NCT
Neumología y Cirugía de Tórax

COVID-19 SERIES REVIEW

Similarities and differences between influenza and COVID-19

Similitudes y diferencias entre la influenza y la COVID-19

José R. Pérez-Padilla[1], Joel A. Vázquez-Pérez[1], Lila M. Cordero-Silis[2], Patricia A. Volkow-Fernández[3], Carmen Hernández-Cárdenas[4] and Gustavo Lugo-Goytia[4]*

[1]Departamento de Investigación en Tabaquismo y Enfermedad Pulmonar Obstructiva Crónica, Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas; [2]Centro Médico Nacional 20 de Noviembre, Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado; [3]Instituto Nacional de Cancerología; [4]Departamento de Áreas Críticas, Instituto Nacional de Enfermedades Respiratorias Ismael Cosío Villegas. Mexico City, México

Abstract

Influenza has caused repeated pandemics throughout human history, the last one in 2009, with a lower mortality impact than that reported in 1918, partly due to the availability of antivirals and a useful vaccine against this new virus within a few months of the pandemic being declared, applied the most vulnerable populations, as well as to intensive care units, and antibiotics administered to manage patients with complicated bacterial superinfections. More recently, in 2019, the COVID-19 pandemic emerged, much more aggressive and lethal than the 2009 influenza pandemic, with greater systemic involvement, vascular and thrombogenic damage, and more sequelae, at a time when there were no useful antivirals or vaccines, but with better care for critically ill patients. Both the influenza virus and SARS-CoV-2 are RNA viruses that primarily affect the respiratory system and require assistance for respiratory failure and the need for mechanical ventilation and critical care. After pandemics, isolated cases of both COVID-19 and influenza continue to occur, but seasonal outbreaks also need to be taken into account to boost immunity with updated vaccines and to emphasize respiratory precautions.

Keywords: *Influenza. SARS-CoV-2. COVID-19. Pandemic.*

Resumen

La influenza ha causado pandemias repetidas a lo largo de la historia de la humanidad, la última en 2009, con menor impacto en mortalidad que la de 1918, en parte por la existencia de antivirales y una vacuna útil contra este nuevo virus a los pocos meses de declarada la pandemia, aplicada a las poblaciones más vulnerables, así como de unidades de terapia intensiva y antibióticos para el manejo de los pacientes complicados con sobreinfecciones bacterianas. Más recientemente, en 2019, apa reció la pandemia de COVID-19, mucho más agresiva y letal que la de influenza de 2009, con más afectación sistémica, daño vascular y trombogénico, y más secuelas, sin antivirales útiles y sin vacuna, pero con mejores cuidados de los pacientes críticos. Tanto el virus de la influenza como el SARS-Co-V2 son virus de RNA, que primordialmente afectan el aparato respiratorio y obligan a la asistencia por falla respiratoria con necesidad de ventilación mecánica y cuidados críticos. Después de las pande mias, se siguen presentando casos aislados tanto de COVID-19 como de influenza, pero también brotes estacionales que deben tomarse en cuenta para reforzar la inmunidad con vacunas actualizadas, además de insistir en las precauciones respiratorias.

Palabras clave: *Influenza. SARS-CoV-2. COVID-19. Pandemia.*

Correspondence:
José R. Pérez-Padilla
E-mail: perezpad@gmail.com

Date of reception: 16-05-2025
Date of acceptance: 03-06-2025
DOI: 10.24875/NCTE.M26000010

Available online: 03-02-2026
Neumol Cir Torax (Eng). 2025;84(1):69-76
www.revistanct.org.mx

Introduction and general concepts

Influenza and COVID-19 are viral infections that primarily affect the respiratory system, are highly contagious, have caused pandemics, and share many characteristics.

