

Analysis of macroscopic and microscopic findings from autopsies of SARS-CoV-2 infected individuals in Lithuania

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Summary

Aim of the study. To compare macroscopic and microscopic organ changes detected at autopsy in SARS-CoV-2 infected individuals between different cause-of-death groups.

Material and methods. A retrospective analysis of the State Forensic Science Service's autopsy data for 2020–2022 was conducted. 79 cases of deceased individuals with a positive nasopharyngeal swab polymerase chain reaction test result for SARS-CoV-2 were selected. Individuals were stratified by cause of death: group I – deaths due to COVID-19 disease, group II – deaths due to other causes. The level of significance was $p < 0.05$.

Results. 21.5% of individuals were in group I, and 78.5% in group II. The most common comorbidity in group I was atherosclerotic heart disease (82.4%). The most frequent causes of death in group II were chronic ischemic heart disease (33.9%) and mechanical asphyxia (19.4%). The mean age was 58.1 years in group I and 58.2 years in group II. Most subjects were male (64.7% in group I and 61.3% in group II). In group I, pleural adhesions were more frequent than in group II (52.9% vs. 25.8%, $p < 0.05$), as were hyperemic mucosa (88.2% vs. 29%, $p < 0.001$), mucous-purulent secretion in the trachea and large bronchi (76.5% vs. 27.4%, $p < 0.001$), increased lung weight (1796.3 g vs. 1193.5 g, $p < 0.001$), stiff lungs (94.1% vs. 25.8%, $p < 0.001$), reduced lung elasticity (100% vs. 50%, $p < 0.05$), purulent secretions in lung sections (41.2% vs. 6.5%, $p = 0.001$), and enlarged brain ventricles (41.2% vs. 17.7%, $p < 0.05$). Macroscopic findings of the heart, liver, kidneys, and microscopic findings of all examined organs were not statistically significantly different ($p > 0.05$).

Conclusions. Statistically significant macroscopic changes were identified in the respiratory system and the brain among the distinct cause-of-death groups. The microscopic findings did not show any statistically significant differences. The impact of SARS-CoV-2 on organ changes is difficult to assess due to the heterogeneity of the findings, age-related changes, and comorbidities.

Keywords: autopsy, COVID-19, macroscopic findings, microscopic findings, pandemic, SARS-CoV-2.

INTRODUCTION

Coronavirus disease 2019 (COVID-19) is an infection caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) [1]. The infectious disease spread rapidly after the first case in December 2019, and on March 11, 2020, the World Health Organization (WHO) declared it a pandemic [1]. There have been more than 770 million cases of COVID-19 and more than 7 million deaths [2]. The rapid spread of the infection, the rising number of individuals experiencing severe forms of the disease, and the increasing mortality rate have presented significant challenges to the healthcare system [3]. COVID-19 has emerged as an urgent public health issue, yet detailed information about the pathological changes induced by the SARS-CoV-2 virus remains limited [3, 4]. To

advance the understanding of the pathogenesis of COVID-19 and to enhance care and treatment strategies in clinical practice, it is crucial to investigate the organs affected by the disease [3, 4]. This article aims to compare macroscopic and microscopic organ changes detected at autopsy in SARS-CoV-2 infected individuals between different cause-of-death groups.

MATERIAL AND METHODS

A retrospective analysis of the Lithuanian State Forensic Science Service's autopsy data for 2020–2022 was conducted. A total of 79 cases involving deceased individuals with a positive nasopharyngeal swab polymerase chain reaction (PCR) test result for SARS-CoV-2 were selected (10 cases from 2020, 46 cases from 2021, and 23 cases from 2022).

During the pandemic, the PCR test for SARS-CoV-2 was routinely performed on all deceased persons at the State Forensic Medicine Service before autopsies. Macroscopic and microscopic changes in the organs were analyzed. Individuals were stratified by cause of death: group I – deaths due to COVID-19 disease, group II – deaths due to other causes. Statistical data analysis was performed using Microsoft Office Excel and IBM SPSS Statistics 29.0 programs. The normality of the data distribution was evaluated using the Shapiro-Wilk test. In cases where the data did not follow a normal distribution, differences between quantitative variables were assessed using the Mann-Whitney U test. Differences between qualitative variables were examined using Fisher's exact test. The results were considered statistically significant when $p < 0.05$.

