Starvation Genocide and the Shoah Syndrome: Psychological and Metabolic Aberrations with Osteoporosis, An Observational, Multi-System, Multi-Generational Study

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Abstract

The Shoah or the Holocaust of WWII remains as the greatest calamity in the human history. The millions of deaths within the Concentration camps were due either to the direct killings, diseases, freezing or starvation, all causes for Genocide. The Clinical Syndrome resulting from the Shoah is presented in acute and chronic phases, dealing with psychology, metabolic (glucose, lipid, cardiac, bone mineral) symptoms and findings. The syndrome has not ended with the end of the war; attention should be directed toward the future generations. Family examples of each system disease are tabulated for interpretation of practitioners caring for the few remaining survivors and their descendants as well as the victims of Famine in the new century.

Keywords: Starvation Genocide; Shoah; Psychology; Metabolic Diseases; Osteoporosis

Introduction

Did the German capitulation in May 1945 end the suffering of the survivors? Is it reasonable to assume that after surviving death camps, survivors could remain with unaltered emotional and physical health, and that the hunger and the conditions in the camps left no permanent sequel? Indeed, recent scientific studies documented that survivor of Shoah suffer more morbidity, but nonetheless they were able to reach increased longevity [1-5].

Materials and Methods

Information obtained from survivors (First generation) and their descendants (second, third and fourth), the available laboratory and historical documents constitute the material used. Survivors were patients, personal friends, or members of the authors’ families. The analysis of information obtained from the survivors and descendants was the method used for this essay. (Pseudonyms’ have been used to ensure confidentiality).

The psyche within the Shoah syndrome: Case 1: Kelly, a 23-year-old woman was detained in Auschwitz from June 1944 till 28 January 1945. Whilst foraging for food next to the surrounding barbed wires, the electrocution left two scars on her chest. This non-fatal event
could have occurred from humidity (rain or snow) diverting the electrical currents to the ground. She survived and recovered. In the acute stage, it would not be difficult to accept that looking daily at her scars and the tattoo on her forearm could be a cause for psychological disturbance. Despite diagnosed bipolar disorder, she managed her life, married, and gave birth to a son and a daughter. She was soon diagnosed with insulin-dependent diabetes. In her chronic stage she suffered from hypertension and myocardial infarct. Her 90 years ended with a cerebral event. In the next generations: son, despite a successful surgical career, suffered multiple morbidities: fear of persecution, diabetes, hypertension and two myocardial events. In the third generation, the grandson suffered from diabetes and hypothyroidism; the granddaughter had learning difficulties, responding to psychological support.

There was no knowledge of any similar pre-war pathology in the family.

**The diabetes and Lipids within the Shoah syndrome:** Alteration of glucose and lipid metabolism was considered in the literature as connected with starvation leading to Metabolic syndrome. Case no. 2: The two fathers (1 and 2) from different families, both were child survivors of the Shoah, during the war being age 2 till 4 and 4 till 8, respectively. Their nutrition was of low quality and diminished quantity. During the war, father 1 was deported to Siberia, whilst father 2 was in open Ghetto for 4 years. Post liberation, both emigrated to Israel, emerging with productive lives. Metabolic (diabetic or lipid) changes appeared for both in their fifties, the father 1 with hypertension, the father 2 with hypertension and coronary disease. Both were united in one family by their children, the second generation: The son was with diabetes and hypertension, the daughter with metabolic, thyroid, and ovarian disturbances. Their respective children (third generation, the grandchildren 1 and 2) presented with glucose metabolic aberrations in early teens. No similar family pathology was known before the war.

<table>
<thead>
<tr>
<th>Name</th>
<th>Born, Duration in camp</th>
<th>Nutrition</th>
<th>Diabetes Age at Diagnosis</th>
<th>Diseases</th>
<th>Glucose mmol/L</th>
<th>Lipids TG/Chol/ HDL/LDL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Father 1</td>
<td>b. 1942</td>
<td>Under nutrition, aged 2-4</td>
<td>63, Type 2 Metformin BMI = 24.2</td>
<td>Hypertension</td>
<td>13.0/10.6</td>
<td>5.7/4.8/0.7/7</td>
</tr>
<tr>
<td></td>
<td>3 years in Siberia and DP in Europe</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Father 2</td>
<td>b. 1936</td>
<td>Under nutrition, aged 4-8</td>
<td>65, insulin diabetes BMI = 20.0</td>
<td>Hypertension coronary stent, aortic calcifications Carotid plaque</td>
<td>6.1/6.7</td>
<td>4.0/5.0/2.8/1.0</td>
</tr>
<tr>
<td></td>
<td>4 years in Ghetto</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Son</td>
<td>b. 1971 Australia</td>
<td>Normal</td>
<td>32, Type 1 insulin, BMI = 35.3</td>
<td>Hypertension</td>
<td>20.0/8.0</td>
<td>7.6/4.6/1.0/7</td>
</tr>
<tr>
<td>Daughter</td>
<td>b. 1972 Israel</td>
<td>Normal</td>
<td>35, Insulin resistance, BMI = 21.3</td>
<td>Hashimoto, polycystic Ovaries</td>
<td>5.0/ 4.9</td>
<td>3.0/6.0/2.7/2.5</td>
</tr>
<tr>
<td>Grandson1</td>
<td>b. 2002 Australia</td>
<td>Normal</td>
<td>Insulin resistance aged 14 BMI = 23.0</td>
<td>Nil</td>
<td>4.7/ 5.4</td>
<td>3.0/6.3/1.4/2.2</td>
</tr>
<tr>
<td>Granddaughter1</td>
<td>b. 2006 Australia</td>
<td>Normal</td>
<td>Insulin =12 BMI = 22.2</td>
<td>Nil</td>
<td>3.9 /4.8</td>
<td>3.2/6.3/1.4/3.2</td>
</tr>
</tbody>
</table>

