Barbiroli, Iotti, and Lodi demonstrated beneficial effects on mitochondrial respiration by administering 150 mg/d of CoQ_{10}. The patients showed also an increase of cytosolic free Mg [23]. In 1996 an elegant experiment by Sarvazyan delivered images that demonstrated oxidation-sensitive fluorescence in isolated cardiac myofibers after administration of 160µM of doxorubicin. This oxidative mechanism was seen already 20 minutes after exposure (Figure 2) [24]. Okoye, Koren, and Wojtovich discussed functional changes of OXPHOS Complex I under hypoxia providing a graphical depiction of this process showing the loss of active state of Complex I by which energy production is compromised (Figure 3) [25].

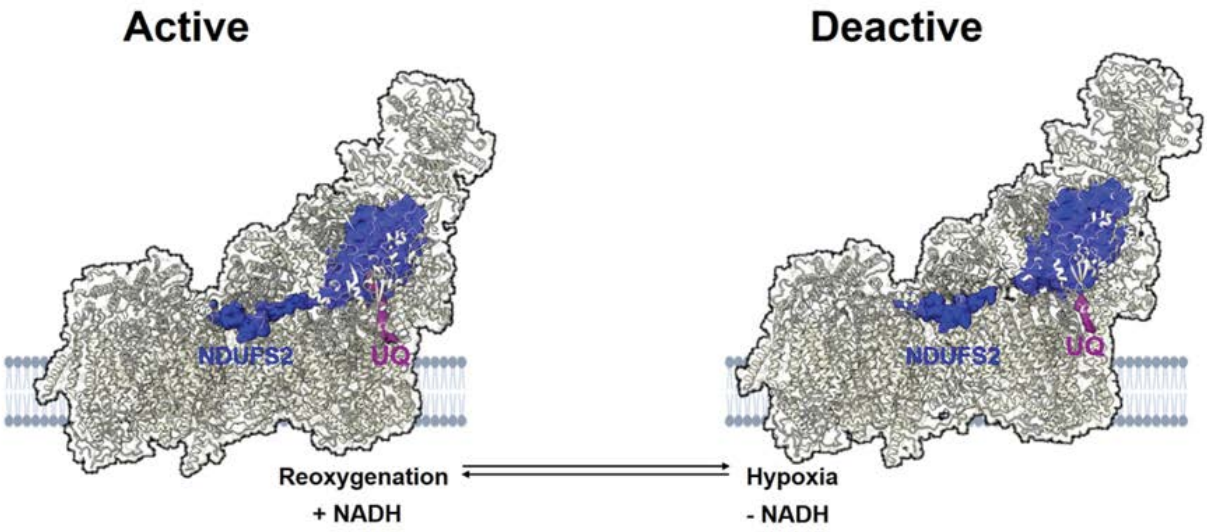


Figure 3: Reproduction of Figure 3 taken from Okoye CN, Koren SA, Wojtovich AP: **Mitochondrial complex I ROS production and redox signaling in hypoxia**. *Redox Biol* 2023, **67**:102926. PMID: 37871533. https://doi.org/10.1016/j.redox.2023.102926 CC BY-NC-ND 4.0 DE License.

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Oncological patients who have received anthracyclines can present elevated natriuretic peptide levels [26,27]. Cardiotoxicity can also appear following treatment with trastuzumab. Pre-treatment cardiac evaluation of breast cancer patients can reveal existing heart disease. These patients can develop symptomatic heart failure more frequently [28,29]. Administration of 200 mg CoQ_{10} daily to the trastuzumab treatment has shown to have beneficial effects such as preventing decline of LVEF and reduction of IL-6 and troponin levels [30]. In 2015 Li et al., were the first to demonstrate a correlation between elevated levels of hypoxia-inducible factor 1α (HIF-1α) and the biomarker NT-proBNP in patients with acute decompensated heart failure with reduced ejection fraction (HFrEF) (Figure 1 and Figure 3C [31]. Li et al. attributed the changes in HIF-1α to hypoxia and ischemia induced by hypoperfusion [31]. Hayakawa demonstrated a negative correlation between BNP levels and LVEF after doxorubicin administration [32]. In 2023 Li and coworkers described a correlation between VEGF and HIF-1α levels in patients waiting for a PCI intervention [33]. Borde, Kand, and Basu described increased cardiac 18F-FDG uptake following adriamycin therapy (Figure1) [34]. This feature demonstrated the metabolic switch to glycolysis. This type of change corresponds to HFrEF. HFrEF is accompanied by higher NT-proBNP levels [35]. Besides cardiotoxicity low levels of CoQ_{10} have been found in cases of heart transplant rejections. In 1993 Karlsson et al., evaluated the cardiac and blood levels of ubiquinone in patients who had undergone heart transplantation. They could demonstrate that patients who developed a fatal complication had low levels of CoQ_{10} [36]. A clinical study by Berman and coworkers showed that the administration of CoQ_{10} improved cardiac function in heart transplant candidates [37]. Kucharská et al., have described the association between low levels of CoQ_{10} in blood and heart tissue in cases of heart transplant failure [38,39]. Research work form the same group showed experimentally that captopril administration resulted in increased CoQ_{10} level in rabbits with smoke cardiomyopathy [40]. Similar observations on heart transplant rejection have shown that mitochondrial function is impaired [41].

