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Investigation of respiratory tract coinfections in Coronavirus disease 2019 infected and suspected cases

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ABSTRACT

OBJECTIVE: The aim of our study is to determine the risk of coinfection with COVID-19 due to the high prevalence of viral agents in Istanbul in autumn (September, October, and November) and winter (December and January) and to investigate the effects of age, gender, season and clinical features on the development of coinfection with COVID-19.

METHODS: In the routine studies of our hospital, COVID-19, reverse transcriptase polymerase chain reaction (RTA kit, Turkiye) and Multiplex PCR Bio-Fire (Bio Merieux Company, France) methods were studied from the nasopharyngeal swab sample and the data were recorded. A total of 400 people with a mean age (7.91±17.80) were included in the study by retrospective scanning.

RESULTS: Considering the virus distribution, Respiratory syncytial virus (RSV), COVID-19, rhino/entero virus did not show a significant difference in autumn and winter, while H. metapneumovirus, adeno virus, influenza A significantly higher rates were observed in winter months. Parainfluenza (1, 2, 3, 4) and Corona OC43 were detected at a higher rate in autumn compared to other viruses. Double and triple coinfection rates with other viral agents were high for 2 years and younger.

CONCLUSION: The risk of coinfection of COVID-19 with influenza A, RSV, parainfluenza, and rhino/entero virus was found to be higher than other viral agents. Especially in winter, the risk of coinfection with influenza A and COVID-19 increases. In terms of treatment management, coinfection should be investigated in risky patients and influenza a vaccine should be offered to risky groups.

Keywords: Coinfection; COVID-19; multiplex PCR.

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COVID-19 global pandemic, as it is known, emerged in the autumn period, when other viruses can often be found. Disease symptoms have shown clinical manifestations ranging from asymptomatic to acute respiratory distress syndrome and death. Is there an additional viral infection in cases with severe clinical course? or cross positivity with other forms of coronavirus in asymptomatic cases? such questions came to mind. However, in the routine, priority was made for COVID-19 research and screening. Due to the expensiveness of diagnostic

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kits containing other viruses, they could not be used for screening purposes. Approximately 5000 tests/day COVID-19 reverse transcriptase polymerase chain reaction (RT PCR) were studied in our hospital, which served as the center of the pandemic in Turkiye, Istanbul. Considering that it will be seen frequently in other viruses in autumn and winter months, a viral respiratory panel has been added to the routine. The data of the patients studied in the routine were evaluated. Common cold coronaviruses that cause respiratory tract infections are HCoV-229E, HCoV-OC43, HCoV-NL63, HCoV-HKU1, four endemic HCoVs, they use different receptor molecules with varying host cell tropism [1]. SARS-CoV-1 and MERS-CoV SARS-CoV-2 are zoonotic emerging epidemic pathogens that cause significant morbidity and mortality. Endemic HCoVs are known to cause coinfections or can be detected with each other or with other respiratory viruses. The distribution of respiratory virus agents differs between countries, states and even cities. Coinfections with influenza A, influenza B, adenovirus, parainfluenza virus, Human metapneumovirus, bocavirus, respiratory syncytial virus (RSV), rhino/enterovirus, Ebstein-Barr virus (EBV), and Cytomegalovirus (CMV) have been reported in studies. Knowing the coinfection is of high importance in the diagnosis, follow-up and treatment of the disease [2–5].

The aim of our study was to investigate the presence of coinfection in patients whose multiplex PCR respiratory panel studied in the autumn and winter of 2021–2022, and to investigate virus infections and coinfections that are common in our region and to evaluate the effects of age, gender, clinic and season on the occurrence of coinfection.

MATERIALS AND METHODS

Study Design

Viral agents and demographic data determined by the syndromic panel in autumn and winter were reviewed retrospectively. Age, gender, and initial complaints of the patients were recorded.

Detection of COVID-19 with RT PCR

The COVID-19 detection was performed by the COVID-19 qPCR test (Direct Detect RTA, Turkiye) on BioRad CFX 96 platform (California, USA) according to the protocols provided by the manufacturer. The qPCR kit targets ORF1ab and the N gene of SARS-CoV-2 and the human RNaseP gene.

Highlight key points

- Coinfection with COVID 19 and other viruses is particularly common in pediatric patients.
- Respiratory panel should be used in the evaluation of risky patient groups.
- Considering that Influenza A and COVID 19 coinfection may also be severe, Influenza A vaccination should be recommended to risky groups.