**Table 1:** Familial diabetic syndrome.
The cardiac disease within the Shoah syndrome: Case 3: Jerry, born in 1894, married and had a son (George). After "the Anschluss" fled from Austria, hiding in the Netherlands. With a weight of 39 kg and hunger oedema on liberation in May 1945, he was admitted to hospital. After recovery, had two sons, Dov (b. in 1948) and Mike (b. 1949). All three sons married and had children: Dov, a daughter (Aliza b. 1975), who respectively married and had a daughter (Lena b. 2001) and a son (Yasha b. in 2003). All descendants became hypertensive, with heart disease and had thyroid dysfunction. Blood tests showed lipid disfunction. Carotid ultrasound assessment of arterial intima, showed in all cases an increase in thickness comparatively to the rest of the population.

(The cIMT (or QIMT, quantitative intima media thickness measurements standardized with Wong protocol: Considered 0.6 mm to be the normal average value in the population is expressed as p-value or percentage).

<table>
<thead>
<tr>
<th>Generation</th>
<th>Age at diagnosis</th>
<th>Weight/Height = BMI</th>
<th>Lipids: TG, HDL-Chol</th>
<th>Blood pressure</th>
<th>EKG. Rhythm/Ischemia</th>
<th>Heart disease/Carotid U/S=cIMT</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Jerry</td>
<td>KZ in 1944.</td>
<td>120/191 = 33.2</td>
<td>2.0/0.9</td>
<td>180/70</td>
<td>A-V block Old MI, CVA</td>
<td>Aortic calcification, No cIMT</td>
</tr>
<tr>
<td></td>
<td>Weight</td>
<td>39 kg, hypertension at 70</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>II a. George</td>
<td>Age 70</td>
<td>75/179 = 23.4</td>
<td>3.1/0.8</td>
<td>160/90</td>
<td>Sinus Rhythm (=SR)/CVA</td>
<td>cIMT above p.100</td>
</tr>
<tr>
<td></td>
<td>Hypertension, Hunger Winter</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>II b. Dov</td>
<td>Hypertension at 60</td>
<td>95/192 =25.8</td>
<td>3.6/0.6</td>
<td>150/85</td>
<td>SR</td>
<td>cIMT above p.125</td>
</tr>
<tr>
<td>II c. Michael</td>
<td>Hypertension at 50</td>
<td>90/190 = 24.9</td>
<td>3.2/0.8</td>
<td>160/85</td>
<td>SR</td>
<td>cIMT p.100</td>
</tr>
<tr>
<td>III. Aliza</td>
<td>Hypertension at 35</td>
<td>69/178 = 21.8</td>
<td>4.3/0.6</td>
<td>150/80</td>
<td>SR</td>
<td>cIMT plaques and p.100</td>
</tr>
<tr>
<td>IVa. Lena</td>
<td>Hypertension at 18</td>
<td>69/173 = 22.4</td>
<td>2.1/0.9</td>
<td>145/75</td>
<td>SR</td>
<td>cIMT above p.100</td>
</tr>
<tr>
<td>IVb. Yasha</td>
<td>At 18, Hypertension at 18</td>
<td>67/173 = 20.2</td>
<td>1.8/1.0</td>
<td>140/70</td>
<td>SR</td>
<td>cIMT slightly above p100</td>
</tr>
</tbody>
</table>

**Table 2: Family recordings of lipid metabolism, blood pressure, EKG and Cmit values.**

There was no record of similar pathology in the family's pre-war period.

The Bone minerals metabolism within the Shoah syndrome: The link between intrauterine and/or early adult starvation with bone mineral metabolism aberrations has been previously established: Family no. 4: Miriam, born in Budapest was first incarcerated in Hungarian Tungsram factory, subsequently transferred to Ravensbruck KZ and then to labour camp near Leipzig. Subjected to hard labour for over 8 months and to severe nutritional deprivation. She recovered after liberation and started a family. Subsequently, she emigrated to Australia, where she lived till 96, despite obesity, thyroid problems, and osteoporosis. There was no knowledge of any fracture in the pre-war period in her family.

During her lifetime, Miriam sustained several fractures and was found to have a T-score Bone density of -4.4. (Normal between -1 and +1). She had three daughters, in good general health, but with low bone density and with T-scores of -3.3, -1.2 and -1.5, respectively. Intriguing was one granddaughter (aged 43), with regular menstrual cycle, but bone densitometry showing scores of T -2.5 / Z-2.1.