Destroying a Myth -The Reversibility of Benign Thyroid Disease

In the last 2 decades, we have conducted clinical research on benign thyroid disease using advanced imaging with 3D power Doppler sonography [42] at our private practice. Over 90% of patients seeked medical consultation because they felt unsatisfied with their conventional hormonal treatment. Imaging with advanced 3D sonography revealed a pathognomonic pattern of hyper-perfusion associated to glycolysis [42]. Basic routine laboratory tests disclosed combined deficiencies of selenium [43], coenzyme Q_{10} (CoQ_{10}), and magnesium [44]. Correction by supplementation restored thyroid morphology and function (Figure 4) [45]. At the same time the unsatisfactory "residual symptoms" of treated patients [46-49] were resolved. This diagnostic and therapeutic approach demonstrated that conventional practical guidelines are insufficient to address the cause of the disease [50,51].

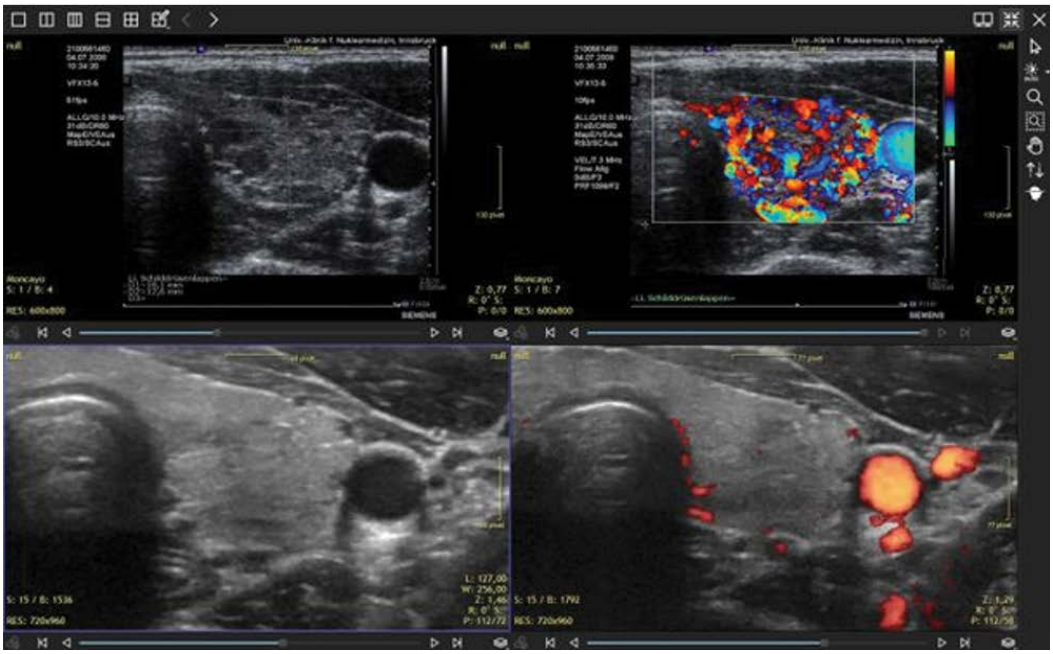


Figure 4: The upper panel shows the initial situation of the thyroid with low echogenicity, fibrosis, and hyper-perfusion in 2008. The lower panel shows the beneficial results of supplementation, i.e. morphology and perfusion have normalized in 2019.

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The detection of secondary CoQ_{10} deficiency is not routinely done. One reason for this is that this laboratory determination generates extra costs for the patient, app. € 28. As a naturally occurring compound, CoQ_{10} cannot be patented. This excludes it from pharmaceutical exploitation and from consideration as a foundational treatment for heart failure (Figure 1) [45]. Furthermore, health insurance companies consider CoQ_{10} as a nutritional supplement. Demonstration of effectiveness requires that CoQ_{10} supplementation at a sufficient dose between 200 and 300 mg/d should increase blood levels to at least 3 mg/L [52]. Therapy success should show a restoration of thyroid morphology and perfusion as well as normalization of NT-proBNP levels. In both disease groups the patient will observe improved exercise capacity and less tiredness [53]. Indirect support for this postulate is found in the 2015 study by Onur and coworkers who showed a negative correlation between NT-proBNP and ubiquinol as well as very low CoQ_{10} levels in heart disease patients [54]. We have identified additional patients showing signs of CoQ_{10} deficiency presenting COVID-19, postpartum thyroiditis, heat stress, and psychological stress (Figure 5) [55,56].

In a subgroup of patients presenting complaints such as easy fatigability, diminished exercise capacity, palpitations, tachycardia, and peripheral edema we looked beyond the standard approach for thyroid disease evaluation [57] and included determinations of NT-proBNP. We found elevated levels, i.e., >125 ng/L, ranging from 300 to 3000 ng/L. After adequate CoQ_{10} supplementation, both clinical and laboratory changes normalized, confirming the causal relation of CoQ_{10} deficiency and corrected.

Spectrum of Clinical Presentation at our Practice

Cases 1 and 2: Medically unexplained symptoms after thyroidectomy

A 39-year-old male patient presented with dissatisfaction with thyroid hormone replacement therapy after undergoing total thyroidectomy 8 years before. The patient complained about being physically unfit, presenting mood changes, a foggy brain, and diminished exercise tolerability. The attending thyroid specialist limited the examinations to the determination of fT4, fT3, and TSH. The dose of thyroid hormone replacement had been increased to 180µg per day without improving the clinical situation. A similar situation was observed in Case 2. The laboratory results disclosed a severe, combined deficiency situation which was missed by the thyroid expert.