Detection of Respiratory Pathogens with Syndromic Panel

By using BioFire FilmArray Multiplex PCR System (BioMerieux, France) Adenovirus, coronavirus 229E, coronavirus HKU1, coronavirus NL63, coronavirus OC43, middle east respiratory syndrome coronavirus (MERS-CoV), Human metapneumovirus, Human rhinovirus/entrerovirus, influenza A, influenza B, parainfluenza virus 1, parainfluenza virus 2, and parainfluenza virus 3 viruses were studied.. In addition to, parainfluenza virus 4, RSV virus, Bordetella parapertussis, Bordatella pertussis, Chlamydia pneumoniae, Mycoplasma pneumoniae among the bacteria found on the panel of the device were investigated.

Statistical Analysis

NCSS (Number Cruncher Statistical System) 2007 (Kaysville, Utah, USA) program was used for statistical analysis. While evaluating the study data, descriptive statistical methods (mean, standard deviation, median, frequency, ratio, minimum, and maximum) were used for groups with normal distribution, independent sample t-test, and for comparison of groups not showing normal distribution, Kruskal-Wallis test was used. Logistic regression analysis was used in the impact analysis. Significance was assessed at levels of $p<0.05$.

RESULTS

The study was conducted with a total of 400 subjects, 57.5% (n=230) male and 42.5% (n=170) female, with a mean age of 7.91±17.80. When their age is examined; 2 years and younger; 66.5% (n=266), 3–10 years old; 15.5% (n=62), 11–18 years old; 6.8% (n=27), 19 years and over; 11.3% (n=45). The average age is 7.91 years. Min–Max (median): 0–91(1). Detected virus rates: RSV; 119 cases, rhino/entero; 155 cases, COVID-19; 96 cases, parainfluenza (1,2,3,4); 30 cases, Corona 29E;4 cases, Corona HKU1; 1 cases, Corona NL63;1

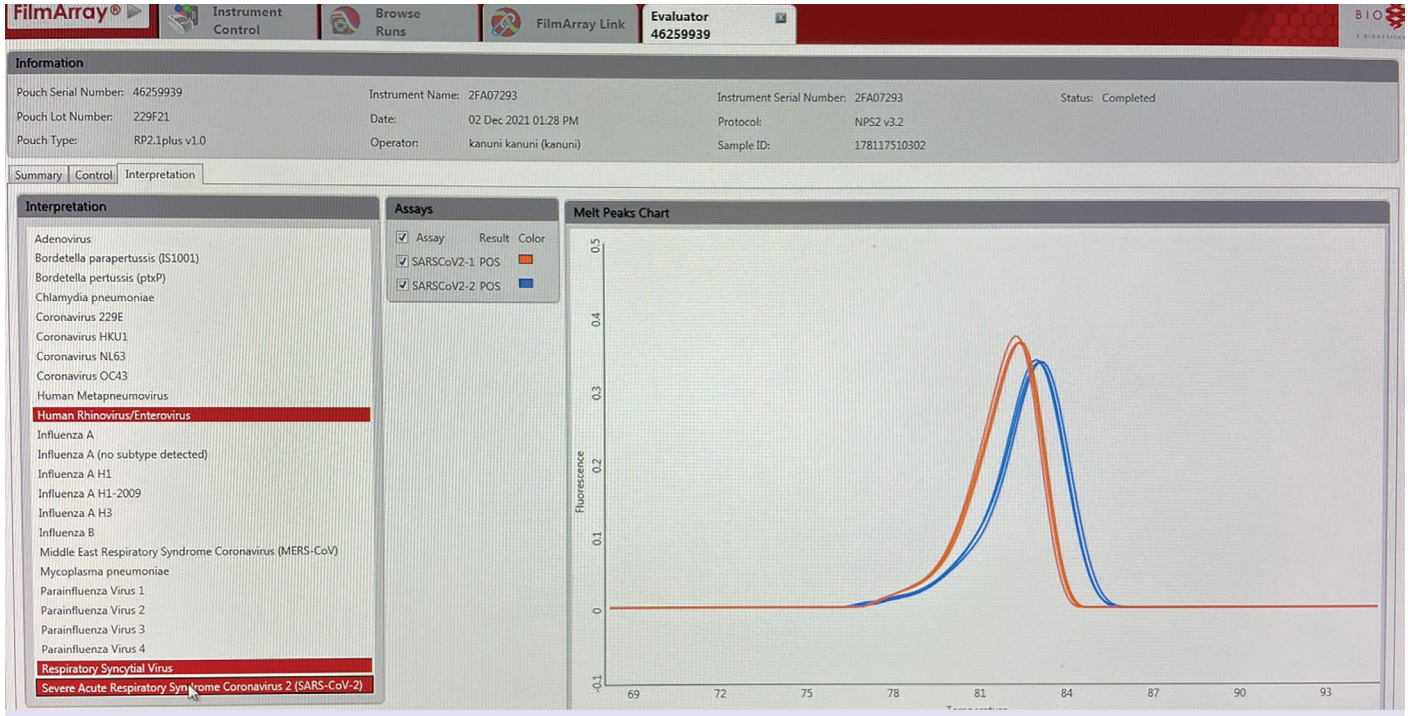

FIGURE 1. Triple coinfection peak of COVID-19.