Virus

Considering the biology of influenza viruses and coronaviruses, characteristics they share and those that distinguish them can be enumerated (Table 1). The influenza virus belongs to the *Orthomyxoviridae* family and has a segmented single-stranded RNA genome. This is one major difference between the two viruses, since coronaviruses (*Coronaviridae* family) have a single-stranded, linear RNA genome, that is, a single continuous strand; in fact, it is among the RNA viruses with the largest number of nucleotides, approximately 30,000. The influenza virus RNA is of negative polarity and requires copying its information to be translated, while coronavirus RNA is of positive polarity and can be immediately translated by the cellular machinery for the production of viral proteins. According to this, their replication cycles within the target cell show differences mainly in transcription and translation.

Cellular tropism (affinity for a specific cell lineage) is defined by the binding of the outermost proteins; in the case of influenza virus, by hemagglutinin (HA), and in coronaviruses, by the spike (S) protein. The presence of mutations in the S protein of the different SARS-CoV-2 variants has been linked to lower effectiveness of antibodies induced by vaccination or infection, which would explain, for example, the outbreaks caused by the delta variant B.1.617.2, with a high viral load, greater transmissibility, and more clinical severity[1]. The influenza virus binds to N-acetylneuraminic acid or sialic acid molecules (α-2-3 and α-2-6) that are found in different mucous membranes, mainly in the upper and lower respiratory tracts. In contrast, SARS-CoV-2 binds to the angiotensin-converting enzyme 2 receptor, which is present in more organs and tissues, thus producing a widespread infection. During the pandemic, SARS-CoV-2 showed changes in its viral tropism as new variants emerged, with an initial affinity for the olfactory epithelium associated with anosmia, and in the case of omicron, a preference for the respiratory epithelium, which resulted in milder symptoms. These variations affect pathogenicity and transmissibility, which underscores the importance of continuous genomic and epidemiological surveillance[2].

Table 1 summarizes the main characteristics of both viruses.

Viral disease

Both infections cause a flu-like syndrome, with fever, cough, dyspnea, fatigue, sore throat, cold, muscle aches, and headache, and can even generate vomiting and diarrhea. Diseases caused by these two viruses, and others, can present a range of severity from asymptomatic infection to death from respiratory and multiorgan failure; however, the trend in SARS-CoV-2, at least in its initial phases, was toward greater severity, longer hospital stays, and greater contagiousness, especially in elderly patients or those with comorbidity. The incubation period, although variable, tends to be shorter in influenza; the infected person is contagious from before the onset of symptoms, at least for 1-2 days, and in both infections, viable virus can continue to be excreted, on average, for 1 week in influenza and 10 days in COVID-19, and for longer periods, viral fragments or incomplete non-infectious viruses can continue to yield positive results in diagnostic tests such as polymerase chain reaction (PCR). Immunocompromised patients or those with severe disease can continue excreting the virus and transmitting it for longer periods.

During the COVID-19 pandemic, as in other pandemics, so-called superspreaders were identified, people who transmit the virus to dozens of others, perhaps due to a very high viral load in respiratory secretions and a very efficient expulsion of small particles, combined with lack of protection and close proximity in a confined or poorly ventilated area, or influenced by air currents. It is unlikely that these episodes are due solely to the characteristics of the individual and the virus, and it is expected that favorable environmental conditions are also required. Children are at higher risk of contracting influenza and of the infection being symptomatic, compared to COVID-19, while the latter can develop, although rarely, into a multisystem inflammatory syndrome, a serious condition with considerable mortality.

Both infections can present complications in various organs or systems, in addition to respiratory failure, which include exacerbations of chronic lung diseases, cardiac lesions, renal fa ilure, multi-organ failure, myositis, myocarditis, pericarditis, cerebral inflammation, and exacerbations of chronic non-pulmonary diseases; furthermore, in COVID-19, the predisposition to develop pulmonary, cerebral, or coronary thromboembolism is notable.

