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Multi-organ involvement and failure in selected accident cases with acute radiation syndrome observed at the Mayak Nuclear Facility

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Abstract. Mayak Production Association (Mayak PA) in the Southern Ural is the first Russian nuclear facility of plutonium production for weapons. Due to the lack of knowledge and experience in operating nuclear facilities, 19 radiation accidents including criticality accidents occurred and 59 individuals developed acute radiation syndrome (ARS) during commissioning and development of Mayak PA (1948–1958). Severe accidents that occurred at this facility are reviewed, and the initial symptoms and clinical courses of ARS in victims are discussed from the perspective of multi-organ involvement and failure.

Introduction

Whole body exposure to high doses of radiation causes acute radiation syndrome (ARS) involving multiple organs and tissues. Development of modern medicine, including intensive care, allows patients highly exposed to radiation to survive longer than before. However, the experience of the Tokai-mura criticality accident that occurred in Japan in 1999 raised problems in the treatment of heavily exposed victims. Treatment of the victims in this accident has demonstrated that bone marrow failure is not necessarily a restricting factor for recovery, but that multi-organ involvement and failure is likely to be such a factor.

Although much experience in the diagnosis and treatment of ARS and local radiation injuries has been accumulated at this facility during the past century, health consequences in ARS victims have been not fully described. This review introduces radiation accidents involving ARS at Mayak Production Association (Mayak PA), and discusses the initial symptoms and clinical courses of those patients highly exposed to radiation.

Accidents at Mayak PA during 1948–1958

After World War II, the former Soviet Union began construction of a plutonium production complex, Mayak PA, for weapons, which was the first Russian nuclear facility [1–3]. Mayak PA is located in the Southern Ural in the Chelyabinsk region, which is approximately 1000 miles east of Moscow. Mayak PA includes nuclear reactors, radiochemical and plutonium plants, and nuclear waste storage areas.

The lack of experience in operating nuclear facilities and flaws in technologies during the first decade of operation (1948–1958) resulted in 19 radiation accidents at Mayak PA, which led to development of ARS in 59 workers. These radiation accidents and ARS cases are summarised in Table 1. In most of the accidents (except one), victims were exposed to external γ or γ-neutron radiation. Among 19 accidents, 7 (37%) occurred owing to technology violation at the radiochemical plant and 4 (21%) happened at nuclear reactors. 4 accidents (21%) occurred at nuclear reactors with fuel rod manipulations. 3 of 19 accidents (16%) were accompanied by a spontaneous chain reaction. In 19 accidents, 49 men and 10 women developed ARS, and 6 men and 1 woman died of ARS.

Criticality accidents at Mayak PA during 1948–1958

A criticality accident involving plutonium nitrate solution in an interim storage vessel

On 15 March 1953 an accident occurred in a building where processing was being carried out to recover plutonium from irradiated uranium rods. Transfer of plutonium solution was made between two vessels, assuming that one vessel was empty that actually was not. The vessel became hot, there was gas release and the solution foamed. The yield was estimated to be approximately 2×10^{17} fissions. Two operators were not aware that a criticality accident was occurring, and they continued to carry out their work plans for the shift. 2 days later, one of the operators became abruptly ill and requested medical assistance; he developed ARS. Dose assessment at that time showed that the absorbed whole body dose was 10 Gy. He also suffered severe radiation burns on his legs, which later necessitated amputation of both legs. He survived for 35 years after the accident [4, 5]. The other operator received 1 Gy of irradiation. According to the data provided by the Safety Department at Mayak PA, three workers, including the two mentioned above, were involved in this accident (Table 1). The third operator involved also had ARS (Table 1). This was confirmed by the archives of primary medical histories at the Southern Ural Biophysics Institute (SUBI). This accident caused no physical damage to any equipment [4, 5].