RESULTS

Demographics

Group I (deaths due to COVID-19) included 21.5% ($n=17$) of the individuals, while group II (deaths due to other causes) included 78.5% ($n=62$). The average age of subjects in both groups was similar (in group I – 58.1 ± 20.7 years, in group II – 58.2 ± 16.7 years, $p > 0.05$). In both groups, more than half of the subjects were men (group I – 64.7% ($n=11$), group II – 61.3% ($n=38$), $p > 0.05$) (Table 1). The most common comorbidity in group I was atherosclerotic heart disease (82.4% ($n=14$)) (Table 2). The most common causes of death in group II were chronic ischemic heart disease (33.9% ($n=21$)) and mechanical asphyxia (19.4% ($n=12$)) (Table 3).

Table 1. Demographic characteristics of subjects

Characteristic	Group I ($n=17$)	Group II ($n=62$)	p-value
Age (years), mean $\pm$ SD	58.1 ± 20.7	58.2 ± 16.7	$p > 0.05$
Gender:			
Male, n (%)	11 (64.7)	38 (61.3)	$p > 0.05$
Female, n (%)	6 (35.3)	24 (38.7)	

Abbreviation: SD – standard deviation.

Table 2. Comorbidities of group I subjects

Concomitant disease	n (%)
Atherosclerotic heart disease (I25.1)	14 (82.4)
Old myocardial infarction (I25.2)	3 (17.6)
Other and unspecified cirrhosis of liver (K74.6)	3 (17.6)
Cyst of kidney (N28.1)	2 (11.8)
Oesophagitis (K20)	2 (11.8)
Gastritis, unspecified (K29.7)	2 (11.8)
Cystic disease of liver (Q44.6)	1 (5.9)
Cardiomyopathy, unspecified (I42.9)	1 (5.9)
Chronic tubulo-interstitial nephritis, unspecified (N11.9)	1 (5.9)
Cholelithiasis (K80)	1 (5.9)
Malignant neoplasm of the upper lobe of the lung (C34.1)	1 (5.9)
Respiratory tuberculosis, not confirmed bacteriologically or histologically (A16)	1 (5.9)
Chronic viral hepatitis C (B18.2)	1 (5.9)
Gastritis and duodenitis (K29)	1 (5.9)

Table 3. Causes of death among group II subjects

Cause of death	n (%)
Chronic ischemic heart disease	21 (33.9)
Mechanical asphyxia	12 (19.4)
Exposure to toxic substances	8 (12.9)
Pneumonia	7 (11.3)
Body beating	6 (9.7)
Liver cirrhosis	3 (4.8)
Body cooling	2 (3.2)
Aneurysm of the anterior communicating artery	1 (1.6)
Cardiomyopathy	1 (1.6)
Bleeding stomach ulcer	1 (1.6)

Macroscopic findings in the respiratory system

Pleural adhesions were observed significantly more frequently in group I compared to group II (52.9% ($n=9$) vs. 25.8% ($n=16$), $p < 0.05$). Hyperemic mucosa of the trachea and large bronchi (88.2% ($n=15$) vs. 29% ($n=18$), $p < 0.001$), as well as mucous-purulent secretion (76.5% ($n=13$) vs. 27.4% ($n=17$), $p < 0.001$), were significantly more common in group I than in group II. The overall mean lung weight was statistically significantly greater in group I compared to group II (1796.3 ± 641.4 g vs. 1193.5 ± 466 g, $p < 0.001$). Lungs were more likely to be stiff in group I compared to group II (94.1% ($n=16$) vs. 25.8% ($n=16$), $p < 0.001$), less elastic (100% ($n=17$) vs. 50% ($n=31$), $p < 0.05$), and with purulent secretion (41.2% ($n=7$) vs. 6.5% ($n=4$), $p = 0.001$) (Table 4).