Table 1. Viral and epidemiological characteristics of influenza virus and SARS-CoV-2 (COVID-19)

Characteristics	Influenza	SARS-CoV-2 (COVID-19)
RNA genome	Negative strand, segmented (8)	Positive strand RNA, linear
Number of nucleotides	13,000	30,000
Proteins	10	29
Receptor binding	Hemagglutinin (HA)	Spike (S)
Cellular receptor	Sialic acid (α 2-6, α 2-3)	Angiotensin-converting enzyme 2
Tropism	Epithelial cells of the upper and lower respiratory tracts	Nasal epithelial cells (secretory and ciliated cells), respiratory epithelia
Incubation (days, average)	3	6
Presymptomatic transmission (days)	3-5	1-2
Transmission by fomites and droplets	Yes; possible from fine aerosols, but less common	Yes, primarily; possible from fine aerosols, but less common
R0*	1.3	2-2.5
Children as vectors	Yes	Less infection (at least symptomatic) and are less relevant vectors
Proportion of severe cases	*	15*
Critical cases	*	5*
Case fatality rate of cases demonstrated by PCR	0.1	3-4%*
Infection fatality rate/100,000 by serology	1-10 (0.01%)	500-1000 (1%), 1.46% in New York
Asymptomatic infections	50%	18-40%

*Varies according to the phase of the outbreak, the denominator used (serologically infected individuals, which includes asymptomatic cases, yielding a lower proportion, versus PCR-confirmed cases) and the control measures implemented.
PCR: polymerase chain reaction.

On the other hand, both infections generate pressure on the hospital system due to an increase in nosocomial infections, mainly ventilator-associated pneumonia and bacteremia with higher mortality rates, as a consequence of the saturation of severely ill patients in hospitals that produce significant work overloads, as well as the need to hire new personnel insufficiently trained in preventive practices, and adverse situations worsened by the accelerated consumption of preventive supplies with frequent risk of shortages or complete unavailability.

For influenza, several highly effective antivirals are available, especially at the onset of the disease, when viral replication is most active. Oseltamivir is an oral drug with good bioavailability. If administered early, it prevents severe disease, acute respiratory distress syndrome (ARDS), and death. The results of available antivirals against SARS-CoV-2 have not shown the same efficacy; they are of limited usefulness, which consists mainly of reducing hospital stays when administered intravenously in hospitalized patients in the case of remdesivir or of low help in terms of the risk of hospitalization and complications in outpatient patients with advanced age, immunosuppression, or risk comorbidity. The combination of nirmatrelvir and ritonavir administered orally has been shown to be effective in patients with mild to moderate COVID-19, achieving a reduction in the risk of hospitalization and intensive care unit admission, as well as all-cause mortality; however, the frequent interactions of ritonavir with medications commonly used in patients with comorbidities may limit its use[3]. The use of monoclonal antibodies administered parenterally reduces the risk of severe disease, but their cost and administration schedule make them of limited utility in a pandemic scenario in most countries, coupled with the rapid obsolescence of products due to frequent viral mutations.

In patients with influenza pneumonia without other respiratory comorbidity, the use of systemic steroids is controversial, and the American Thoracic Society and the Infectious Diseases Society of America recommend against their use regardless of disease severity. This contrasts with solid evidence supporting their use in the treatment of COVID-19 in patients with hypoxemia, according to National Institutes of Health guidelines[4].

Brief history of the influenza A H1N1 and COVID-19 pandemics

Influenza A subtype H1N1 was first reported in Mexico in April 2009[5] and was the first pandemic of the 21st century that was associated with severe pneumonia and ARDS, with an associated hospital mortality of 25-41% depending on the country and hospital[6]. The influenza virus is a significant cause of disease and death each year, with variable numbers depending on the virus strain, but an average of 390,000 deaths per year has been reported[7]. The World Health Organization (WHO) considers that influenza can affect 5-10% of adults and up to 20-30% of children and causes complications especially at the extremes of life and in the presence of immunosuppression and other comorbidities[8].

The respiratory infection outbreak by SARS-CoV-2 began in December 2019, in Wuhan, China[9,10], and spread to all countries of the world as a severe pandemic[11]. As of June 30, 2021, nearly 182 million cases and 4 million deaths had been reported worldwide[11], mainly from respiratory failure, although it has accumulated a long list of complications and effects on various organs and systems. COVID-19 is also associated with severe pneumonia and acute respiratory distress syndrome (ARDS), with high requirements for critical care and respiratory support in approximately 5% of cases, and with mortality that varied between 30% and 60% in different sites (average of 41%)[12].