A criticality accident involving filtration of uranyl oxalate precipitate

On 22 April 1957 there was an accident in a building that housed various operations regarding highly enriched uranium. The accident occurred in a glove box in which there was an excess accumulation of uranium during

Table 1. Radiation accidents involving the acute radiation syndrome (ARS) at Mayak Production Association (1948–1958)

Date of accident	No. persons with ARS (fatalities)		Type of accident	Main radiation factors	Type of exposure	References
	Males	Females				
19 August 1950	1	—	R/ch	γ	External	[5]
13 September 1950	1	—	R/ch	γ	External	SUBI
20 September 1950	1	—	R/ch	γ	External	[5]
1 October 1951	3 (1)	—	R/ch	γ	External	[5]
2 December 1951	3	—	R(FR)	γ-n	External	[5]
15 December 1951	2	—	R	γ-n	External	SUBI
4 March 1952	1	—	R	γ-n	External	[5]
4 July 1952	2	—	R	γ-n	External	[5]
20 September 1952	1	—	R/ch	γ	External	[5]
4 January 1953	2 (2)	—	Unknown	β (HTO)	Internal	[5]
15 March 1953	2 (1)	1	Crit	γ-n	External	[4, 5]
18 September 1953	2	—	R(FR)	γ-n	External	[5]
13 October 1953	5	—	R(FR)	γ-n	External	[5]
28 December 1953	11	—	R(FR)	γ-n	External	[5, 6]
6 November 1954	1	—	R/ch	γ	External	SUBI
3 June 1955	4	—	R	γ-n	External	[5]
22 December 1955	1	—	R/ch	γ	External	SUBI
22 April 1957	3	8 (1)	Crit	γ-n	External	[4–6]
2 January 1958	3 (3)	1	Crit	γ-n	External	[4–6]
Total	49 (6)	10 (1)				

R/ch, technology violation at the radiochemical plant; R(FR), nuclear reactor incident with fuel rod manipulations; R, nuclear reactor incident; Crit, lost control of criticality of fissionable materials.
n, neutron; β (HTO), tritiated water.
SUBI, data from the archives of primary medical histories at the Southern Ural Biophysics Institute.

filtration of uranyl oxalate precipitate. Looking through the glove box window, the operator (Patient S) was the filter vessel fabric bulge upward, followed by a violent release of gas and ejection of some of the precipitate out of the filter vessel and onto the glove box floor. The operator instinctively gathered up the precipitate by hand and put it back into the filter vessel. There was no criticality alarm system or other means of alerting the operator or nearby personnel that a criticality accident had occurred. The fact that a criticality accident had occurred was determined by a radiation control person who was called to the scene. The measurements were made approximately 15–20 min after the accident. The yield was estimated to be approximately 1×10^{17} fissions. The accident caused no mechanical damage to the vessel, and the room was not contaminated. The glove box was taken apart, cleaned and reassembled with essentially the original equipment. Operation was resumed after just a few days [4–6].

17 h after the accident, measurement of the specific activity of ^{24}Na in the operator's blood showed 245 Bq cm^{-3} [4]. The estimated absorbed dose was approximately 46 Gy (Table 2). Patient S developed nausea, vomiting, headache, and weakness/fatigue 20–30 min after the accidental exposure, suggesting the prodromal symptoms of ARS. The number of neutrophils reached zero on day 7 (Table 3). The patient had diarrhoea on day 10 and alterations were observed in the electrocardiogram. Thus, she developed gastrointestinal and neurovascular syndromes (Table 4). Furthermore, she had pulmonary symptoms such as dyspnoea on day 10. She died of severe cardiovascular collapse and acute brain oedema as well as bone marrow failure 12 days after the exposure.

Five other operators received doses up to 3 Gy [4, 5]. In this accident, another five individuals were also involved and they received doses up to 1 Gy. These were described in the record of the Safety Department at Mayak PA and confirmed by the archives of primary medical histories at the SUBI. All of these patients later recovered. This accident led to the decision to set up an in-plant critical experiment measurement capability to better determine critical parameters for vessels in routine use. The next criticality accident at Mayak on 2 January 1958 involved this critical experiment set-up [4–6].