Macroscopic findings in the heart

The average weight of the heart was 506.3 ± 234.9 g in group I and 423.9 ± 100.4 g in group II ($p > 0.05$). Most subjects had coronary artery atherosclerosis (82.4% ($n=14$) in group I and 80.6% ($n=50$) in group II), myocardial fibrosis (64.7% ($n=11$) in group I and 66.1% ($n=41$) in group II), and aortic atherosclerotic changes (94.1% ($n=16$) in group I and 96.8% ($n=60$) in group II) ($p > 0.05$) (Table 4).

Macroscopic findings in the liver

The average liver weight was 1724.8 ± 684.3 g in group I and 1755.7 ± 617.8 g in group II ($p > 0.05$). The liver was most commonly smooth-surfaced (82.4% ($n=14$) in group I and 83.9% ($n=52$) in group II), with a rounded free edge (64.7% ($n=11$) in group I and 80.6% ($n=50$) in group II), and with steatosis (58.8% ($n=10$) in group I and 62.9% ($n=39$) in group II) ($p > 0.05$) (Table 4).

Macroscopic findings in the kidneys

The mean weight of both kidneys was 315.5 ± 109.8 g in group I and 307.6 ± 81.0 g in group II ($p > 0.05$). The kidneys were most commonly smooth-surfaced (52.9% ($n=9$) in group I and 72.6% ($n=45$) in group II) and purple (47.1% ($n=8$) in group I and 72.6% ($n=45$) in group II) ($p > 0.05$) (Table 4).

Macroscopic findings in the brain

Brain weight in group I was 1290.8 ± 140.9 g, in group II – 1308.7 ± 145 g ($p>0.05$). The pia mater was most commonly opaque (94.1% (n=16) in group I and 90.3% (n=56) in group II) and fibrotic (70.6% (n=12) in group I and

82.3% (n=51) in group II) ($p>0.05$). At least one enlarged cerebral ventricle was observed significantly more frequently in group I than in group II (41.2% (n=7) vs. 17.7% (n=11), $p<0.05$). Atherosclerosis of the basal cerebral vessels was found in 47.1% (n=8) of subjects in group I and 38.7% (n=24) in group II ($p>0.05$) (Table 4).

Table 4. Macroscopic findings at autopsies

Characteristic	Group I (n=17)	Group II (n=62)	p-value
Respiratory system			
Pleural adhesions:			
Yes, n (%)	9 (52.9)	16 (25.8)	p<0.05
No, n (%)	8 (47.1)	46 (74.2)	
Mucosa of the trachea and large bronchi:			
Hyperemic, n (%)	15 (88.2)	18 (29)	p<0.001
Grayish, n (%)	2 (11.8)	44 (71)	
Mucous-purulent secretion in the lumen of the trachea and large bronchi:			
Yes, n (%)	13 (76.5)	17 (27.4)	p<0.001
No, n (%)	4 (23.5)	45 (72.6)	
Total lung weight (g), mean ± SD	1796.3 ± 641.4	1193.5 ± 466	p<0.001
Left lung weight (g), mean ± SD	832.6 ± 342.3	544.0 ± 217.7	p<0.001
Right lung weight (g), mean ± SD	963.7 ± 322.1	649.4 ± 268.0	p<0.001
Lung consistency:			
Stiff, n (%)	16 (94.1)	16 (25.8)	p<0.001
Soft, n (%)	1 (5.9)	40 (64.5)	
Not specified, n (%)	0 (0)	6 (9.7)	
Lung elasticity:			
Low elasticity, n (%)	17 (100)	31 (50)	p<0.05
Elastic, n (%)	0 (0)	29 (46.8)	
Not specified, n (%)	0 (0)	2 (3.2)	
Purulent secretion in lung sections:			
Yes, n (%)	7 (41.2)	4 (6.5)	p=0.001
No, n (%)	10 (58.8)	58 (93.5)	
Heart			
Heart weight (g), mean ± SD	506.3 ± 234.9	423.9 ± 100.4	p>0.05
Atherosclerosis of coronary arteries:			
Yes, n (%)	14 (82.4)	50 (80.6)	p>0.05
No, n (%)	3 (17.6)	12 (19.4)	
Myocardial fibrosis:			
Yes, n (%)	11 (64.7)	41 (66.1)	p>0.05
No, n (%)	6 (35.3)	21 (33.9)	
Atherosclerotic changes of the aorta:			
Yes, n (%)	16 (94.1)	60 (96.8)	p>0.05
No, n (%)	1 (5.9)	2 (3.2)	
Liver			
Liver weight (g), mean ± SD	1724.8 ± 684.3	1755.7 ± 617.8	p>0.05
Liver surface:			
Rough, n (%)	3 (17.6)	10 (16.1)	p>0.05
Smooth, n (%)	14 (82.4)	52 (83.9)	
Liver free edge:			
Rounded, n (%)	11 (64.7)	50 (80.6)	p>0.05
Sharp, n (%)	6 (35.3)	12 (19.4)	
Hepatic steatosis:			
Yes, n (%)	10 (58.8)	39 (62.9)	p>0.05
No, n (%)	7 (41.2)	23 (37.1)	
Kidney			
Total kidney weight (g), mean ± SD	315.5 ± 109.8	307.6 ± 81.0	p>0.05
Kidney surface:			