Diagnosis of influenza virus and SARS-CoV-2 infections

The diagnosis of influenza is usually based on clinical presentation, although virological confirmation is recommended to guide treatment and epidemiological surveillance. Diagnostic methods include antigen tests and RT-qPCR, the latter with high sensitivity and specificity, in addition to allowing viral subtyping[13]. For SARS-CoV-2, RT-qPCR has also been the reference method since the beginning of the pandemic, but its use requires greater investment and trained personnel[14]. Therefore, rapid antigen tests were developed, some combined with influenza A/B diagnosis, which are useful in early phases despite their lower sensitivity[15].

Serological tests have also been used, being useful in late stages, as well as novel technologies such as LAMP (loop-mediated isothermal amplification), NGS (new generation sequencing), and CRISPR (clustered regularly interspaced short palindromic repeats). Currently, RT-qPCR remains the reference technique for both infections, complemented by multiple PCR panels that allow detection of several respiratory viruses in a single sample, optimizing resources in clinical settings especially with high frequency of immunosuppressed patients or those with multimorbidity, as well as for epidemiological surveillance.

Comparison between the characteristics of influenza and COVID-19

Table 1 shows the main characteristics of both viruses and the diseases they cause, although the comparison is to some extent unfair because influenza includes both seasonal and pandemic forms, the latter being much more aggressive in general, while the COVID-19 data presented derive from the initial cases of the pandemic, when general immunity was absent or almost absent. Among influenza pandemics, there have been some mild ones, such as that caused by influenza A H1N1 virus in 2009, and at the other extreme the 1918 pandemic, which caused between 20 and 60 million deaths, many more than those COVID-19 has caused so far, although in an era lacking antibiotics, effective vaccines for the virus, rapid diagnostic tests, and intensive care therapies.

It is clear that both primarily affect the respiratory system and that the main cause of death is respiratory failure and its complications. On the other hand, both diseases can also produce multisystemic involvement, more frequently in COVID-19, especially regarding endothelial damage and the tendency toward coagulation that led to the recommendation to offer full anticoagulation in subjects at higher risk or with risk markers for thrombosis.

The symptoms and radiological findings are very similar, with pneumonia being highly frequent in patients diagnosed with COVID-19. From the clinical point of view, much emphasis has been placed on the higher frequency of anosmia in COVID-19, which also occurs, but much more rarely, in influenza and other viral infections of the upper airway that affect the nasal mucosa.

Table 2. Comparison of some characteristics of patients with acute respiratory distress syndrome due to influenza A H1N1 (winter 2019-2020) and COVID-19 (spring 2020)

Characteristics	COVID-19 (n = 147)	Influenza A H1N1 (n = 94)	p
Age, years	45 ± 11	47 ± 11	0.147
Fever	129 (87.7)	92 (98)	0.005
Dyspnea	125 (85)	94(100)	< 0.001
Myalgia	81 (55.1)	79 (84)	< 0.001
Headache	72 (48.9)	63 (67)	0.005
Leukocytes/µl	10.9 ± 4.6	9.1 ± 4.7	0.004
Lymphocytes/µl	978 ± 738	897 ± 475	0.35
Platelets × 10^3/µl	269 ± 105	200 ± 85	0.001
Creatinine (0.7-1.2 mg/dl)	1.28 ± 1.17	1.37 ± 1.36	0.61
LDH (98-192 IU) IU	468 ± 184	712 ± 366	0.001
Glucose (mg/dl)	140 ± 46	184 ± 97	0.001
Mean arterial pressure (mmHg)	80 ± 10	70 ± 8	< 0.001
Heart rate (min^{-1})	82 ± 15	98 ± 14	< 0.001
Use of norepinephrine	55 (37.4)	56 (59.5)	< 0.001
PaO_2/FiO_2 (mmHg)	143 ± 58	96 ± 43	< 0.001
PEEP (cmH_2O)	11 ± 2	12 ± 3	0.001
Plateau respiratory pressure (cmH_2O)	28 ± 6	26 ± 4	0.01
Days in prone position ventilation	3.7 ± 4.1	2.1 ± 3.2	0.002
Pulmonary compliance (ml/cmH_2O)	26 ± 12	30 ± 9	0.002
SOFA score	5.2 ± 2.3	6.4 ± 2.0	< 0.001
Ventilatory quotient	2.4 ± 0.7	2.3 ± 0.5	0.25