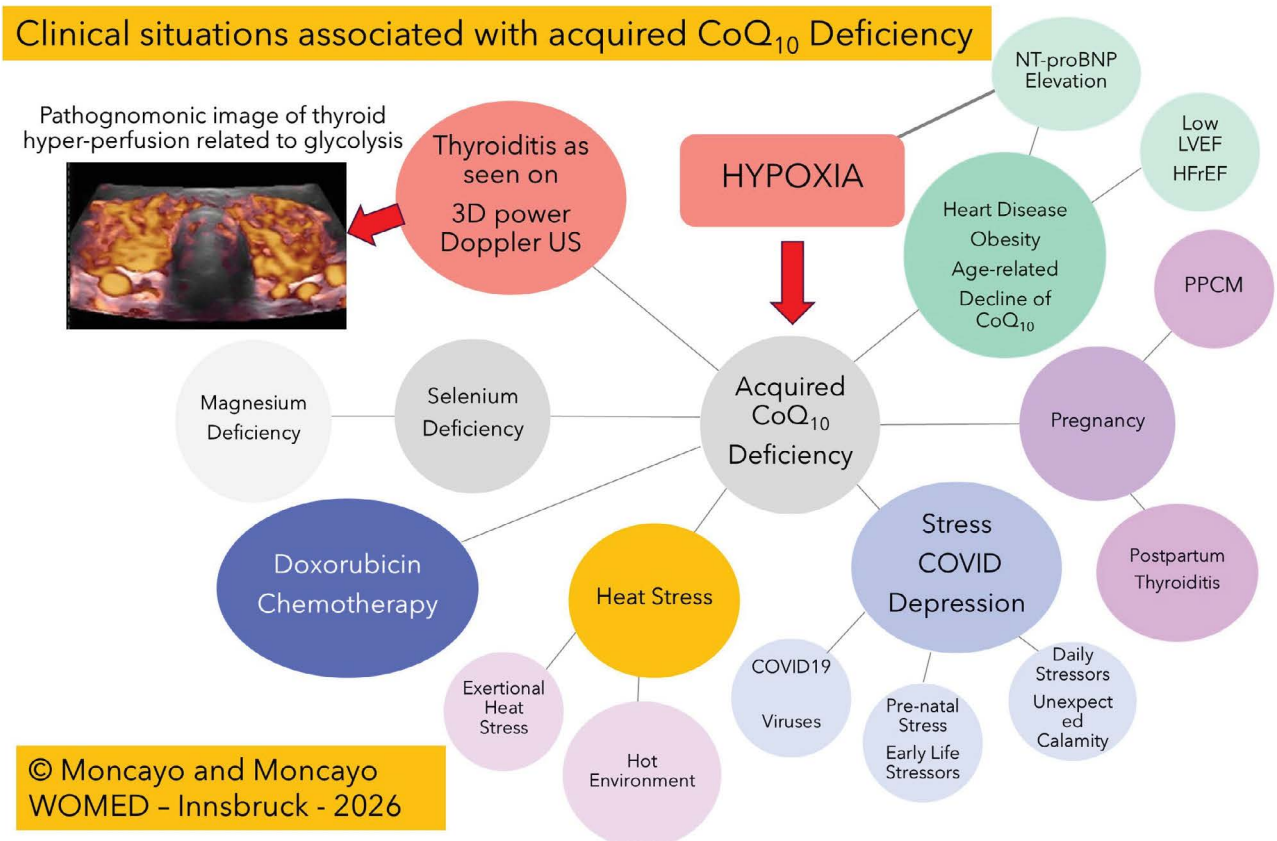


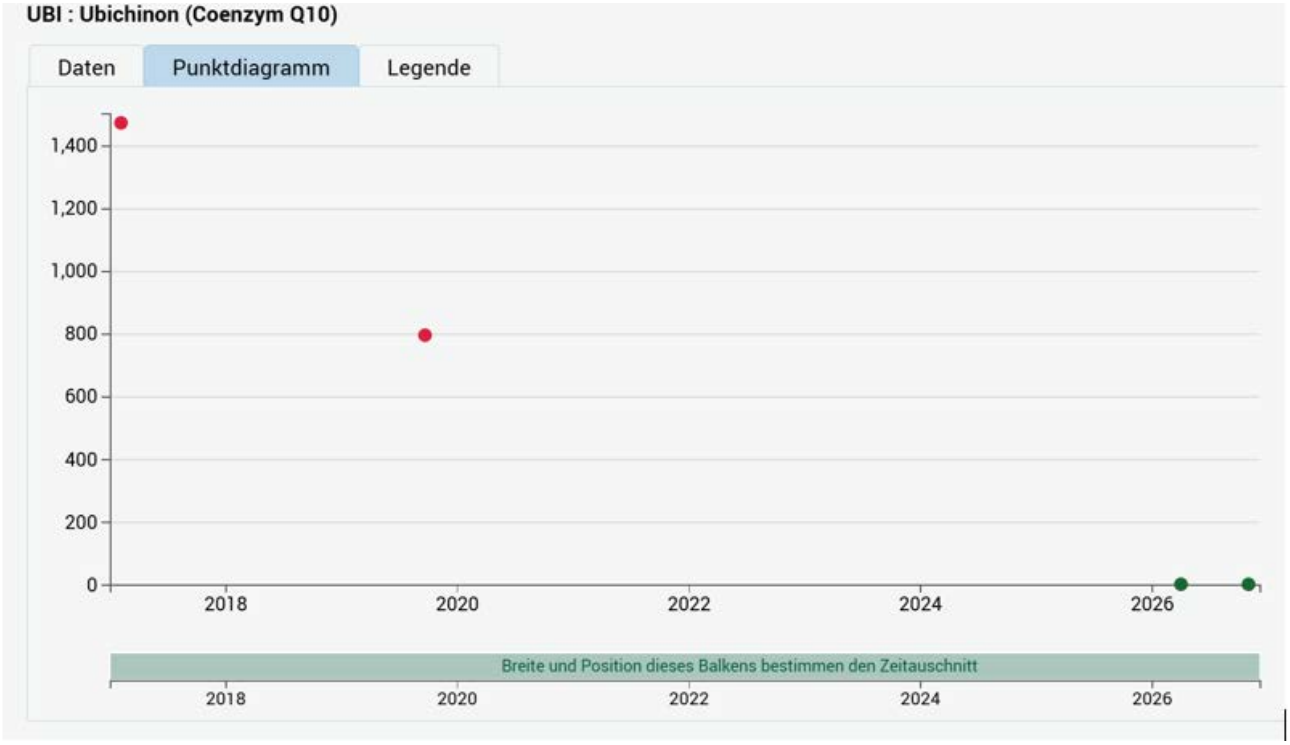
Figure 5: The relation of hypoxia to acquired CoQ10 deficiency as a primary mechanism of disease affecting the thyroid and the heart. Additional alterations of CoQ10 levels can occur in magnesium deficiency, heat stress, psychological stress, COVID-19 and pregnancy.