Rough, n (%)	8 (47.1)	17 (25.8)	p>0.05
Smooth, n (%)	9 (52.9)	45 (72.6)	
Kidney color:			
Purple, n (%)	8 (47.1)	45 (72.6)	p>0.05
Pink, n (%)	7 (41.2)	7 (11.3)	
Grayish, n (%)	2 (11.8)	9 (14.5)	
Grayish green, n (%)	0 (0)	1 (1.6)	
Brain			
Brain weight (g), mean ± SD	1290.8 ± 140.9	1308.7 ± 145	p>0.05
Transparency of the pia mater:			
Opaque, n (%)	16 (94.1)	56 (90.3)	p>0.05
Transparent, n (%)	1 (5.9)	6 (9.7)	
Fibrosis of the pia mater:			
Yes, n (%)	12 (70.6)	51 (82.3)	p>0.05
No, n (%)	5 (29.4)	11 (17.7)	
Enlarged brain ventricles:			
Yes, n (%)	7 (41.2)	11 (17.7)	p<0.05
No, n (%)	10 (58.8)	51 (82.3)	
Atherosclerosis of the basal cerebral vessels:			
Yes, n (%)	8 (47.1)	24 (38.7)	p>0.05

Abbreviation: SD – standard deviation.

Microscopic findings

Histological examination of the organs was performed on 24 out of 79 individuals (n=10 in group I and n=14 in group II). No statistically significant differences were found in the frequency of microscopic findings between the groups (p>0.05). The most common changes in the lungs of individuals in group I were fibrosis (30%, 3 out of 10 cases), hyaline membranes in the alveoli (30%, 3 out of 10 cases), interstitial pneumonia (30%, 3 out of 10 cases), and purulent-fibrinous pneumonia (30%, 3 out of 10 cases). In group II, the most common findings were hyaline membranes in the alveoli (23.1%, 3 out of 13 cases). In the heart, the principal microscopic changes in group I were myocardial fibrosis (83.3%, 5 out of 6 cases) and cardiomyocyte hypertrophy (50%, 3 out of 6 cases).

In group II, the principal heart findings were myocardial lipidosis (66.7%, 8 out of 12 cases), myocardial fibrosis (58.3%, 7 out of 12 cases), and cardiomyocyte hypertrophy (50%, 6 out of 12 cases). In the liver, steatosis was histologically detected in most individuals (71.4%, 5 out of 7 cases in group I and 70%, 7 out of 10 cases in group II). In the kidneys, nephrosclerosis was found in most cases (66.7%, 4 out of 6 cases in group I and 53.8%, 7 out of 13 cases in group II). The most frequently observed microscopic finding in group I was brain edema (66.7%, 2 out of 5 cases). In group II, brain edema was observed in 44.4% (4 out of 9 cases), and signs of neurophagia in the cerebral cortex were noted in 33.3% of individuals (3 out of 9 cases). More detailed data on the microscopic findings are presented in Table 5.