Comorbidity was similar in both groups, except for chronic kidney failure.
LDH: lactate dehydrogenase; FiO_2: fraction of inspired oxygen; PaO_2: partial pressure of oxygen; PEEP: positive end-expiratory pressure; SOFA: Sepsis-related Organ Failure Assessment.
Modified from Hernández-Cárdenas et al.[18].

Computed tomography findings are very similar in both infections, with ground-glass opacities[16]. COVID-19 patients have often shown vascular inflammation and immunothrombosis[17], to a greater degree than those with influenza, with a requirement for thrombosis prophylaxis in hospitalized patients. Opacities are usually located peripherally and predominantly affect the lower lobes, and furthermore, the so-called "crazy-paving" pattern predominates, and they more frequently tend to consolidate. Furthermore, in COVID-19 patients, vascular engorgement, pleural thickening, and subpleural lines have been described, while in influenza cases, complications such as pneumomediastinum and pneumothorax have been reported[16].

Comparative lethality between seasonal influenza and COVID-19

It is of interest to evaluate the lethality of ARDS due to COVID-19 and influenza A H1N1, adjusting for known prognostic risk factors. At the National Institute of Respiratory Diseases (INER) in Mexico, a comparative analysis was conducted in a group of patients with influenza A H1N1 hospitalized between October 2019 and February 2020, before the COVID-19 epidemic, with those of the first COVID-19 outbreak (from March 2020 to October 2020) admitted to the same unit a few months later, all with positive RT-PCR test and with respiratory failure that required intensive care[18],

Table 3. Some characteristics of epidemic infections by influenza viruses and coronaviruses

Infections	R0*	Lethality	Incubation (days)	Hospitalized	Community spread	Infected
Influenza	1.3	0.05-0.1	1-4	2%	10-20%	1000 million
COVID-19	2-2.5	3.4	4-14	19%	30-40%	?
SARS	3	10	2-7	Most	10-60%	8000
MERS	0.3-0.8	34	6	Most	4-13%	420

*R0 is not fixed and changes during an outbreak, especially if protective measures are taken.
MERS: Middle East respiratory syndrome; SARS: severe acute respiratory syndrome.

Table 4. Characteristics of influenza and COVID-19 vaccines

Characteristics	COVID-19 vaccines	Influenza vaccines
Types of approved vaccines	Viral vector, mRNA, inactivated, protein	Inactivated, attenuated, recombinant
Main antigen	Spike protein (S)	Hemagglutinin (HA) and neuraminidase (NA)
Initial doses	2 primary doses (mRNA); 1 dose (viral vector, inactivated vaccines)	1 annual dose
Boosters	Yes, recommended in general population and risk groups, with vaccines updated according to variants	Not considered a booster; it is an adapted annual vaccination
Efficacy against symptomatic infection	Initially high (>90% with mRNA vaccine), but decreases for new variants	40-60%, depending on concordance with circulating strains
Efficacy against severe disease	High (>80%), especially with boosters	Good (>60%), especially in elderly and risk groups
Use in children	Approved from 6 months (depending on formulation)	Approved from 6 months
Route of administration	Intramuscular	Intramuscular or intranasal (attenuated)
Safety	Safe profile; mild and transitory adverse effects in most cases	Safe profile; mild effects such as fever or local pain
Main contraindications	Severe allergy to vaccine components (e.g., polyethylene glycol)	Severe egg allergy (only some formulations)

