

## Causes of Death in Patients with Diphtheria

*Malvina Javakhadze*

Tbilisi State Medical Academy of Post-diploma Education, Department of Infectious Diseases

### **Abstract**

On the basis of analyzing the patients who died of diphtheria in the Center of Infection Pathology in 1993-2001 we have studied clinical and functional peculiarities, course and results of diphtheria complications. Reasons causing death during diphtheria were determined. 1) In 92% of death cases miocarditis was developed, in 89% of them it caused death; among them early miocarditis prevailed (62%). 2) Infection-toxic shock was developed in 11% and in all the cases it caused death. 3) Polyneuritis was developed in 32% of the patients and nephrosis in 27%, but none of them died of it. 4) Syndrome of primary croup during this epidemy was not met in our material, but 7,6% had secondary croup. Obstruction of respiratory tract did not cause death in any of the cases.

**Keywords:** *diphtheria, complications, mortality*

The diphtheria epidemic in Georgia in 1993-2000 has revealed specific and nonspecific aftereffects, which declare themselves at the various stages of the disease. The aftereffects proceeded with high frequency and comparatively gravely in those patients who died. The analysis of aftereffects has been the goal of our research.

We studied and analyzed the histories of all patients died with the diagnosis of diphtheria available at the Center of Infectious Pathologies in 1993-2001. We studied the clinical and functional peculiarities of the diphtheria complications, their course and outcomes. Based on these facts we could develop the causes of death within diphtheria more or less completely.

In deceased patients at various stages of course of diphtheria developed a number of specific and nonspecific complications. Table 1 shows the frequency of complications by clinic forms of disease.

As we can see in the Table miocarditis developed in 61 patients (92%), multiple neuritis - in 21 (32 %),

nephrosis - in 18 (27%), bronchopneumonia - in 18 (27 %). Miocarditis as the specific complication of diphtheria developed in the majority of patients and this was the main cause of death. Miocarditis during the spread subtoxic and toxic diphtheria of I degree developed in all cases, and during hypertoxic and hemorrhagic forms the patients died due to the infectious and toxic shock so quickly that miocarditis had no time to develop.

The early miocarditis (revealed before 7 days of the course of disease) developed in 41 patients (62%), the late miocarditis (developed from 8 to 20 days of the course of disease) - in 20 % (38%), i.e. the main cause of death was the early miocarditis. Miocarditis by the clinical and objective examination was revealed by the following features: general paleness (5%), cyanosis of oral and nasal area and further general cyanosis (12%), the feeling of gravity and further the constrictive pain in the heart area (21%), tachycardia (22%) or bradycardia (15%) sometimes a fatal one. Arrhythmia (tachi-, bradyarrhythmia or fibrillation) - 30%, mute heart tones (32%), extrasystoles (10%), duplication of the first tone on the peak (5%), diastolic leap rhythm (5%), enlarged heart borders (10%).

Facial diphtheria	Total	Miocarditis		Polyneuritis		Nephrosis		Infectious-toxic shock		Encephalopathy		Gangrene of lungs and extremities		Bronchopneumonia	
		n	%	n	%	n	%	n	%	n	%	n	%	n	%
Localized															
Spread	10	10	100	2	20	3	30	-	-	-	-	1	10	3	30
Subtoxic	4	4	100	1	25	1	25	-	-	1	25	-	-	1	25
Toxic I.	3	3	100	1	33	-	-	-	-	-	-	-	-	2	66
Toxic II.	22	21	95	8	36	5	23	2	9	-	-	-	-	4	18
Toxic III.	14	12	86	6	43	6	43	-	-	-	-	-	-	5	36
Hypertoxic	3	2	67	-	-	1	33	2	66	-	-	-	-	1	33
	10	9	90	3	30	2	20	3	30	-	-	-	-	2	20
Total	66	61	92	21	32	18	27	7	11	1	1,5	1	1,5	18	27

**Tab.1** *The frequency of complications by clinic forms of disease.*

During the late miocarditis the blockade developed 1.5-times more than during the early miocarditis as the odds ration (OR) = 1.5 and the dystrophic alterations and myocardium hypoxia prevailed during the late miocarditis (OR=1.6).

As we see in the Table by the frequency of revelation the neuritis are on the second place. They developed in 21 patients (32%). Both the mono- and polyneuritis were exposed and not only during the toxic diphtheria; the chance of revelation of neuritis increased along with increase of the toxicosis. Namely, during the toxic larynx diphtheria of III degree it was 3-times more than during the spread diphtheria (OR=3). During the hypertoxic diphtheria, the patients died so quickly (in three cases) that polyneuritis had not had time to develop.