Table 5. Macroscopic findings at autopsies

Characteristic	Group		p-value
Lung			
	Group I (n=10)	Group II (n=13)	
Pulmonary fibrosis, n (%)	3 (30)	1 (7.7)	p>0.05
Hyaline membranes in the alveoli, n (%)	3 (30)	3 (23.1)	p>0.05
Interstitial pneumonia, n (%)	3 (30)	0 (0)	p>0.05
Purulent-fibrinous pneumonia, n (%)	3 (30)	1 (7.7)	p>0.05
Microthrombi in the lungs, n (%)	0 (0)	1 (7.7)	p>0.05
Heart			
	Group I (n=6)	Group II (n=12)	
Myocardial fibrosis, n (%)	5 (83.3)	7 (58.3)	p>0.05
Cardiomyocyte hypertrophy, n (%)	3 (50)	6 (50)	p>0.05
Myocardial lipidosis, n (%)	1 (16.7)	8 (66.7)	p>0.05
Focal myocardial ischemic changes, n (%)	1 (16.7)	2 (16.7)	p>0.05
Foci of myocardial necrosis, n (%)	0 (0)	1 (8.3)	p>0.05
Liver			
	Group I (n=7)	Group II (n=10)	
Hepatic steatosis, n (%)	5 (71.4)	7 (70)	p>0.05
Liver cirrhosis, n (%)	2 (28.6)	1 (10)	p>0.05

Kidney			
	Group I (n=6)	Group II (n=13)	
Nephrosclerosis, n (%)	4 (66.7)	7 (53.8)	p>0.05
Nephrocalcinosis, n (%)	1 (16.7)	2 (13.4)	p>0.05
Brain			
	Group I (n=5)	Group II (n=9)	
Brain edema, n (%)	2 (66.7)	4 (44.4)	p>0.05
Cerebral cortex neurophagy, n (%)	1 (20)	3 (33.3)	p>0.05
Microglial proliferation, n (%)	0 (0)	1 (11.1)	p>0.05
Fibrin microthrombi in subcortical blood vessels, n (%)	1 (20)	1 (11.1)	p>0.05

DISCUSSION

Concomitant diseases

The main target of the SARS-CoV-2 virus is the respiratory tract, but damage to other organ systems also occurs [5]. SARS-CoV-2 binds to angiotensin-converting enzyme 2 (ACE2) receptors localized in many tissues [3]. The clinical condition of patients is aggravated not only by the direct cytopathic effects of the virus but also by coagulopathy, superinfections, comorbidities, and mechanical ventilation-induced damage [6]. Patients who succumb to COVID-19 often have comorbidities that are prevalent in the general population, including arterial hypertension, cardiovascular diseases, diabetes mellitus, and obesity [6]. In our study, coronary artery atherosclerosis was the most prevalent comorbid condition observed in individuals who died from COVID-19.

Respiratory system

The literature primarily focuses on the macroscopic and microscopic changes in the respiratory system [3]. Our study found that individuals who died from COVID-19 had statistically significantly heavier lungs on average (1796.3 ± 641.4 g) compared to those who died from other causes (1193.5 ± 466 g). In the group of those who died of COVID-19, the lungs were also statistically significantly stiffer (94.1% vs. 25.8%), less elastic (100% vs. 50%), and had purulent secretions (41.2% vs. 6.5%) compared to the group who died from other causes. These findings of increased lung weight and stiffness in SARS-CoV-2-infected individuals align with autopsy studies from Germany (average total lung weight 1988 g), Hungary (average left lung weight 820 g, right lung 965 g), Switzerland, and Belgium [4, 7–9]. Rimmelink et al. reported pleural adhesions in 23.5% (4 out of 17 cases) and Grassi et al. in 9% (5 out of 60 cases) of SARS-CoV-2-infected individuals [9, 10]. In our study, pleural adhesions were statistically significantly more frequent in the group of those who died of COVID-19 than in the group of those who died of other causes (52.9% vs. 25.8%). Although our study did not report macroscopic thromboembolic complications, other studies have frequently found deep vein thrombosis and pulmonary embolism in autopsies of COVID-19 patients [3]. Wichmann et al. found deep vein thrombosis in 58% (7 out of 12 cases) of patients, with pulmonary embolism being the direct cause of death in one-third (4 out of 12 cases) of cases [4]. Sang et al. reported pulmonary embolism in 50% (25 out of 50 cases) of cases [11].