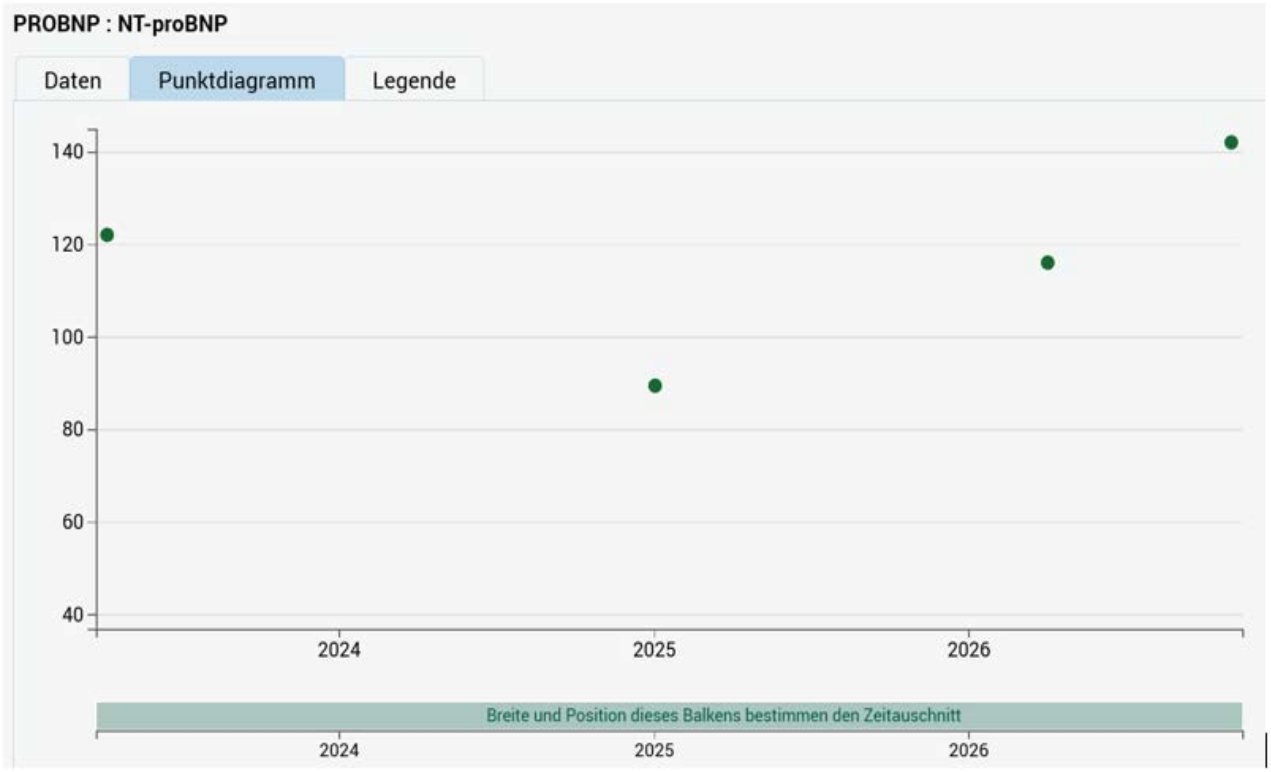
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Case 3: Routine laboratory check

A young woman underwent a yearly routine laboratory check-up examination. The laboratory test showed low CoQ10 and elevated NT-proBNP (Figures 6A and 6B).