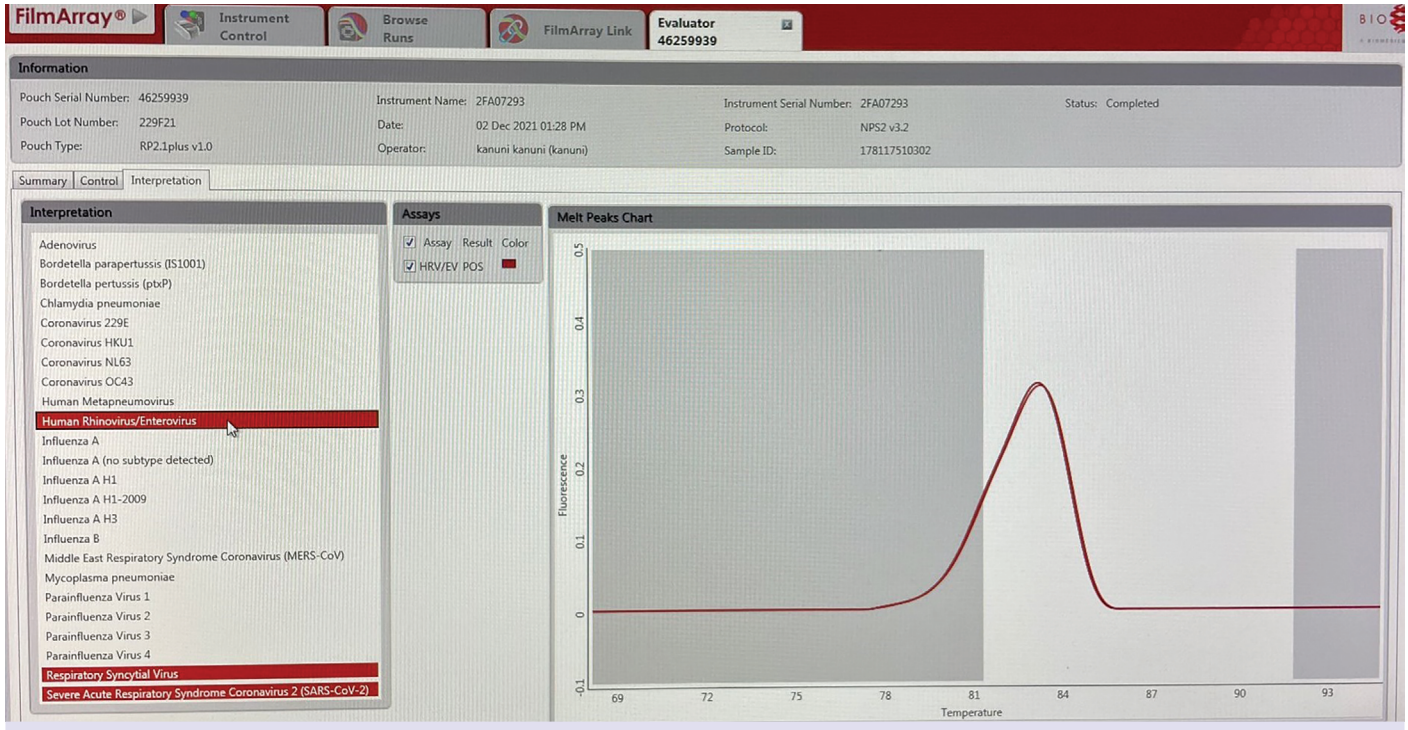

FIGURE 2. Triple coinfection peak of RSV.

cases, Corona OC43;12 cases, Adeno virus; 21 cases, H. metapneumovirus; 21 cases, influenza A; 22 cases. The number of negative detected cases is 40. When coinfection is evaluated; 32.5% (n=80) in dual coinfection; triple coinfection 6.8% (n=18), dual coinfection with COVID-19:5.7% (n=15) triple coinfection with COVID-19:4.5% (n=12). Peaks of a patient with triple coinfection are visible in Figures 1–3. Figure 1;