meeting the definition of ARDS[19]. Both groups were of similar age (mean of 45 years), with a predominance of men; on average, they presented 9 days of symptoms, and the stay in intensive care was 15 days. An adjustment was made for a wasted ventilation index[20] and for the SOFA (Sepsis-related Organ Failure Assessment) score[21]. Patients with influenza had a slightly higher body mass index, glucose, lactate dehydrogenase, and SOFA score, as well as a higher proportion of individuals with obesity, fever, difficulty breathing, muscle pain, and headache, with lower neutrophils, and in a worse clinical situation, since they showed greater hypotension and hypoxemia (Table 2). Despite this, COVID-19 patients had higher crude mortality in intensive care (39% versus 22%; p = 0.007), lower respiratory compliance, and spent more days in prone position. In a multivariate Cox proportional hazards model, adjusted for age, sex, comorbidity, symptoms, laboratory, SOFA, and ventilatory quotient, COVID-19 patients had almost four times higher risk of death (odds ratio: 3.7; 95% confidence interval: 1.9-7.4) than influenza patients. Table 3 also shows that the described alterations are similar in terms of type, but there are several differences in the degree of alteration.

In an extensive study conducted in hospitalized patients in France, 89,530 subjects with COVID-19 and 45,819 with influenza were compared, and the greater aggressiveness and mortality of COVID-19 was corroborated. The median age of patients was 68 years for COVID-19 and 71 years for influenza. Comorbidity differed somewhat between the two infections: in COVID-19, obesity, diabetes, hypertension, and dyslipidemia were

found more frequently, while for influenza, there were more patients with heart failure, chronic respiratory disease, cirrhosis, and nutrient deficiency anemia[13]. COVID-19 patients experienced more complications from acute respiratory failure, pulmonary embolism, septic shock, or hemorrhagic stroke, and influenza patients presented more myocardial infarction or atrial fibrillation. Hospital mortality was three times higher in COVID-19 patients than in influenza patients (16.9% vs. 5.8%). Greater pediatric hospitalization attributable to influenza than to COVID-19 was also corroborated, but there was a greater requirement for intensive care in children under 5 years old for COVID-19 than for influenza. On the other hand, in a study conducted in children in the United States of America, no differences in severity were found between seasonal influenza and COVID-19[22].

The better prognosis of influenza compared to the initial outbreak of COVID-19 can be explained by the availability of oseltamivir and an influenza vaccine of variable effectiveness, although only around 5% of those who reached intensive care had been vaccinated. Among the COVID-19 patients, the majority received systemic corticosteroids, but neither antiviral treatment nor the protection of a prior vaccine. As an observational study, it is subject to possible biases, notwithstanding adjustment for predictive variables of death. Studies conducted in other countries have reached similar conclusions[23-26].

Current status of influenza and COVID-19

Since the WHO's declaration of the end of the international health emergency for COVID-19, in May 2023, a low incidence of the disease has been maintained, with lower mortality and fewer complications, as a result of natural immunity in people who became ill and immunity from vaccinations. In addition to reduced transmission throughout the year, seasonal outbreaks have occurred in winter, which has been attributed to greater survival of the virus in cold and humid air, and to behavioral changes that favor transmission, as previously described[27-29]. This behavior indicates the need to strengthen immunity against both viruses before winter or throughout the year in places that do not show a seasonal pattern. Table 4 describes the main characteristics of influenza and COVID-19 vaccines. The discontinuation of non-pharmacological preventive measures implemented for COVID-19 has facilitated the emergence of outbreaks of various respiratory viruses that occur regularly, and for their clinical differentiation from influenza and COVID-19, virological surveillance is necessarily required.