Figures 6A and 6B: Sequential laboratory examinations showing a drop in CoQ10 levels and an increase of NT-proBNP.

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Case 4: Late thyroid dysfunction after chemotherapy

A 35-year-old female patient presented with recent recurrent hyperthyroidism. She had received chemotherapy when she was 12 years old. Soon after chemotherapy a secondary ovarian insufficiency developed. The 3D power Doppler sonography of the thyroid showed a hypoechogenic gland with hyper-perfusion.

Case 5: Hyperthyroidism after an unexpected calamity

A 55-year-old female patient presented with newly detected hyperthyroidism. She experienced an unexpected calamity in the family early in 2026. She noted a continuous decline of her physical condition in March 2026. In Apri 2026 she presented clinical signs of exhaustion and ankle edema, and NT-proBNP levels were elevated (Figure 4). The patient received the combined supplementation achieving normal NT-proBNP levels and recovery of physical condition within 3 months (Figure 7).

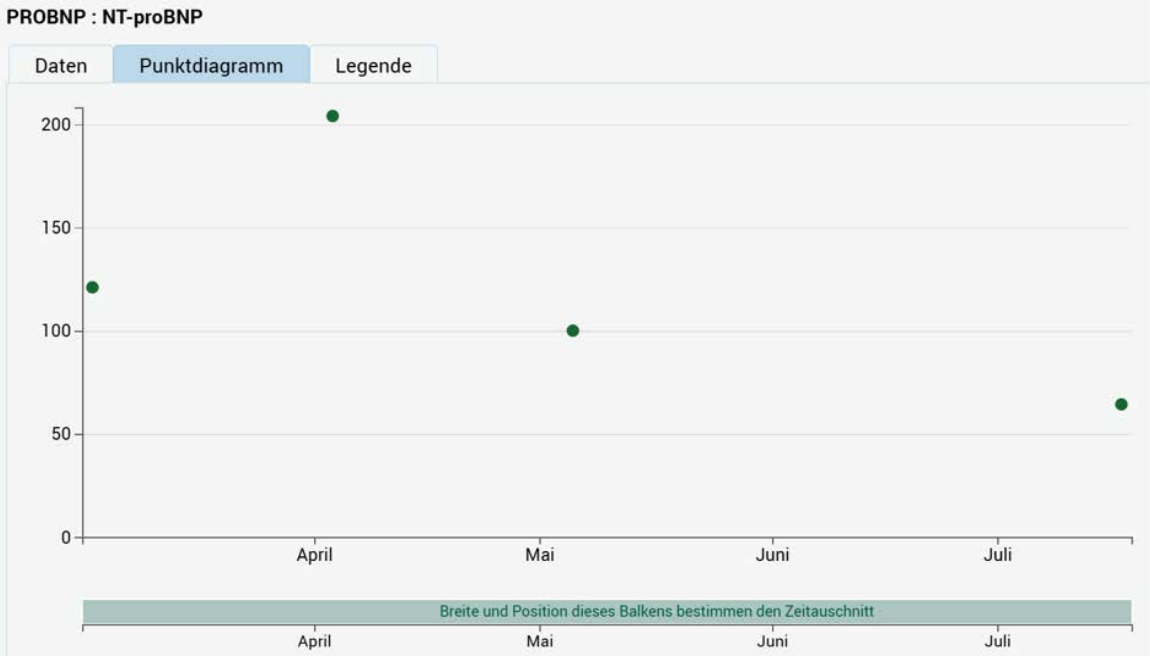


Figure 7: Normalized levels of NT-proBNP and improved physical exercise after supplementation.

Case 6: Woman with papillary thyroid carcinoma undergoing ART for natural cycle – a preconception examination.

A 37-year-old female patient was seen in a pre-conception phase undergoing assisted reproduction techniques in a natural cycle. Her external laboratory results, including magnesium and thyroid function parameters, were uneventful. In this situation only TCM diagnostics were possible. The pulse and tongue examination showed Qi deficiency, i.e. a low level of energy. Qi deficiency is a frequent finding in women examined pre-conception (58). The level of CoQ10 was 0.88 mg/l, i.e. deficient. The treatment plan included acupuncture on St 36 and Ki3 and CoQ10 supplementation beginning with 3x100mg/d.

Table 1: Summary of laboratory data cases 1 to 6.

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Parameter	**Threshold**	**1**	**2**	**3**	**4**	**5**	**6**	**Parameter**
Magnesium	> 0.85 mmol/l	0.82	0.89	0.87	0.87	0.82		Magnesium
Ferritin	> 100 ng/ml	388.1	31.5	27.5	50.5	388.1		Ferritin
NT-proBNP	< 125 pg/ml	31.9	51.4	51.7	142	31.9		NT-proBNP
Co Q_{10}	2-3 mg/l	0.59	0.79	0.92	0.85	0.59	0.88	Co Q_{10}
Selenium	> 10 µg/dl	7.47	9.5					Selenium