An accident involving uranyl nitrate solution in an experimental vessel

The accident, which occurred on 2 January 1958, involved enriched uranyl nitrate solution that was being

Table 2. Profiles of victims involved in criticality accidents at Mayak Production Association and their absorbed doses

Patient	Sex (age (years))	Date of accident	Whole body absorbed dose (Gy)[a]	Dose rate (Gy min^{-1})
S	Female (25)	22 April 1957	46.03	15.36
B	Male (28)	2 January 1958	30.20–48.70	15.12–24.36
L	Male (30)	2 January 1958	69.50–124.50	34.74–62.28
M	Male (24)	2 January 1958	76.30–131.30	38.16–65.64
K	Female (33)	2 January 1958	7.13–12.13	3.54–6.06

[a]Neutron and γ absorbed acute dose.

Table 3. Initial symptoms in victims involved in criticality accidents at Mayak Production Association

Symptoms and signs	Patient S	Patient B	Patient L	Patient M	Patient K
Vomiting	Multiple, in 20 min	Multiple, in 10 min	Continuous, in 5 min	Continuous, in 10 min	Multiple, in 30 min
Erythema within 1 h	Face	Face, upper extremities	Face, chest, upper extremities	Face, chest, upper extremities	Face, upper extremities
Headache	Strong, in 30 min	Strong, in 15 min	Very strong, in 1 min	Very strong, in 5–10 min	Moderate, in 20 min
Weakness/fatigue	Moderate, in 30 min	Strong, in 15 min	Very strong, in 30 s, unable to walk	Very strong, in 5 min	Moderate, in 30 min
Abdominal pains within 2 h	Yes	Yes	Yes	Yes	No
Diarrhoea within 2 h	No	Yes	Yes	Yes	No
Blood pressure before	110/75	140/80	130/70	140/80	140/80
(mmHg) within 4 h	100/60	110/60	90/50	80/35	120/80
Heart rate in 2 h (per min)	96	100	116	110	120
Respiratory rate in 2 h (per min)	18	20	24	20	18
Body temperature in 2 h (℃)	37.4	38.2	39.3	39.2	37.0
Reflex (hyperactive)	Yes	Yes	Yes	Yes	No
Sensory disturbance	Yes	Yes	Yes	Yes	No
Mental state	Normal	Excited	Disorientated, excited	Disorientated, excited	Excited

Table 4. Symptoms at early phase of acute radiation syndrome in victims involved in criticality accidents at Mayak Production Association

Symptoms/signs	Patient S	Patient B	Patient L	Patient M	Patient K
Peripheral blood					
Agranulocytosis	Day 7	Day 6	Ni, Leukopenia on day 6	Day 6	Day 7
Thrombopenia $<150\times10^9\ l^{-1}$	Day 2	Day 3	Day 5	Day 2	Day 3
$<30\times10^9\ l^{-1}$	Day 9	Day 7	No	Day 5	Day 11
Anaemia ($<3\times10^{12}\ l^{-1}$)	No	No	No	No	No
Circulatory system					
Hypotension	Days 1–12	Days 1–9	Days 1–6	Days 1–7	No
Alterations in electrocardiogram	Day 2	Day 2	Day 3	Day 2	Days 2–4
Tachycardia	Days 1–12	Days 1–9	Days 1–6	Days 1–7	Days 1–60
Respiratory system					
Dyspnoea, increased respiratory rates	Day 10	Day 5	Day 5	Day 4	No
Gastrointestinal tract					
Diarrhoea	Day 10	No	No	No	No
Intestinal obstruction	No	Day 8	Day 5	Day 3	No
Vomiting and abdominal pains	No	No	Yes	Yes	No
Nervous system					
Disturbance of consciousness (somnolent state)	Day 12	Day 6	Day 4	Day 3	No
Reflex (hyperactivity)	Day 2	Day 2	Days 1–6	Days 2–7	Days 2–44
Sensory disturbance	No	No	Days 1–6	Day 2	No
Ataxia	No	No	Day 3	Day 2	No
Renal function					
Oliguria, proteinuria	No	No	Day 3	Day 4	No
Others					
Haemorrhage	Day 10	Day 8	Day 2	Day 2	No
Infection	Day 9 (lacunar tonsillitis)	No	No	No	Day 15 (lacunar tonsillitis)
Epilation	Day 11	Day 8	No	No	Day 11
Death	Day 12	Day 10	Day 6	Day 7	1982
Cause of death (disease/ICD-10 code)	ARS/T66	ARS/T66	ARS/T66	ARS/T66	Lung cancer/C34.9