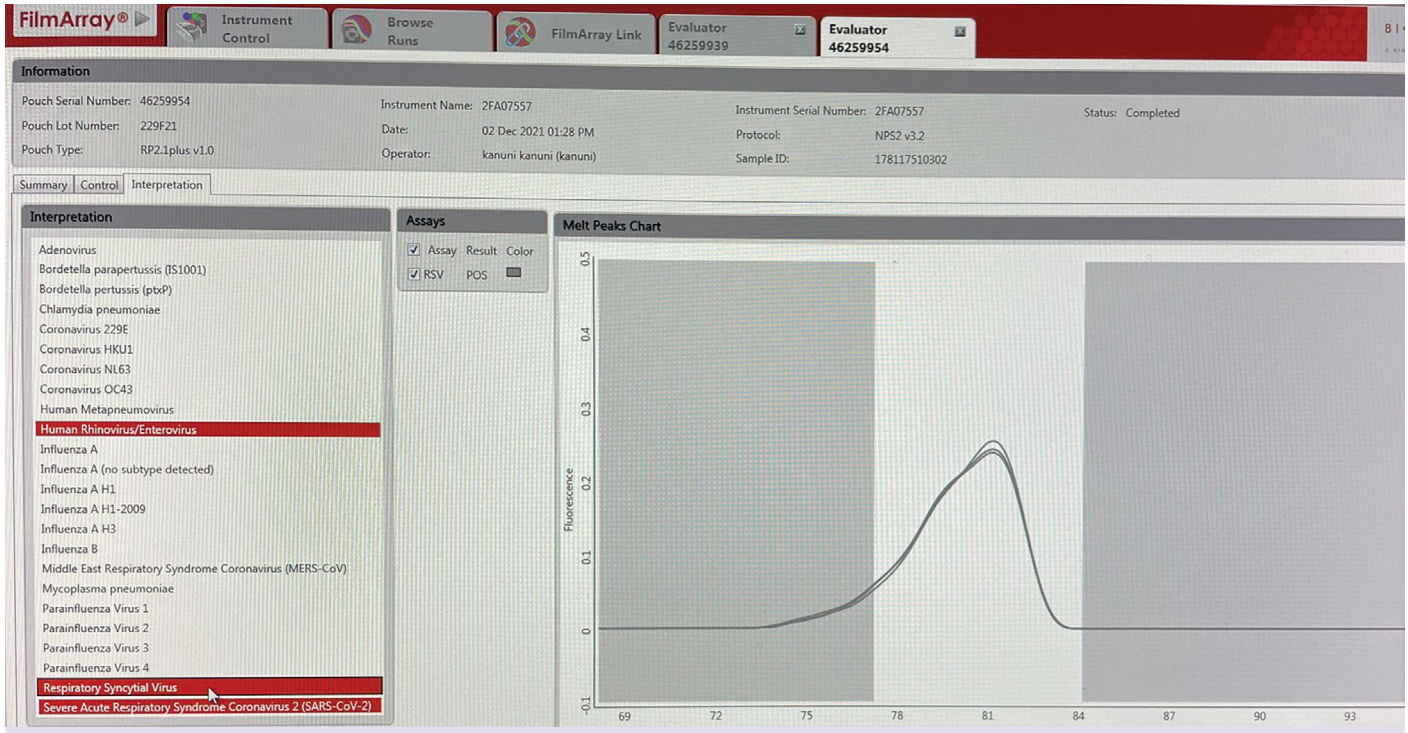

FIGURE 3. Triple coinfection peak of Rhino/entero virus.

TABLE 1. Distribution of viral agents by gender, age, season and clinic

	Gender		Season		Age				Clinic	
	Woman	Man	Autumn	Winter	0–2 age	3–10 age	11–18 age	19< age	Upper respiratory tract	Lower respiratory tract
RSV	71	48	54	65	107	8	3	1	76	43
H. metapneumovirus	15	11	0	26	23	2	0	1	16	10
Parainfluenza	17	13	23	7	21	7	1	1	22	8
COVID-19	53	43	47	49	49	11	9	27	76	20
Corona 229E	2	2	2	2	2	0	0	2	3	1
Corona NL63	2	1	0	3	2	1	0	0	3	0
Corona HKU	1	0	1	0	0	1	0	0	0	1
Corona CO43	10	2	12	0	2	2	5	3	9	3
Adeno virus	15	6	3	18	17	2	1	1	13	8
Influenza A	14	8	1	21	12	6	1	3	16	6
Rhino/entero	90	65	62	93	116	34	1	4	100	55