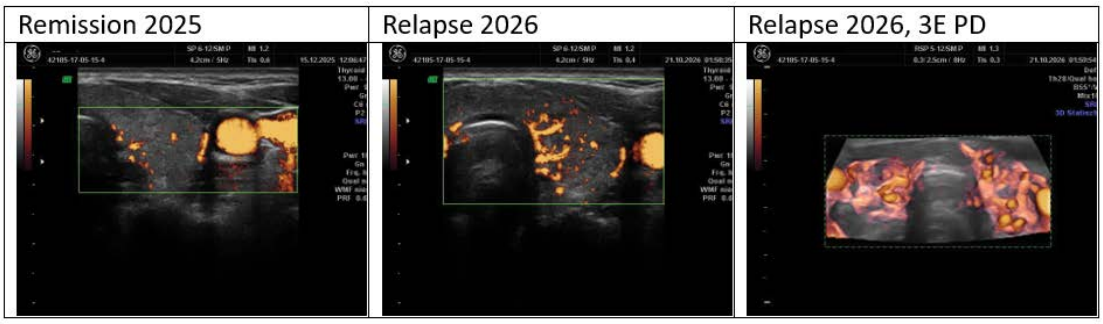
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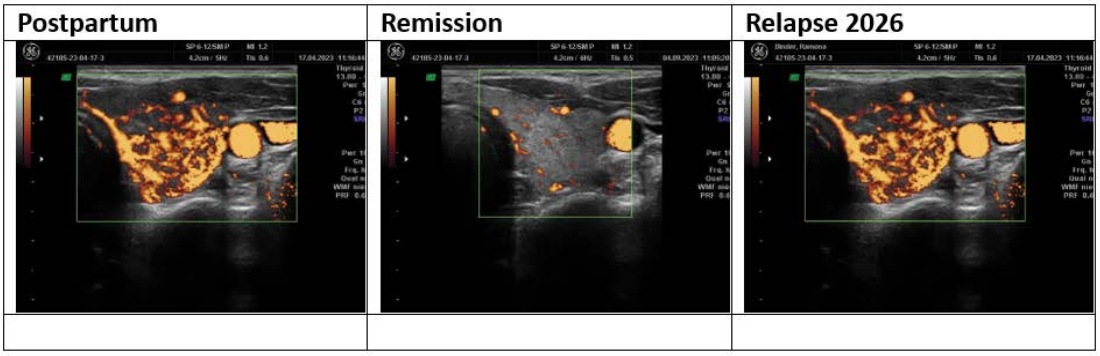
3D Power Doppler sonography approach

A fast evaluation of CoQ10 body levels can be achieved using 3D power Doppler thyroid sonography. Low levels of CoQ10 are associated with hyper-perfusion. The following cases demonstrate the utility of the method.

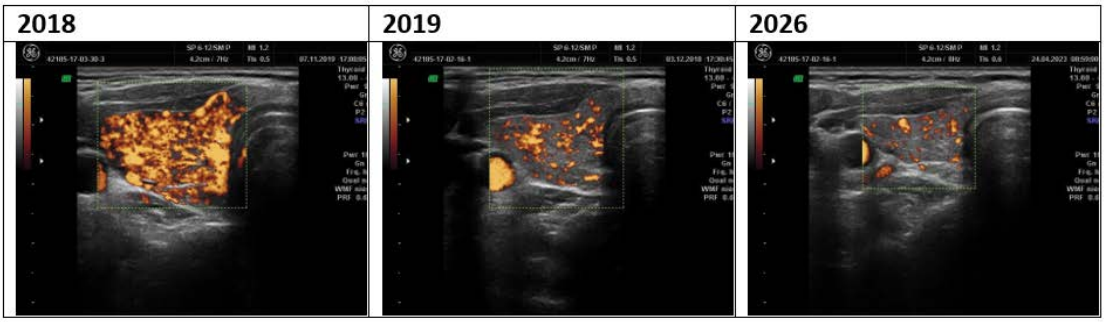
Case 7: Postpartum hyperthyroidism, normalization and relapse



Case 8: Postpartum hyperthyroidism, normalization and relapse



Case 9: Thyroiditis, normalization and near remission



Case 10 Obesity: Ultrasound and laboratory examination



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The initial evaluation of this 34-year-old woman presenting obesity revealed a positive image in the 3D-power Doppler sonography (3D PD US) showing the hyper-perfusion pattern of CoQ_{10} deficiency while the simple planar sonography was uneventful. In 2026 3D PD US showed partial remission.

In May 2025 CoQ_{10} was low 1.1 mg/l and NT-proBNP was elevated to 404 pg/ml. Ten months after combined supplementation CoQ_{10} normalized to 3.08 mg/l and NT-proBNP decreased to normal reaching 55.4 pg/ml.