Microscopically, the lungs frequently exhibit diffuse alveolar damage characteristic of acute respiratory distress syndrome (ARDS) [3, 4]. In the research by Wichmann et al., diffuse alveolar damage was observed in 8 out of 12 individuals, with microscopic findings including hyaline membranes, microvascular thromboembolism, interstitial edema, and lymphocytic infiltration [4]. In our study, hyaline membranes in the alveoli were also among the primary microscopic findings in the lungs (30% in group I and 23.1% in group II).

Heart

In our study, the heart weight in the group of individuals who died from COVID-19 (506.3 ± 234.9 g) did not differ significantly compared to the group of individuals who died from other causes (423.9 ± 100.4 g). Similar heart weights were reported in autopsy studies of SARS-CoV-2 infected individuals conducted in the USA (median 500 g), Canada (mean 447 g), and Italy (mean 523 g) [11–13]. In this study, the majority of autopsies revealed coronary artery atherosclerosis, myocardial fibrosis, and atherosclerotic changes in the aorta. These macroscopic findings are linked to age-related changes and comorbid cardiovascular diseases and are partly in line with findings from other countries. Sang et al. most frequently observed ventricular hypertrophy (72%, 36 out of 50 cases), Hanson et al. reported left (48%, 10 out of 21 cases) and right (29%, 6 out of 21 cases) ventricular hypertrophy, and hemorrhagic myocardial infarction (29%, 6 out of 21 cases), while Ferro et al. identified ischemic (60%, 14 out of 23 cases) and hypoxic (34%, 8 out of 23 cases) cardiomyopathy [11–13].

The SARS-CoV-2 virus enters various heart tissues [12]. Hanson et al. detected viral RNA or spike protein in the cardiomyocytes, endothelium, interstitial spaces, and myocardial adipocytes of most subjects [12]. There are three main hypotheses regarding myocardial injury in COVID-19 infection: direct damage, thrombosis induced by vasculitis and endotheliitis, and harm resulting from the immune response [12]. Myocardial fibrosis, an indicator of chronic cardiovascular diseases, is frequently observed microscopically in individuals infected with SARS-CoV-2 [11–13]. In our study, myocardial fibrosis was also one of the most common microscopic findings in the heart (83.3% in group I, and 58.3% in group II). Ferro et al. observed varying degrees of myocardial fibrosis, cardiomyocyte hypertrophy, necrosis, and apoptosis in all 40 cases examined microscopically [13]. Viral infection is a common cause of myocarditis, but it is relatively

uncommon in the case of COVID-19 [13]. Sang et al. histologically identified myocarditis in 4% (2 out of 50 cases) of the cases, while Ferro et al. diagnosed it in 2.5% (1 out of 40 cases) [11, 13]. However, Remmelink et al. and Grassi et al. did not find histological signs of myocarditis [9, 10].

Liver

In our study, the average liver weight in the group of individuals who died from COVID-19 (1724.8 ± 684.3 g) and those who died from other causes (1755.7 ± 617.8 g) was similar. Hepatic steatosis was identified in more than half of the cases in both groups (58.8% in group I and 62.9% in group II). Comparable average liver weight (1544 g and 1987.85 g, respectively) and frequency of steatosis (63% and 57%, respectively) were reported in studies conducted in Hungary and Italy [7, 14].