RSV: Respiratory syncytial virus.

peak of COVID-19, Figure 2; peak of RSV, and Figure 3; peak of rhino/entero virüs. The distribution of cases is given in Tables 1 and 2. Gender has no effect on the distribution of viral infections ($p>0.05$), RSV, COVID-19, Rhino/entero virus did not show a significant difference in autumn and winter, H. metapneumovirus, adeno, influenza A was significantly higher in winter, parainfluenza and Corona OC43 were significantly higher in autumn ($p<0.05$). When age is evaluated, RSV is significantly more common under 2 years

TABLE 2. Distribution of coinfections

Binary coinfections	n	Triple coinfections	n
RSV+Rhino/entero	35	Paraifluenza+Corona OC43+Rhino/entero	2
RSV+Parainfluenza	2	COVID-19+RSV+Rhino/entero	5
RSV+COVID-19	3	H. Metapneumo+COVID-19+RSV	1
RSV+Adeno	2	Paraifluenza+Adeno+Rhino/entero	1
RSV+Influenza A	1	Paraifluenza+Influenza A+Rhino/entero	1
RSV+H. metapneumo	1	Paraifluenza+SARS CoV2+Rhino/entero	2
H. metapneumo+Influenza A	1	COVID-19+Adeno+Rhino/entero	3
H. metapneumo+Rhino/entero	3	RSV+Parainfluenza+Corona 229E	1
H. metapneumo+Corona 229E	1	RSV+Adeno+Influenza A	1
COVID-19+Influenza A	1	RSV+Adeno+ Rhino/Entero	1
COVID-19+Rhino/entero	10	RSV+Parainfluenza+Rhino/entero	1
COVID-19+Corona OC43	1	H. metapneumo+ COVID-19+Adeno	1
Adeno+Influenza A	1	H. metapneumo+Adeno+Rhino/entero	1
Adeno+Rhino/entero	4	H. metapneumo+COVID-19+Rhino/entero	2
Influenza A+Rhino/entero	3		
Parainfluenza+Rhino/entero	10		
Rhino/entero+Corona HKU1	1		
Total	80		18

RSV: Respiratory syncytial virus.

of age, rhino/entero virus is significant at 3 years old and above, Corona OC43 is significant in 11–18 age group, COVID-19 is significant in 19 years old and above ($p<0.05$). When evaluated in terms of clinical upper and lower respiratory tract infections, while there was no significant difference in other viruses, admission with COVID-19 upper respiratory tract infection symptoms was found to be significantly higher ($p<0.05$) (Table 3) evaluation of the effects of other viral agents on season, gender, age, and clinical COVID-19 Logistic regression analysis (Table 4).

DISCUSSION

There are limited data on coinfection since the beginning of the pandemic. In our study, in addition to dual coinfections, triple coinfections were detected especially in newborns and children (Table 3). In COVID-19 infection, lymphocytopenia, overexpression of inflammatory cytokines, and dysfunction of the acquired immune system may predispose to the development of coinfection, which may result in harmful sequelae in a healthy individual [3, 6]. Given the fact that more than one mechanism is involved in the emergence of coinfections, it can be assumed that the mere presence of one virus and its effect on the immune system may serve as the basis for the replication and suppression of the other virus [6, 7].

Coinfection can be detected at the first application of the disease as well as later. A nasopharyngeal swab was taken for a respiratory pathogen panel as well as COVID-19 RT-PCR in a patient who presented to the emergency department with cough and shortness of breath. In a study, the respiratory pathogen panel was found to be Human metapneumovirus positive and the patient was treated and discharged. COVID-19 RT PCR was positive after 24 hours. This case reflects that COVID-19 testing algorithms that exclude patients who routinely test positive for viral pathogens may miss patients co-infected with COVID-19 [8].

The distribution of endemic viruses differs from region to region. From June 2020 to January 2021 in India, other respiratory pathogens were detected in 33 of 191 patients with COVID-19. The coinfection rate of these, with human adenovirus and human rhinovirus being the most common, was found to be 7.3% [3].

