Starvation Genocide: Myocardial Infarction in the Lodz Ghetto

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KEY WORDS: ghetto medicine, myocardial infarction, starvation

IMAJ 2022; 24: 210–211

Medical records discovered after the liberation of ghettos in Nazi-occupied Europe are unique documents that report on the suffering of inmates, on ravaging infectious diseases, and on starvation-related organ degeneration and the resulting mortality. We attempt an explanation for the scarcity of acute myocardial infarction in the Lodz Ghetto, Poland.

BRIEF HISTORICAL BACKGROUND
At the start of World War II, the Polish city of Lodz was a flourishing industrial city with over 750,000 inhabitants. Approximately 30% of the city’s population was Jewish. Germany occupied the city and by January 1941, the Jews were forced to live in a ghetto that was completely walled off by May 1941. Initially, life was quasi normal, with education and even entertainment allowed. There were five hospitals, one specializing in infectious diseases and one in psychiatry. They were served by 170 physicians, many nurses, and midwives. Soon, however, food supply was reduced and there was a minimal supply of medications, the pharmacies were empty. Over a period of 3.5 years of the ghetto’s existence, the entire Jewish population was gradually sent to extermination camps in Chelmno and Auschwitz [1].

The post-liberation discoveries of the chronicles of the Ghetto Lodz [2] and the hospital death records [3] serve as detailed information on morbidity and the diagnosed causes of death. The Chronicles of the Ghetto, the first source on medicine, was compiled by administrators and physicians. It was discovered by Nachman Zonnabend after the liberation and was published initially in five volumes in German. Later, an abridged volume was published in English. [2]. The chronicles included records of information on births, deaths, food supply, and disease. The almost daily reports started in January 1941 (with one birth and one suicide) and continued uninterrupted until the last day of the ghetto on 31 July 1944 (with one birth the day before and one suicide on the last day).

Previous medical publications [1,4] were based on the Chronicles reporting on infectious diseases (typhus, tuberculosis) and heart disease resulting from starvation. The second source on medicine was the hospital death records [3], which noted a total of 602 deaths, diagnosed by well-educated and experienced physicians.

RESULTS
We studied the chronicles and the hospital records for the presence of myocardial infarctions. In the chronicle, during the 4.5 years of records, we found only two cases of acute infarct that occurred during the Gestapo interrogation, a condition that perhaps would be takotsubo cardiomyopathy, unknown at the time. The hospital death records diagnosed two cases of infarct and two cases of angina [3].

How can the lack of infarctions in such a stressful environment be explained? We suggest the pathogenesis is based on the research on hunger disease, which was conducted in similar conditions in the Warsaw Ghetto. These studies were conducted by the staff at the clandestine medical school. The staff was still teaching, even while starving in the ghetto. The Warsaw study results were smuggled out of the ghetto and later published in Polish and French. They were eventually translated into English and analyzed by the staff at the Mt. Sinai Hospital staff [4,5].

At the ghetto school, 27 teachers recorded abnormalities found during starvation, in particular the slowing down of basal metabolism and circulation. There were cases of bradycardia, hypotension, low cortisol, prolonged circulation time, low ST levels on electrocardiogram, and lack of response to cardiac stimulants. Was it cardiac hibernation? The heart was small with very thin walls and histology indicated myofibrillar degeneration, lipofuscin deposit, and brown and yellow atrophy.

Indeed, the reduced caloric intake was one of the Nazi’s weapons of extermination. Initially only 1200 calories were allowed for workers and only half for the nonworking free eaters. This caloric quantity was gradually reduced to 800, then to 400, and finally to 300 calories a day, before complete starvation. This reduction was a slow and gradual process of myocardial atrophy, but no coronary
occlusion. It was explained based on starvation cardiomyopathy [1,4].

CONCLUSIONS
In this observational study on a population of 160,000 in the Lodz Ghetto, we found evidence of various diseases, but very few recorded cases of myocardial infarctions despite having major emotional and physical stresses.

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References

Hall et al. investigated the duration and effectiveness of immunity in a prospective cohort of asymptomatic healthcare workers in the United Kingdom who underwent routine polymerase-chain-reaction (PCR) testing. Vaccine effectiveness (≤10 months after the first dose of vaccine) and infection-acquired immunity were assessed by comparing the time to PCR-confirmed infection in vaccinated persons with that in unvaccinated persons, stratified according to previous infection status. The authors used a Cox regression model with adjustment for previous severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection status, vaccine type and dosing interval, demographic characteristics, and workplace exposure to SARS-CoV-2. Of 35,768 participants, 27% (9488) had a previous SARS-CoV-2 infection. Vaccine coverage was high: 95% of the participants had received two doses (78% had received BNT162b2 vaccine [Pfizer-BioNTech] with a long interval between doses, 9% BNT162b2 vaccine with a short interval between doses, and 8% ChAdOx1 nCoV-19 vaccine [AstraZeneca]).

between 7 December 2020 and 21 September 2021, a total of 2747 primary infections and 210 reinfections were observed. Among previously uninfected participants who received long-interval BNT162b2 vaccine, adjusted vaccine effectiveness decreased from 85% (95% confidence interval [95%CI] 72–92) to 73 days after the second dose to 51% (95%CI 22–69) at a median of 201 days (interquartile range 197–205) after the second dose, this effectiveness did not differ significantly between the long-interval and short-interval BNT162b2 vaccine recipients. At 14 to 73 days after the second dose, adjusted vaccine effectiveness among ChAdOx1 nCoV-19 vaccine recipients was 58% (95%CI–77), which was considerably lower than that among BNT162b2 vaccine recipients. Infection-acquired immunity waned after 1 year in unvaccinated participants but remained consistently higher than 90% in those who were subsequently vaccinated, even in persons infected more than 18 months previously.

Jarvis et al. included a total of 844 participants; 814 in the intention-to-treat population. At 10 weeks, deaths were reported in 101 participants (24.8%, 95% confidence interval [95%CI] 20.7–29.3) in the liposomal amphotericin B group and 117 (28.7%, 95%CI 24.4–33.4) in the control group (difference, −3.9 percentage points); the upper boundary of the one-sided 95% confidence interval was 1.2 percentage points (within the noninferiority margin; P < 0.001 for noninferiority). Fungal clearance from cerebrospinal fluid was −0.42 log_{10} colony-forming units (CFU) per milliliter per day in the liposomal amphotericin B group and −0.42 log_{10} CFU per milliliter per day in the control group. Fewer participants had grade 3 or 4 adverse events in the liposomal amphotericin B group than in the control group (50.0% vs. 62.3%). The authors conclude that a single-dose liposomal amphotericin B combined with fluycosine and fluconazole was noninferior to the WHO-recommended treatment for HIV-associated cryptococcal meningitis and was associated with fewer adverse events.