transferred from an experimental vessel to bottles with more favourable geometry. Rather than use a drain line as prescribed, the three workers (Patients B, L and M) lifted the experimental vessel and began to move it to pour the contents manually into other bottles. They immediately noticed a flash and simultaneously fissile solution was violently ejected, reaching the ceiling approximately 5 m above. A fourth worker (Patient K), who was 2.5 m away, was also exposed to irradiation. They were all decontaminated and transferred to a hospital. Based on fusion product activity in the solution, the single-pulse yield was estimated to be approximately 2×10^{17} fissions. Owing to the severe consequences of this accident, the experimental apparatus was disassembled

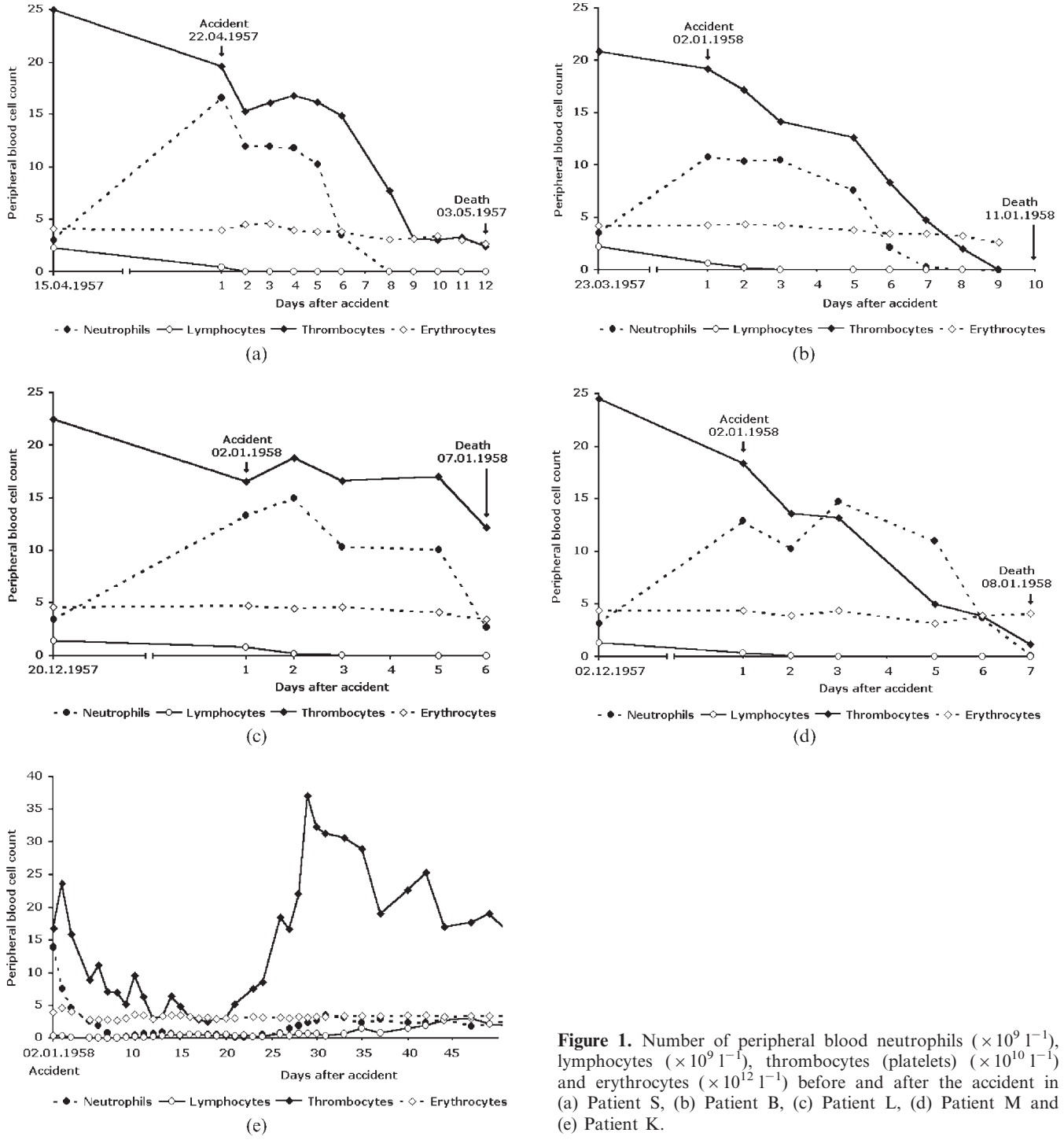

Figure 1. Number of peripheral blood neutrophils ($\times 10^9\ l^{-1}$), lymphocytes ($\times 10^9\ l^{-1}$), thrombocytes (platelets) ($\times 10^{10}\ l^{-1}$) and erythrocytes ($\times 10^{12}\ l^{-1}$) before and after the accident in (a) Patient S, (b) Patient B, (c) Patient L, (d) Patient M and (e) Patient K.

and the critical experiment program at the plant was terminated [4, 5]. Total neutron and γ absorbed whole body doses for Patients B, L, M and K were estimated at approximately 30–48 Gy, 70–125 Gy, 76–131 Gy and 7–12 Gy, respectively (Table 2).