Table 3. The relationship of viral infections with gender, season, age and clinic

	Gender	Season	Age		Clinic
RSV					
Chi-square	0.113	0.176	42.888		2.045
Sig.	0.737	0.675	0.000*	2> age significant	0.153
H. metapneumovirus					
Chi-square	0.006	21.878	5.934		0.693
Sig.	0.937	0.000*	0.115		0.405
Parainfluenza					
Chi-square	0.042	14.357	3.362		0.311
Sig.	0.838	0.000*	0.339		0.577
COVID-19					
Chi-square	0.561	1.410	43.425		6.558
Sig.	0.454	0.235	0.000*	19< age significant	0.010*
Corona CO43					
Chi-square	3.172	15.921	35.571		0.221
Sig.	0.075	0.000*	0.000	11–18 age significant	0.638
Adeno virus					
Chi-square	1.553	7.907	2.015		0.497
Sig.	0.213	0.005*	0.569		0.481
Influenza A					
Chi-square	0.262	14.693	2.730		0.167
Sig.	0.609	0.000*	0.435		0.683
Rhino/entero					
Chi-square	0.015	1.627	39.351		2.370
Sig.	0.903	0.202	0.000*	3<age significant	0.124

Results are based on nonempty rows and columns in each innermost subtable. *: The Chi-square statistic is significant at the, 05 level; RSV: Respiratory syncytial virus.

In a study conducted in China, Mycoplasma and RSV were the most common coinfections in patients with COVID-19 pneumonia and were seen among rhino/entero, RSV, and other coranavirus species. Increased procalcitonin levels in patients with COVID-19 pneumonia have been associated with coinfection [9].

Coinfections in patients infected with COVID-19 in the greater metropolitan area of New York City have been identified as common respiratory viruses co-occurring with rhinovirus/enterovirus, Influenza viruses and coronavirus NL63 and other coronavirus family in the community. Additional studies are needed to determine whether concomitant viral infection could potentially lead to viral interference or affect disease outcomes in COVID-19 patients [10].

In our study, rhino/entero, RSV, parainfluenza, coranavirus OC43 emerged predominantly in the autumn of 2021 in Istanbul. Influenza A was seen in one case. It was observed that rhinovirus, RSV, coronavirus OC43, and parainfluenza coinfections seen with COVID-19 did not increase mortality and morbidity when compared to other patients. In other publications, there are data showing that influenza B, which increases mortality mostly by influenza A, increases chronicity [11, 12]. In our study, it was determined that the risk of co-occurrence of influenza A and COVID-19 is higher than other viruses. However, a clinical interpretation could not be made. Influenza B was not seen. It has been determined that influenza coinfection can trigger pus with COVID-19 hyperinflammatory state and cardiac effects are more. However, it may be difficult to detect COVID-19 mortality differences as the available data are only for critically ill patients [13, 14]. Studies have found that coinfections are more common than expected [10]. Currently, we

Table 4. Evaluation of the effects of other viral agents on season, gender, age, and clinical COVID-19 logistic regression analysis

	B	SE	Wald	Sig.	ODDS	95% CI for EXP(B)	
						Lower	Upper
Step 1							
RSV(1)	2.576	0.447	33.280	0.000	13.146	5.479	31.544
H. metapneumovirus(1)	0.966	0.556	3.018	0.082	2.627	0.884	7.808
Parainfluenza (1)	1.644	0.693	5.618	0.018	5.174	1.329	20.143
Adeno virus(1)	0.601	0.654	0.844	0.358	1.824	0.506	6.577
Influenza A(1)	3.605	1.095	10.834	0.001	36.794	4.299	314.881
Rhinoentero(1)	1.738	0.359	23.473	0.000	5.684	2.814	11.481
Seasons (1)	-0.445	0.361	1.519	0.218	0.641	0.315	1.301
(0–2) age			6.791	0.079			
(3–10) age	-1.051	0.515	4.165	0.041	0.350	0.127	0.959
(11–18) age	-1.364	0.589	5.366	0.021	0.256	0.081	0.811
(19<) age	-1.354	0.658	4.233	0.040	0.258	0.071	0.938
Gender(1)	-0.127	0.296	0.184	0.668	0.881	0.493	1.574
Clinic(1)	0.648	0.346	3.507	0.061	1.911	0.970	3.765
Constant	-9.728	1.851	27.635	0.000	0.000		

Variable(s) entered on step 1: Gender, RSV, H. metapneumovirus, parainfluenza, adeno virus, influenzaa, rhino/entero, season, age, clinic. SE: Standart error; ODDS: Risk ratio (OR); CI: Confidence interval; EXP: Exponansiyel beta.

have little understanding of the viral kinetic parameters of COVID-19 infection, which makes it difficult to determine the dynamics of coinfection. It has been shown that viruses trigger competitive advantage between different mechanisms by which immune responses are elicited [10].

Studies have also shown that clinical symptoms and transmission dynamics are quite similar in patients with only COVID-19 infection and patients with Influenza virus coinfection [15, 16]. It has been found that bacterial coinfections are more mortal than viral coinfections [17].