Conclusions

Both the study conducted at INER and those conducted in other units to compare the behavior of COVID-19 and influenza show substantially higher lethality for COVID-19, despite both presenting with respiratory failure and ARDS, and after taking into account several predictive factors of death such as age, sex, some laboratory measurements, and ventilatory parameters, which indicates more severe lung damage and fewer possibilities of recovery in COVID-19, with increased sequelae. Influenza and COVID-19 can cause severe damage to the lungs and other organs and lead to death. It is unknown whether this can be attributed to direct viral injury, individual susceptibility, or an exaggerated inflammatory reaction. A cytokine storm is often mentioned, but COVID-19 patients have not shown higher cytokine levels compared to influenza patients[12], which does not support that the storm is greater in COVID-19 and that this determines the higher mortality.

Funding

None.

Conflicts of interest

The authors declare no conflicts of interest.

Ethical considerations

Protection of people and animals. The authors declare that no experiments have been conducted on humans or animals for this research.

Confidentiality, informed consent, and ethical approval. The study does not involve personal patient data or require ethical approval. SAGER guidelines do not apply.

Declaration on the use of artificial intelligence. The authors declare that they did not use any type of generative artificial intelligence for the writing of this manuscript.

References

1. Planas D, Veyer D, Baidaliuk A, Staropoli I, Guivel-Benhassine F, Rajah MM, et al. Reduced sensitivity of SARS-CoV-2 variant Delta to antibody neutralization. Nature. 2021;596:276-80.