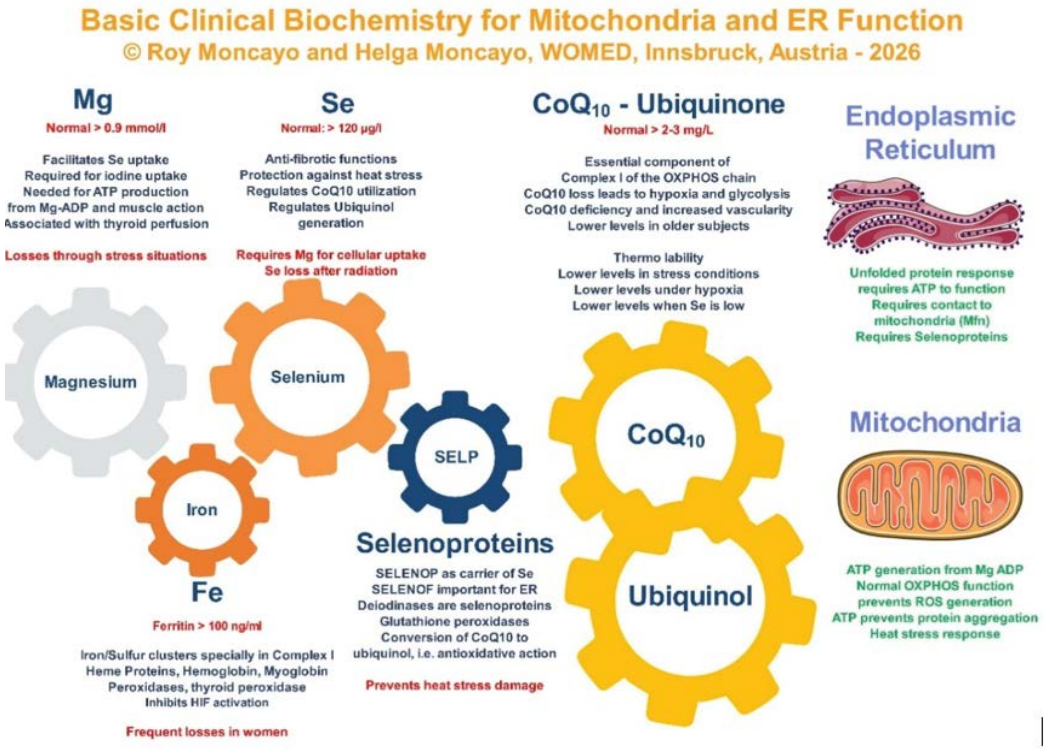


Figure 8: The interlaced relations between magnesium, selenium, CoQ10 and their relation to mitochondrial function.

Supplementation-nutrients in context of mitochondrial function

The treatment plan includes supplementation with soluble CoQ10 beginning with 3x100mg/d. Magnesium is given as Mg citrate 400mg capsules, i.d. Selenium is given twice per week. After 6-8 weeks the dose of soluble CoQ10 can be reduced to twice a day. Should symptoms or signs persist, ubiquinol (Kaneka formulation) is added, once at midday. Our supplementation plans are conceived to cover different aspects of the interlaced mitochondrial function (Figure 8). Determination of single biochemical parameters will not capture the full dimension of the biochemical situation.

Discussion

Johan P. Mackenbach has described disease as: "a failure to cope with, one or more unbalancing changes in its internal environment" [59]. Identification of the unbalancing changes, including nutrient deficiencies, can lead to approaches to prevent disease (Box 1) [59]. We firmly believe that identification of disease causality is the most important process in a medical examination. The data obtained from detailed scrutiny will serve to design a successful treatment.

The cases described herein represent only a small group of situations where CoQ_{10} deficiency was suspected. We have successfully treated several women that presented thyroiditis during pregnancy as well as post-partum thyroiditis and during preconception control prior to IVF. Many other situations are related to secondary CoQ_{10} deficiency (Figure 2). Improvement of clinical signs and symptoms after correcting the levels of CoQ10 supports our causal hypothesis. Considering thyroid and heart disease as entities that result from acquired specific deficiencies, clinical evaluation should include laboratory parameters that characterize each patient. The use of fixed reference values is essential for successful interpretation and management. The term resilience in clinical practice best describes a balanced biochemical environment having magnesium, selenium, and CoQ_{10} in the normal range. Our causal concept states that disease ensues when there is deficiency of these elements. Our case presentations show how varied the clinical situation can be, and how to approach energetic maintenance. Figure 9 illustrates our resilience concept showing that blood levels can vary according to stressors.

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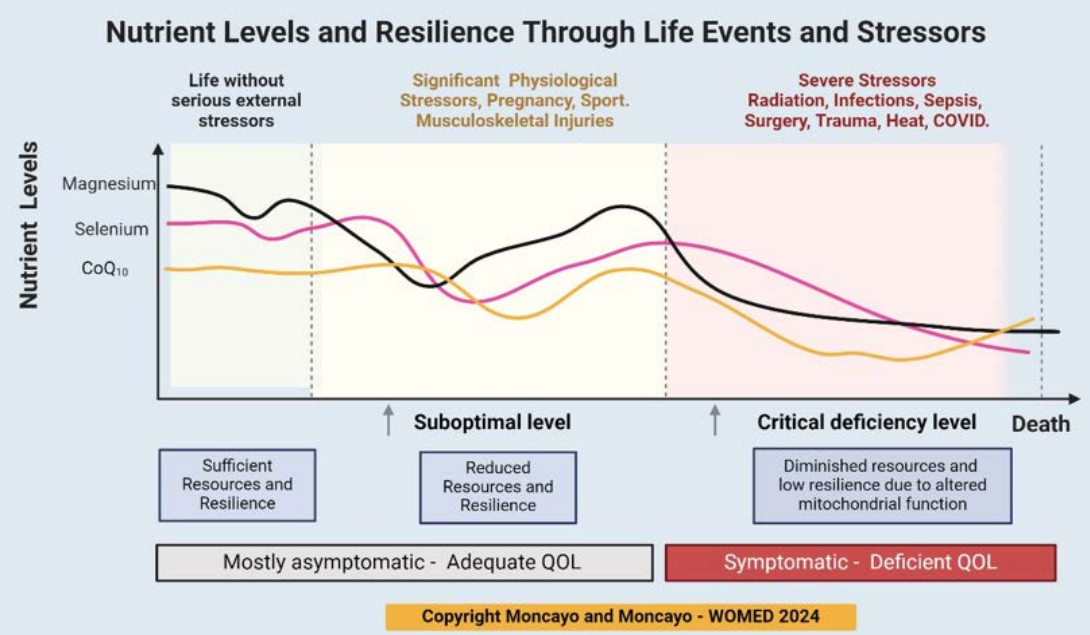


Figure 9: Idealized relation between Mg, Se, and CoQ10 and disease.