EBV should also be investigated in high-risk groups such as immunocompromised and transplanted patients. EBV coinfection causes an increase in mortality [18]. Severe lymphocytopenia occurs in severe COVID-19 infection, leading to cellular immune system deficiencies and consequent CMV re-infection or reactivation. Subsequently, disruption of the reticuloendothelial and hematopoietic systems such as the bone marrow, spleen, and lymph nodes may result in the death of T cells in severe COVID-19. In this context, as the population of effector and CD8+ cytotoxic T cells decreases, the effects of COVID-19 infection become more serious, leading to innate and adaptive immune system failure, and ultimately predisposing individuals to viral infections [19–21].

COVID-19 and adenovirus coinfection may predispose the patient to mechanical ventilation by initiating a series of deleterious sequelae such as lymphopenia, thrombocytopenia, and septic shock. To date, coinfection of adenovirus and COVID-19 has been reported in limited numbers [22–24]. In addition, coinfections increase the spread of viruses. While the period of spread of the virus in influenza-related diseases is 5–10 days, it is 2–5 weeks in COVID-19 infection [25, 26].

A better understanding of the prevalence of coinfection with other respiratory pathogens and the profile of the pathogens in COVID-19 patients may contribute to effective patient management and management of therapy during the current pandemic [27].

The inadequacy of traditional methods in detecting coinfection and the lack of sufficient evidence may lead to underdiagnosis [3]. In patients with underlying disease, coinfection should be investigated [28].

Conclusion

As can be seen, coinfections are substantial and their presence increases the duration of transmission. Mechanisms of action differ according to the difference of viruses, and mortality and morbidity change. It is also necessary to develop new strategies to better understand the clinical manifestations of coinfections and explore appropriate treatment options for them. Accordingly, early detection of coinfections is important because of differences in treatment and favorable prognosis. It should be routinely investigated in newborns and children, especially in the case of immunodeficiency, and the treatment should be planned accordingly. In our study, it was determined that the risk of COVID-19 coinfection with the increase of influenza A virus in winter may be higher than other viruses. Therefore, the risk group should be included in the influenza a v accine p rogram, which is revised every year. Further studies are needed in terms of underlying mechanisms and treatment protocols.

Ethics Committee Approval: The Health Sciences University Kanuni Sultan Suleyman Training and Research Hospital Clinical Research Ethics Committee granted approval for this study (date: 14.10.2021, number: 2021.10.252).