<table>
<thead>
<tr>
<th>Name</th>
<th>Age in camp</th>
<th>Age at diagnosis</th>
<th>Nutrition</th>
<th>Bone density T/Z score</th>
<th>Co-Morbidity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mother</td>
<td>26</td>
<td>61</td>
<td>Semi-starvation 8 months</td>
<td>-4.4/-2.9</td>
<td>Thyroid, obesity, fractures</td>
</tr>
<tr>
<td>Daughter 1</td>
<td>---</td>
<td>55</td>
<td>Normal</td>
<td>-2.8/-1.9</td>
<td>Hypothyroid, obesity</td>
</tr>
<tr>
<td>Daughter 2</td>
<td>---</td>
<td>39</td>
<td>Normal</td>
<td>-3.3/-2.3</td>
<td>Hypothyroid</td>
</tr>
<tr>
<td>Daughter 3</td>
<td>---</td>
<td>53</td>
<td>Normal</td>
<td>-1.0/-0.2</td>
<td>-?</td>
</tr>
<tr>
<td>Grand daughter</td>
<td>---</td>
<td>43</td>
<td>Normal</td>
<td>-2.5/-2.1</td>
<td>Normal</td>
</tr>
</tbody>
</table>

*Table 3: Familial osteoporosis within the Shoah syndrome.*

**Discussion**

World history recorded some 50 million human losses at the end of WWII including 6 million Germans, 6 million Jews, and by mid-1945, leaving behind over 1 million Jewish survivors. Raphael Lemkin, a lawyer, and translator at the Nuremberg Tribunal coined the term Genocide in 1944, carried out as social, economic, biological, physical, religious, or moral. As a result of metabolic studies in Survivors, added is now the concept of Starvation Genocide, in which the Shoah syndrome is suggested as one aspect [2].

Recalling the perennial question: Is it realistic to expect that the Shoah, would allow the memory of the Survivors to fade, that their morbidity would be like the rest of the population and that successive generations would be spared from any emotional or organic pathological consequences?

As there is no possibility of human experimentation to support the concept of Shoah Syndrome, only clinical observations and epidemiological studies would be the correct approach for diagnoses, with relevance also to the Famine victims of the 21st Century.

**Observations on the psyche aspect of the syndrome:** Medical and paramedical therapists established the list of pathologies dependant, on the age on the length of incarceration and the severity of hunger suffered [3-12]. For the children, the main effect listed was feeling of abandonment, identity problems, isolation. Adolescents aged 14 - 16, expressed similar symptoms, some remained uneducated, others reaching to successes.

In the early stages, only psychological symptoms were recorded. More recently, psychiatric disorders such as schizophrenia have been found to be statistically increased in survivors’ group [8], a pathology reflected also in the second and third generations. At a later stage, objective chemical and anatomopathological changes were also discovered such as methylation changes in the DNA [8] as well as MRI brain and EEG changes in the second generation of the survivors [9].

**Observations on the diabetic aspect of the syndrome [12-17]:** Despite reduced caloric supply, the lack of insulin supply in the camps resulted either in death, a change into T2D (type 2 diabetes) or no diabetes and its re-appearance on abundant post-war nutrition.

**Observations on the cardio-vascular aspect in the syndrome:** Cardiovascular disease in Holocaust offspring has been noted before [4,19-23]. In the acute stages, documents lead to descriptions of hunger cardiomyopathy [16,17]. In the chronic stages, survivors were found with altered lipid metabolism, coronary calcifications, and carotid wall thickening, changes not known in the pre-war period of their families [19-23].

Observations on the bone mineral aspect in the syndrome: The starvation related bone metabolism disturbance was recognised with delay compared to other metabolic components, but was eventually well documented in US, UK, Australia, and Israel [5,24,25]. The effect of malnutrition leading to abnormal bone development has been accepted clinically, epidemiologically, and experimentally and established the connection between hunger and osteoporosis. In the acute stage, in the Warsaw Ghetto, the confined physicians studied the "Hunger disease". They recorded all metabolic and physiological decline, including bone histology (information buried and recouped after the war), no cases of bone healing were recorded, and metallic bone fracture fixation was abandoned [15,16]. In the chronic cases, previous starvation "in utero" or in early childhood was found to lead to premature adult diseases, osteomalacia and osteoporosis [24,25].

Conclusive Words

The authors suggest that the Shoah syndrome is a complex psychological and metabolic, (glucose, lipid, bone mineral) disease, is multi-system and multi-generational, is one aspect of Starvation Genocide.

Limitation of the Study

The limitations of this observational clinical study are the lack of statistical analysis, lack of control cohort and lack of genetic studies. Although the genetic inheritance could not be excluded, a propensity of inheritance based on epigenetic mechanism is suggested, resulting from the conditions that prevailed during incarceration [27]. It remains for the scientists with access to data on a larger population of survivors, to verify the epidemiological value of our observations. Osteoporosis remains an occult disease, consequent and present in three generations post Starvation.

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