Soon after the exposure, three of the patients (Patients B, L and M) developed typical prodromal symptoms of ARS such as vomiting, headache, weakness/fatigue, diarrhoea and fever (Table 3). 2 h after exposure, four workers involved in this accident were hospitalised with a suspicion to ARS into a specialised clinic at the former Branch No. 1 of Biophysics Institute (now SUBI). Neurological examination revealed that Patients L and M, who were exposed to extremely high doses of radiation, showed disorientation during the first hours after acute exposure. They had sensory disturbance. From the first day, Patients L and M had hypotension and tachycardia, and they had ataxia, oliguria and haemorrhage syndrome 2–4 days after the exposure (Table 4). Unlike Patients L and M, Patient B was exposed to a smaller dose and did not have disorientation, ataxia or oliguria (Table 4). Patients L, M and B showed gastrointestinal and neurovascular syndromes at the critical phase of ARS (Table 4). Patient L died of severe cardiovascular collapse

and brain oedema on the sixth day after acute exposure. Patient M died of brain oedema, and cardiovascular and respiratory failure in addition to bone marrow system suppression on the seventh day after exposure. Patient B died probably of acute renal failure as a result of massive necrosis of the skeletal muscles and the mucous coat of the digestive tract, as well as also bone marrow suppression on the tenth day after acute exposure.

Although Patient K had similar prodromal symptoms to the other three patients, she had no diarrhoea or fever at an early phase and neurovascular syndrome was slight (Tables 3 and 4). After recovery from ARS, she survived for 24 years and died of lung cancer in 1982.