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REFERENCES

1. Pyrc K, Dijkman R, Deng L, Jebbink MF, Ross HA, Berkhout B, et al. Mosaic structure of human coronavirus NL63, one thousand years of evolution. J Mol Biol 2006;364:964–73. [CrossRef]
2. Chaung J, Chan D, Pada S, Tambyah PA. Coinfection with COVID-19 and coronavirus HKU1-The critical need for repeat testing if clinically indicated. J Med Virol 2020;92:1785–6. [CrossRef]
3. Aghbash PS, Eslami N, Shirvaliloo M, Baghi HB. Viral coinfections in COVID-19. J Med Virol 2021;93:5310–22. [CrossRef]
4. Ozaras R, Cirpin R, Duran A, Duman H, Arslan O, Bakcan Y, et al. Influenza and COVID-19 coinfection: Report of six cases and review of the literature. J Med Virol 2020;92:2657–65. [CrossRef]
5. Rodriguez JA, Rubio-Gomez H, Roa AA, Miller N, Eckardt PA. Co-infection with SARS-COV-2 and parainfluenza in a young adult patient with pneumonia: Case Report. IDCases 2020;20:e00762. [CrossRef]
6. Sakamoto H, Ishikane M, Ueda P. Seasonal influenza activity during the SARS-CoV-2 outbreak in Japan. JAMA 2020;323:1969–71. [CrossRef]
7. D'Abramo A, Lepore L, Palazzolo C, Barreca F, Liuzzi G, Lalle E, et al. Acute respiratory distress syndrome due to SARS-CoV-2 and Influenza A co-infection in an Italian patient: Mini-review of the literature. Int J Infect Dis 2020;97:236–9. [CrossRef]
8. Touzard-Romo F, Tapé C, Lonks JR. Co-infection with SARS-CoV-2 and human metapneumovirus. R I Med J (2013) 2020;103:75–6.
9. Tang ML, Li YQ, Chen X, Lin H, Jiang ZC, Gu DL, et al Co-infection with common respiratory pathogens and SARS-CoV-2 in patients with COVID-19 pneumonia and laboratory biochemistry findings: a retrospective cross-sectional study of 78 patients from a single center in China. Med Sci Monit 2021;27:e929783. [CrossRef]
10. Nowak MD, Sordillo EM, Gitman MR, Paniz Mondolfi AE. Coinfection in SARS-CoV-2 infected patients: Where are influenza virus and rhinovirus/enterovirus? J Med Virol 2020;92:1699–700. [CrossRef]
11. Hashemi SA, Safamanesh S, Ghafouri M, Taghavi MR, Mohajer Zadeh Heydari MS, Namdar Ahmadabad H, et al. Co-infection with COVID-19 and influenza A virus in two died patients with acute respiratory syndrome, Bojnurd, Iran. J Med Virol 2020;92:2319–21. [CrossRef]
12. Yue H, Zhang M, Xing L, Wang K, Rao X, Liu H, et al. The epidemiology and clinical characteristics of co-infection of SARS-CoV-2 and influenza viruses in patients during COVID-19 outbreak. J Med Virol 2020;92:2870–3.
13. Suwanwongse K, Shabarek N. Can coinfection with influenza worsen COVID-19 outcomes? J Investig Med High Impact Case Rep 2020;8:2324709620953282. [CrossRef]
14. Sarkar S, Khanna P, Singh AK. Impact of COVID-19 in patients with concurrent co-infections: A systematic review and meta-analyses. J Med Virol 2021;93:2385–95. [CrossRef]
15. Lai CC, Liu YH, Wang CY, Wang YH, Hsueh SC, Yen MY, et al. Asymptomatic carrier state, acute respiratory disease, and pneumonia due to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2): Facts and myths. J Microbiol Immunol Infect 2020;53(3):404–12. [CrossRef]
16. Chow EJ, Doyle JD, Uyeki TM. Influenza virus-related critical illness: prevention, diagnosis, treatment. Crit Care 2019;23:214. [CrossRef]
17. Sharov KS. SARS-CoV-2-related pneumonia cases in pneumonia picture in Russia in March-May 2020: Secondary bacterial pneumonia and viral co-infections. J Glob Health 2020;10:020504. [CrossRef]
18. Roncati L, Lusenti B, Nasillo V, Manenti A. Fatal SARS-CoV-2 coinfection in course of EBV-associated lymphoproliferative disease. Ann Hematol 2020;99:1945–6. [CrossRef]
19. Zheng M, Gao Y, Wang G, Song G, Liu S, Sun D, et al. Functional exhaustion of antiviral lymphocytes in COVID-19 patients. Cell Mol Immunol 2020;17:533–5. [CrossRef]
20. Maucourant C, Filipovic I, Ponzetta A, Aleman S, Cornillet M, Hertwig L, et al; Karolinska COVID-19 Study Group. Natural killer cell immunotypes related to COVID-19 disease severity. Sci Immunol 2020;5:eabd6832.
21. Jeannet R, Daix T, Formento R, Feuillard J, François B. Severe COVID-19 is associated with deep and sustained multifaceted cellular immunosuppression. Intensive Care Med 2020;46:1769–71. [CrossRef]
22. Zhu X, Ge Y, Wu T, Zhao K, Chen Y, Wu B, et al. Co-infection with respiratory pathogens among COVID-2019 cases. Virus Res 2020;285:198005.
23. Lin D, Liu L, Zhang M, Hu Y, Yang Q, Guo J, et al. Co-infections of SARS-CoV-2 with multiple common respiratory pathogens in infected patients. Sci China Life Sci 2020;63:606–9. [CrossRef]
24. Kim D, Quinn J, Pinsky B, Shah NH, Brown I. Rates of co-infection between SARS-CoV-2 and other respiratory pathogens. JAMA 2020;323:2085–6.
25. Fang Z, Zhang Y, Hang C, Ai J, Li S, Zhang W. Comparisons of viral shedding time of SARS-CoV-2 of different samples in ICU and non-ICU patients. J Infect 2020;81:147–78. [CrossRef]
26. Jin CC, Zhu L, Gao C, Zhang S. Correlation between viral RNA shedding and serum antibodies in individuals with coronavirus disease 2019. Clin Microbiol Infect 2020;26:1280–2. [CrossRef]
27. Sreenath K, Batra P, Vinayaraj EV, Bhatia R, SaiKiran K, Singh V, et al. Coinfections with other respiratory pathogens among patients with COVID-19. Microbiol Spectr 2021;9:e0016321. [CrossRef]
28. Danley K, Kent P. 4-month-old boy coinfected with COVID-19 and adenovirus. BMJ Case Rep 2020;13:e236264. [CrossRef]