Microscopically, we most commonly observed steatosis in the liver (71.4% in group I and 70% in group II). Schmit et al. reported not only steatosis but also varying degrees of centrilobular necrosis (86%), lobular and portal inflammatory infiltration (86%) among the microscopic findings [14]. The authors suggest that these liver changes are likely related to comorbidities, hypoxia, and drug hepatotoxicity rather than direct viral effects [14]. Grassi et al. did not find significant macroscopic or microscopic pathological changes in the liver [10].

Kidney

In our cohort, the overall kidney weight was similar between the groups (315.5 ± 109.8 g in group I and 307.6 ± 81.0 g in group II). For comparison, a study conducted in Hungary reported an average kidney weight of 182 g for the left kidney and 171 g for the right kidney [7]. We did not identify any macroscopic pathological kidney changes that could be associated with COVID-19 infection. In the study by Remmelink et al., kidneys were predominantly enlarged, pale, and exhibited petechiae [9]. Grassi et al. reported somewhat different results, finding renal hypoplasia, pale cortices, and edema in 48% (29 of 60) of their subjects [10].

In our research, the primary microscopic finding in the kidneys was nephrosclerosis (66.7% in group I and 53.8% in group II). According to a literature review, acute tubular necrosis is the most commonly observed microscopic finding in kidneys, typically resulting from renal hypoperfusion [6]. Nevertheless, a cytokine storm (characterized by the sudden and excessive release of pro-inflammatory markers) or a direct effect of the virus may also play a role [6, 15]

Brain

In some individuals, COVID-19 infection manifests with anosmia, ageusia, headaches, dizziness, and complications such as seizures, viral meningitis, ischemic stroke, or other neurological disorders [16]. The prevailing hypothesis suggests that the SARS-CoV-2 virus accesses brain tissue by entering the ACE2 receptors in the olfactory epithelium or the endothelial cells of the blood-brain barrier [16]. Although there is evidence suggesting that the virus can penetrate brain tissue, most observed damage is associated with coagulopathy and ischemia, with no specific signs of direct viral effect

[6, 16]. In our study, the brain weight in the group of individuals who died from COVID-19 (1290.8 ± 140.9 g) did not differ significantly from those who died from other causes (1308.7 ± 145 g). Similar data on average brain weight were reported by researchers in Germany (1302 g) and Italy (1445 g) [5, 16]. In our study, the predominant macroscopic brain findings in both groups were opaque and fibrotic pia mater, as well as atherosclerosis of the basal vessels, which were associated with age-related changes and comorbid conditions. Matschke et al. observed varying degrees of atherosclerosis of the basal vessels in all 43 cases, mild to moderate brain edema in 53% of cases, and recent ischemic infarcts in 14% of cases [5]. Agrawal et al. reported that the main macroscopic findings were acute or subacute ischemic foci (55%) and hemorrhages in the parenchyma or subarachnoid space (35%) [17]. In our study, dilated cerebral ventricles were significantly more frequent in the group of individuals who died from COVID-19 compared to those who died from other causes. Other authors' autopsy studies did not report data on ventricular dilation. However, there are cases where individuals who recovered from COVID-19 were diagnosed with hydrocephalus [18, 19].

In our cases, the main microscopic brain finding in group I was edema (66.7%), and in group II, edema (44.4%), along with signs of neuronophagia in the cerebral cortex (33.3%). According to other authors, microscopic examination of the brain commonly reveals microglial activation and cytotoxic T lymphocyte infiltration, most pronounced in the brainstem [5, 16, 17]. Matschke et al. documented substantial astrogliosis and microgliosis in the olfactory bulb, indicating a potential association with anosmia [5].

Study limitations

Our study has several limitations. First, the small sample size of patients who died from COVID-19 restricts the generalizability of our results to a broader population. Second, the lack of comprehensive clinical data on the individuals studied complicates the differentiation between changes caused by COVID-19 and those due to comorbidities. Third, some subjects lacked histological examination of internal organs, which provides more detailed information on the disease's pathogenesis than macroscopic evaluation alone. Additionally, the completeness of the data is constrained by the retrospective study design. Future studies should address these limitations by including a larger sample size and more detailed clinical and histological data.