The latter caused disorder of metabolism of myelin, lipoproteids and common proteins and developed from the beginning of disease until the 7th day. The early polyneuritis developed in 11 patients in our material (53%), it was mainly revealed clinically. The patients suffered palatal paralysis (due to the closeness to the diphtheria centre) resulted in rhinophonia then mumbling, the drunk liquid leaked from the nose, pharynx was slackened and does not contracted, the saliva and sputum gathered in the oral cavity (the patients could not swallow).

The later (developed from the 7th to 20th days of disease) and delayed (after 21st day) neuritis revealed by the flabby paralysis of extremities and abdominal muscles. The late polyneuritis developed in five patients (24%) and the delayed - in 5 (24%). Generally, palatal paralysis developed in all such patients as other clinical manifestations of polyneuritis developed 6-times more during the late polyneuritis than during the early polyneuritis (OR=6.2). Namely, tetraparesis developed

in 4 patients on the 34th -63rd day of the disease (in single cases the diaphragm nerve palsy developed on the 43rd day of the disease and the dry gangrene of the left lower extremity - on the 28th day).

The third organ injured during the diphtheria is kidney. In our research the clinically expressed nephrosis (some authors call it the nephrotoxic glomerulonephritis - M. Turianov, 1996) developed in 18 patients (27%). The most frequent it was revealed on the peak of intoxication and at that time the direct effect of toxin was of the top priority. Clinically the patients had difficulties in urination and decrease in urine amount to the complete anuria, hypostasis of lower extremities, particularly in the ankle area. The urine of such patients was characterized with proteinuria (protein - 0.033-3.3%), erythrocyturia (erythrocytes - 3-5 - 10-12 mm/area), leukocyturia (leukocyte - 4-5 - 30-40 mm/area), cylindruria (cylinders 3-4 mm/area). One of the most important complications of diphtheria is the infectious and toxic shock. It was developed in three cases (11%) of our research and was always the cause of death. It developed in the first days of disease (7 days), was characterized by the fulminant course and ended by death from some hours to 2 days. During light and medium gravity forms of diphtheria, the cardiovascular damage is hardly manifested (in no case of our research). During heavy course of disease, the vasculites and thromboembolic complications were manifested. Gangrene of left lower extremities and lung in this material developed in one patient who was a boy of 12 with the laryngeal subtoxic diphtheria. On the 28th day, he suffered paresthesias and pains in the both lower extremities, mainly in the distal part of ankle. Then revealed blew spots, which blackened and on the 34th day developed gangrene of the left extremity (with the necrosis spots on the total area). At the same day, the diagnosis of lung gangrene was established and on the 46th day, the patient died.

In consideration of the latest data, we think that such complications are resulted from not only toxemia but also the specific (diphtheria) bacteremia.

In our material the alterations in lungs were in 32 patients (48.5%), where bronchopneumonia was developed in 18 (27%), and in other 14 patients under the background of myocarditis in the lungs were manifested edema events. Bronchopneumonia during all clinical forms was manifested with the similar frequency (comparatively frequent during the toxic type of I degree).

The rare non-specific complication - voluminous ventricle bleeding, profuse oral bleeding (blood leaked from the mucous tunic of oral cavity) and vaginal profuse bleeding (menses-like hemorrhage) was manifested in two patients.

We did not reveal the primary croup syndrome during this epidemic. Noteworthy is that during the secondary croup syndrome (dissemination of the process during the laryngeal diphtheria to the pharyngeal) developed in 15 (7.6%). In no case, obstruction of the respiratory tract was the cause of death.

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## Причины смерти среди умерших от дифтерией

*Мальвина Джавахадзе*

Кафедра инфекционных заболеваний Государственной медицинской академии  
последипломного образования, Тбилиси, Грузия

### РЕЗЮМЕ

На основе анализа историй болезней больных, умерших от дифтерии в центре инфекционных патологии 1993-2001 гг изучены клиничко-функциональные особенности дифтеринных осложнений и их клинического течения. Установлены причины смерти от дифтерии: 92% умер в результате развился миокардита - в 89% преобладал ранний миокардит (62%); инфекционно-токсический шок развился в 11% и явился причиной смерти. Полиневрит отмечался у 32% пациентов, а нефроз у 27%, но по этой причине летальный исход не установлен. Синдром первичного крупа во время данной эпидемии в наших материалах не отмечен, а вторичный круп развился у 7,6%. Причиной смерти ни разу не послужила обструкция дыхательных путей.

**Ключевые слова:** *дифтерия, осложнения, причина смерти*