In cases of early heart failure, the goal is to normalize the levels of NT-proBNP (Figure 7) and to restore physical exercise capacity. The interpretation of laboratory results must follow the use of fixed reference values. Unfortunately, clinicians tend to introduce an adaptive change in the threshold for NT-proBNP without discussing the reason for higher values in some subjects [60]. One clinically important condition heart transplant rejection where low levels of CoQ_{10} are known to correlate with bad prognosis [38,39]. Correcting a deficiency state would be beneficial for these patients. Since CoQ_{10} is a naturally occurring chemical compound, it cannot be patented. By this it is precluded from pharmaceutical development and is not considered as an option for foundational treatment of heart failure (Figure 1 in [61]. Demonstration of effectiveness requires that CoQ_{10} supplementation at a sufficient dose between 200 and 300 mg/d should increase blood levels to at least 3 mg/L [52] and NT-proBNP levels should fall significantly [53]. Indirect support for this postulate is found in the 2015 study by Onur and coworkers who showed a negative correlation between NT-proBNP and ubiquinol as well as very low CoQ_{10} levels in heart disease patients [54]. In 2003 Alehagen and coworkers presented an evaluation of NT-proBNP as an early sign of altered heart function. They found higher levels of NT-proBNP when systolic dysfunction was present, i.e., low EF (Table 2) [62]. By supplementing selenium together with CoQ10 Johansson showed that NT-proBNP levels were lower in group of elderly patients treated over a period of 4 years (Table 1) [63]. NT-proBNP has been recently included in the classification of pre-heart failure studies applying the correct upper threshold of 125 pg/ml [64]. Oncological patients who have received anthracyclines can present elevated natriuretic peptide levels [26,27]. Cardiotoxicity can also appear following treatment with trastuzumab. Pre-treatment cardiac evaluation of breast cancer patients can reveal existing heart disease. These patients can develop symptomatic heart failure more frequently [28,29]. Administration of 200 mg CoQ_{10} daily to the trastuzumab treatment has shown to have beneficial effects such as preventing decline of LVEF and reduction of IL-6 and troponin levels [30]. In 2015 Li et al., were the first to demonstrate a correlation between elevated levels of hypoxia-inducible factor 1α (HIF-1α) and the biomarker NT-proBNP in patients with acute decompensated heart failure with reduced ejection fraction (HFrEF) (Figure 1 and Figure 3C) [31]. Li et al. attributed the changes in HIF-1α to hypoxia and ischemia induced by hypoperfusion [31]. Hayakawa demonstrated a negative correlation between BNP levels and LVEF after doxorubicin administration [32]. In 2023 Li and coworkers described a correlation between VEGF and HIF-1α levels in patients waiting for a PCI intervention [33]. Borde, Kand, and Basu described increased cardiac 18F-FDG uptake following adriamycin therapy (Figure 1) [34]. This feature demonstrated the metabolic switch to glycolysis. This type of change corresponds to HFrEF. HFrEF is accompanied by higher NT-proBNP levels [35]. Psychologic stress in preconception and pregnancy is seen with elevated IL-6 [65,66]. Patients with depression present low levels of CoQ_{10} and this can be aggravated by amitriptyline [67]. In the field of obesity, one can find some publications that have evaluated CoQ_{10} levels. Bour et al. proposed an

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antiadipogenic property of CoQ10 involved in adipocyte differentiation (68). Grenier-Larouche and coworkers evaluated CoQ_{10} content in the omentum of adipose women and found lower levels with increasing BMI values (Figure 1) in [69]. Zhang and coworkers described a beneficial effect of CoQ_{10} on the glucolipid profile and adipokine dysfunction in dyslipidemic subjects. CoQ_{10} was given daily at a dose of 120mg for 24 weeks [70]. Case 10 depicted above shows the compound picture of overweight, CoQ_{10} deficit, together with an elevated level of the cardiac biomarker NT-proBNP. This clinical picture responded to supplementation. We propose that obese patients need an initial laboratory evaluation for NT-proBNP and CoQ_{10} before sophisticated treatment options are implemented [71,72].

Conclusion

Using simple and practical biochemical investigations and fixed reference levels for interpretation, several elements that can alter mitochondrial function can be identified. This type of information can be gained neither through OMICS procedures, nor through impersonal systematic reviews and is not included in thyroid practice guidelines. Good clinical practice demands that each patient be examined individually, adding detailed account of improvement of clinical signs and symptoms after correcting the levels of CoQ_{10}. The terms residual symptoms and unexplained medical symptoms are misplaced in medical essays and should be avoided.

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Competing Interests

The authors have nothing to disclose. The authors have read the manuscript and declare no relevant financial or non-financial interests. No writing assistance was utilized in the production of this manuscript.

Consent for Publication

All authors have read the manuscript and consented for publication.

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