2. Zazhytska M, Kodra A, Hoagland DA, Frere J, Fullard JF, Shayya H, et al. Non-cell-autonomous disruption of nuclear architecture as a potential cause of COVID-19-induced anosmia. Cell. 2022;185:1052-64e1012.
3. Wang Y, Yang Y, Shan R, Zhao L, Bai Y, Feng L. Paxlovid for the treatment of COVID-19: a systematic review and meta-analysis. J Infect Dev Ctries. 2024;18:1169-78.
4. Pirracchio R, Venkatesh B, Legrand M. Low-dose corticosteroids for critically ill adults with severe pulmonary infections: a review. JAMA. 2024;332:318-28.
5. Perez-Padilla R, de la Rosa-Zamboni D, Ponce de León S, Hernández M, Quiñones-Falconi F, Bautista E, et al. Pneumonia and respiratory failure from swine-origin influenza A (H1N1) in Mexico. N Engl J Med. 2009;361:680-9.
6. Duggal A, Pinto R, Rubenfeld G, Fowler RA. Global variability in reported mortality for critical illness during the 2009-10 influenza A(H1N1) pandemic: a systematic review and meta-regression to guide reporting of outcomes during disease outbreaks. PLoS One. 2016;11:e0155044.
7. Paget J, Spreeuwenberg P, Charu V, Taylor RJ, Iuliano AD, Bresee J, et al. Global mortality associated with seasonal influenza epidemics: new burden estimates and predictors from the GLaMOR Project. J Glob Health. 2019;9:020421.
8. World Health Organization. Influenza (seasonal). 2020 June 1, 2020. Available from: https://www.who.int/news-room/fact-sheets/detail/influenza-(seasonal).
9. Chan JF, Yuan S, Kok KH, To KK, Chu H, Yang J, et al. A familial cluster of pneumonia associated with the 2019 novel coronavirus indicating person-to-person transmission: a study of a family cluster. Lancet. 2020;395:514-23.
10. Chen N, Zhou M, Dong X, Qu J, Gong F, Han Y, et al. Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. Lancet. 2020;395:507-13.
11. World Health Organization. Coronavirus disease (COVID-19) situation report. 2020. Available from: https://www.who.int/publications/m/item/weekly-epidemiological-update---1-december-2020.
12. Armstrong RA, Kane AD, Cook TM. Outcomes from intensive care in patients with COVID-19: a systematic review and meta-analysis of observational studies. Anaesthesia. 2020;75:1340-9.
13. Havasi A, Visan S, Cainap C, Cainap SS, Mihaila AA, Pop LA. Influenza A, influenza B, and SARS-CoV-2 similarities and differences — a focus on diagnosis. Front Microbiol. 2022;13:908525.
14. Vilca-Alosilla JJ, Candia-Puma MA, Coronel-Monje K, Goyzueta-Mamani LD, Galdino AS, Machado-de-Avila RA, et al. A systematic review and meta-analysis comparing the diagnostic accuracy tests of COVID-19. Diagnostics (Basel). 2023;13:1549.
15. Widyasari K, Kim S, Kim S, Lim CS. Performance evaluation of STANDARD Q COVID/FLU Ag Combo for detection of SARS-CoV-2 and influenza A/B. Diagnostics (Basel). 2022;13:32.
16. Onigbinde SO, Ojo AS, Fleary L, Hage R. Chest computed tomography f indings in COVID-19 and influenza: a narrative review. Biomed Res Int. 2020;2020:6928368.
17. Nicolai L, Leunig A, Brambs S, Kaiser R, Joppich M, Hoffknecht M, et al. Vascular neutrophilic inflammation and immunothrombosis distinguish severe COVID-19 from influenza pneumonia. J Thromb Haemost. 2021;19:574-81.
18. Hernández-Cárdenas C, Lugo-Goytia G, Hernández-García D, Pérez-Padilla R. Comparison of the clinical characteristics and mortality in acute respiratory distress syndrome due to COVID-19 versus due to Influenza A-H1N1pdm09. Med Intensiva (Engl Ed). 2022;46:345-7.
19. Ranieri VM, Rubenfeld GD, Thompson BT, Ferguson ND, Caldwell E, Fan E, et al. Acute respiratory distress syndrome: the Berlin Definition. JAMA. 2012;307:2526-33.
20. Sinha P, Calfee CS, Beitler JR, Soni N, Ho K, Matthay MA, et al. Physiologic analysis and clinical performance of the ventilatory ratio in acute respiratory distress syndrome. Am J Respir Crit Care Med. 2019;199: 333-41.
21. Vincent JL, Moreno R, Takala J, Willatts S, De Mendonça A, Bruining H, et al. The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/failure. On behalf of the Working Group on Sepsis-Related Problems of the European Society of Intensive Care Medicine. Intensive Care Med. 1996;22:707-10.
22. Song X, Delaney M, Shah RK, Campos JM, Wessel DL, DeBiasi RL. Comparison of clinical features of COVID-19 vs seasonal influenza A and B in US children. JAMA Netw Open. 2020;3:e2020495.
23. Merkler AE, Parikh NS, Mir S, Gupta A, Kamel H, Lin E, et al. Risk of ischemic stroke in patients with coronavirus disease 2019 (COVID-19) vs patients with influenza. JAMA Neurol. 2020;77:1-7.
24. Ludwig M, Jacob J, Basedow F, Andersohn F, Walker J. Clinical outcomes and characteristics of patients hospitalized for influenza or COVID-19 in Germany. Int J Infect Dis. 2021;103:316-22.
25. Donnino MW, Moskowitz A, Thompson GS, Heydrick SJ, Pawar RD, Berg KM, et al. Comparison between patients hospitalized with influenza and COVID-19 at a tertiary care center. J Gen Intern Med. 2021;36:1689-95.
26. Brehm TT, van der Meirschen M, Hennigs A, Roedl K, Jarczak D, Wichmann D, et al. Comparison of clinical characteristics and disease outcome of COVID-19 and seasonal influenza. Sci Rep. 2021;11:5803.
27. Wiemken TL, Khan F, Puzniak L, Yang W, Simmering J, Polgreen P, et al. Seasonal trends in COVID-19 cases, hospitalizations, and mortality in the United States and Europe. Sci Rep. 2023;13:3886.
28. Kyaw MH, Spinardi JR, Jagun O, Franco Villalobos C, Kapetanakis V, Sharf-Williams R, et al. Descriptive analysis to assess seasonal patterns of COVID-19 and influenza in low-income and middle-income countries in Asia, the Middle East and Latin America. BMJ Open. 2024;14:e081019.
29. Inaida S, Paul RE, Matsuno S. Viral transmissibility of SARS-CoV-2 accelerates in the winter, similarly to influenza epidemics. Am J Infect Control. 2022;50:1070-6.