Haematological changes in victims highly exposed in criticality accidents at Mayak PA

The haematopoietic system is known to be one of the most radiosensitive systems [6–15]. Figure 1 illustrates changes in the peripheral blood cell counts in Patients S, B, L, M and K. Compared with levels before the radiation accident, a significant increase in neutrophil count was observed within a few hours after exposure, and it reached zero by days 7 or 8 after exposure, except for in Patient L, who died before the neutrophil count reached zero (Figure 1c). A severe lymphopenia ($<0.1 \times 10^9\ l^{-1}$) was noticed in the peripheral blood of all patients on the second day, which remained until they died, except for Patient K who was exposed to approximately 10 Gy (Figure 1e). A decrease in the platelet (thrombocyte) count below $150 \times 10^9\ l^{-1}$ was observed in all patients during the first week, but critical thrombocytopenia ($<30 \times 10^9\ l^{-1}$) was dependent upon the dose of radiation and was observed in all patients except for Patient L, who died on 6 day after exposure (Figure 1). A decrease in the erythrocyte count of the peripheral blood was not observed in any of these patients (Figure 1), which could be explained by the lower radiosensitivity of erythrocytes and their half-life in the peripheral blood [16].

Discussion

It is well known that the primary response to radiation exposure, *i.e.* the prodromal syndrome, appears within a few hours after exposure to high dose radiation [6, 7, 17–22]. The duration of the prodromal syndrome and the latent phase is dependent on acute absorbed dose [6, 16, 21, 23–26]. We summarised and analysed the initial symptoms and signs of the prodromal syndrome in victims heavily exposed during criticality accidents at Mayak PA. The prodromal syndrome in patients exposed to radiation doses above 10 Gy was characterised by more pronounced symptoms and signs, reflecting more severe damage in organs and systems. Moreover, vomiting observed within a few minutes after exposure and diarrhoea developing within hours along with a strong weakness/fatigue are prognostic signs of the most severe ARS (*i.e.* cardiovascular and cerebral forms) [6, 16, 27]. In these patients, these symptoms and signs of prodromal syndrome developed within a few hours of the accident. Furthermore, a number of symptoms and signs present at an early phase of ARS continued to be observed within the first week in multiple organs and tissues, including the nervous system (central and vegetative compartments), the gastrointestinal tract and the cardiovascular system in these patients. These data indicate evidence that several organs were involved in the effect caused by exposure to high dose radiation. However, four of the patients described here died of cardiovascular collapse at an early phase of ARS. This is probably because intensive or critical care medicine had not yet been developed, and recently available therapeutic approaches such as cytokines were not available at the time of these accidents. On the other hand, patients in the Tokai-mura accident were intensively treated with advanced therapeutic measures. Moreover, a latent phase was not observed in our cases, suggesting that the estimated doses were higher in the Mayak cases than those in the Tokai-mura cases. Therefore, we cannot discuss our cases from the perspective of late or secondary multi-organ involvement and failure by comparing accidents between Mayak and Tokai-mura. We do not know how treatment might have modified the pathophysiology of patients leading to late or secondary multi-organ involvement and failure. However, side effects of various drugs and therapeutic means may have contributed, at least in part, to the lethal outcome and in the Tokai-mura accident victims, multi-organ involvement and failure can be seen as a secondary manifestation of both the early non-specific response to radiation and the specific radiation-induced direct damage of highly sensitive cells, *i.e.* stem cells, intestinal epithelium vascular endothelium.

In response to external insults such as infection, burns and trauma, the immune system becomes activated, seen as inflammation. Systemic inflammatory response syndrome (SIRS) is a concept that is defined as a primary response of the body to any extreme aggressive exposure, including radiation [28]. Elevated body temperature, transient increase in the number of neutrophils and increased permeability of blood vessels are typical symptoms of inflammation. These symptoms were indeed observed during the prodromal phase in our patients as well as later on. The primary response to radiation may be not specific but more likely SIRS. Both the primary non-specific response to radiation and the radiation-induced immediate cell damage may result in a breakdown of the regulatory mechanisms indispensable for the function of the organism. Further study and understanding of the pathophysiological mechanisms of multi-organ involvement and failure open new promising ways to improve treatment of ARS.

Conclusions

(i) Multiple organs or tissues are involved in severe ARS at an early period after acute exposure. (ii) Cardiovascular collapse may be an important pathophysiological state at an early phase in heavily irradiated patients. (iii) Late multi-organ involvement and failure is a secondary manifestation both of the primary non-specific response to radiation and of specific radiation-induced direct damage of highly sensitive cells.

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