CONCLUSIONS

In autopsies of SARS-CoV-2 infected individuals, the main macroscopic changes were in the respiratory system. Among individuals who died of COVID-19, the following macroscopic changes were statistically significantly more common: adhesions in the pleural cavity, hyperemic mucosa and mucoid-purulent secretions in the trachea and large bronchi, increased weight, stiff and less elastic lungs with purulent secretions, and enlarged cerebral ventricles. In contrast, no statistically significant macroscopic changes were found in the heart, liver, and kidneys between the groups.

No statistically significant differences in microscopic findings were identified between the study groups. Evaluating the impact of SARS-CoV-2 on macroscopic and microscopic organ

changes is challenging due to the heterogeneity of findings, age-related changes, and damage caused by comorbidities.

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Santrauka

SARS-CoV-2 INFEKUOTŲ ASMENŲ AUTOPSIJŲ METU NUSTATYTŲ MAKROSKOPINIŲ IR MIKROSKOPINIŲ RADINIŲ ANALIZĖ LIETUVOJE

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Tyrimo tikslas. Palyginti autopsijų metu nustatytus SARS-CoV-2 infekuotų asmenų makroskopinius ir mikroskopinius organų pokyčius tarp skirtingų mirties priežasčių grupių.

Tyrimo medžiaga ir metodai. Atlikta retrospektyvioji Valstybinės teismo medicinos tarnybos 2020–2022 m. autopsijų duomenų analizė. Atrinkti 79 atvejai, kai mirusiems asmenims nustatytas teigiamas nosiaryklės tepinėlio polimerazės grandininės reakcijos SARS-CoV-2 tyrimo rezultatas. Asmenys suskirstyti pagal mirties priežastį: I grupė – mirusieji nuo COVID-19 ligos, II grupė – mirusieji dėl kitų priežasčių. Reikšmingumo lygmuo buvo $p < 0,05$.

Rezultatai. I grupę sudarė 21,5 %, II grupę – 78,5 % asmenų. I grupėje dažniausia gretutinė liga buvo atero-

sklerozinė širdies liga (82,4 %). II grupėje dažniausios mirties priežastys buvo lėtinė išeminė širdies liga (33,9 %) ir mechaninė asfiksija (19,4 %). Vidutinis I grupės tiriamųjų amžius buvo 58,1 metų, II grupės – 58,2 metų. Dauguma tiriamųjų buvo vyrai (I grupėje 64,7 %, II grupėje 61,3 %). I grupėje dažniau nei II grupėje nustatytos sąaugos krūtinplėvės ertmėje (atitinkamai 52,9 % ir 25,8 %, $p < 0,05$), hiperemiška gleivinė (88,2 % ir 29 %, $p < 0,001$), gleivgaspūlingas sekretas (76,5 % ir 27,4 %, $p < 0,001$) trachėjoje ir stambiuosiuose bronchuose, padidėjusio svorio (1796,3 g ir 1193,5 g, $p < 0,001$), standūs (94,1 % ir 25,8 %, $p < 0,001$), sumažėjusio elastingumo plaučiai (100 % ir 50 %, $p < 0,05$), pūlingas sekretas plaučių pjūviuose (41,2 % ir 6,5 %, $p = 0,001$), išsiplėtę galvos smegenų skilveliai (41,2 % ir 17,7 %, $p < 0,05$). Širdies, kepenų, inkstų makroskopiniai radiniai ir visų tirtų organų mikroskopiniai radiniai statistškai reikšmingai nesiskyrė ($p > 0,05$).

Išvados. Nustatyta statistškai reikšmingų makroskopinių kvėpavimo sistemos ir galvos smegenų pokyčių tarp skirtingų mirties priežasčių grupių. Statistiškai reikšmingų mikroskopinių radinių skirtumų nerasta. Įvertinti SARS-CoV-2 įtaką organų pokyčiams sunku dėl radinių heterogeniškumo, amžinių pokyčių ir gretutinių ligų sukeltų pažeidimų.

Reikšminiai žodžiai: autopsija, COVID-19, makroskopiniai radiniai, mikroskopiniai radiniai, pandemija, SARS-CoV-2.

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