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Existence and relevance of fulminant severe community-acquired pneumonia

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Abstract

Background Severe community-acquired pneumonia (sCAP) is a major contributor to global hospital mortality, with some patients rapidly progressing to death within a few days due to acute respiratory distress syndrome (ARDS), septic shock, or multiorgan failure (MOFS). Despite the significant health burden, these cases remain poorly defined, often overlooked in clinical practice, and insufficiently addressed in existing guidelines.

Methods In this multicenter retrospective study, we analyzed 1,517 hospitalized patients with sCAP for assessing the incidence of fulminant pneumonia -defined as sCAP leading to death within 7 days from hospital admission- and to identify factors associated with either an increased or reduced risk of unfavorable outcome.

Results Our findings revealed a 5.9% incidence of fulminant pneumonia, with early death primarily due to ARDS (93%), sepsis (70%), and MOFS (73%). Older age, obesity, diabetes, cardiovascular disease, and elevated serum creatinine were associated with increased risk, while early corticosteroid administration was associated with a significant mortality reduction within seven days, both in the univariate and multivariate analyses (OR 0.22, CI 95% 0.12–0.38, $p < 0.001$). Baseline respiratory impairment and inflammatory markers were not associated with early mortality.

Conclusion These findings highlight the importance of recognizing sCAP as a time-dependent condition, requiring early identification and treatment of cases at risk for developing fulminant pneumonia at hospital admission. Larger studies and randomized controlled trials are necessary to validate these findings and optimize treatment strategies.

Clinical trial registration ClinicalTrials.gov NCT06516601.

Keywords Community-acquired pneumonia, Early death, ARDS, Sepsis, Multiorgan failure

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Introduction

Severe community-acquired pneumonia (sCAP) is a major global cause of hospital mortality [1]. Some sCAP cases are characterized by rapid clinical deterioration due to the development of acute respiratory distress syndrome (ARDS) and/or septic shock, leading to death within hours to a few days. This form of sCAP has sometimes been referred to as “fulminant pneumonia” [2]. The COVID-19 pandemic has highlighted the significant burden of fulminant pneumonia both on individual health and healthcare systems, prompting the need for further research [3, 4]. While a formal definition for fulminant pneumonia has not been established to date, we deem that, in hospitalized patients, a rapidly progressive CAP leading to death within 7 days from hospital admission should be considered as a fulminant form of sCAP [5].

Nonetheless, the incidence and optimal treatment strategies remain poorly understood. This knowledge gap is partly due to two factors: the limited attention typically given to pneumonia as a time-dependent clinical entity and the tendency to study and manage overlapping clinical conditions –such as sCAP, ARDS, and sepsis– as distinct disease processes, despite pneumonia being the leading cause of both ARDS and sepsis [6, 7]. Recent evidence suggests that early and prolonged corticosteroid therapy reduces mortality in patients with severe CAP/ARDS/sepsis caused by either bacterial or viral infections, including SARS-CoV-2 [8–13]. However, it remains unclear whether the use of corticosteroids has an impact on reducing also early mortality in sCAP.

In this report, we have retrospectively analyzed a pooled multicentre large dataset of individual patients with sCAP, with the two aims: (i) to determine the incidence of fulminant pneumonia among hospitalized patients with sCAP, and (ii) to identify plausible factors associated with fulminant pneumonia.

Methods

Study design and inclusion/exclusion criteria

This is a retrospective, observational, multicenter, study conducted across five Italian and Spanish centers (University Hospital of Trieste, Italy; Instituto de Salud Carlos III, Madrid, Spain; Hospital San Giuseppe Multimedica, Milan, Italy; Ospedale S.Maria degli Angeli, Pordenone, Italy; Ospedale San Paolo, Milan, Italy) between January 2019 and July 2024. Consecutive patients affected by sCAP were included according to the criteria described below. Patients enrolled in concurrent randomized controlled trials (RCTs) were also included [12, 14]. Data were extracted from electronic medical records. The study was registered on ClinicalTrials.gov (NCT06516601), and received approval from the referral ethics committee at the coordinating center, the University Hospital of Trieste (CEUR-2024-OS-6). The protocol

and trial were conducted following the Declaration of Helsinki, International Council for Harmonisation E6 Guideline for Good Clinical Practice and European regulations. Signed informed written consent was obtained from the patient or her/his next of kin if the patient was unable, and all recorded data were anonymized.

Hospitalized patients were included if they met the following criteria: bilateral infiltrates caused by community-acquired pneumonia, onset of symptoms within 10 days, a $\text{PaO}_2/\text{FiO}_2$ ratio ≤ 250 , and at least two of the following conditions: (1) C-reactive protein (CRP) > 99 mg/L, (2) respiratory rate > 25 bpm or signs of respiratory distress, (3) requirement for respiratory support such as continuous positive airway pressure (CPAP), mechanical ventilation, or high-flow nasal cannula (HFNC), (4) serum creatinine ≥ 1.5 mg/dL, (5) blood urea nitrogen > 60 mg/dL, or (6) ALT > 70 U/L. Exclusion criteria encompassed: hospital-acquired pneumonia, healthcare-associated community-acquired pneumonia, long-term oxygen therapy or home mechanical ventilation, heart failure, decompensated liver disease, immunosuppression (e.g. active cancer, post-organ transplantation, HIV-positive status, or ongoing immunosuppressive therapy), chronic steroid therapy or immunomodulatory therapy, dialysis dependency, neurodegenerative conditions, dementia or decompensated psychiatric disorder, severe stroke sequelae (e.g. quadriplegia, hemiplegia, quadriparesis, hemiparesis), recent surgery within the past 30 days, and trauma patients.

Antibiotic treatment was administered within 6 h of admission according to usual practice. The use of corticosteroids varied depending on ongoing randomized controlled trial (RCT) protocols, institutional policies, and the clinical judgment of the attending physician [12, 14].

Variables and data collection

Severe CAP was defined as “fulminant” if the patient died within 7 days from hospital admission due to complications related to CAP. Collected patient characteristics and clinical data included obesity and other comorbidities, smoking history, age, oxygenation status, vital signs, CRP levels, complete blood count, ALT levels, glucocorticoid therapy and adverse events. The study baseline was defined as the time of hospital admission. Demographic details and laboratory, clinical, and outcome variables were manually extracted from electronic medical records or charts and anonymously coded into a standardized data collection form.

Endpoints

The primary endpoint of this study was to determine the incidence of fulminant pneumonia among hospitalized patients with sCAP. The secondary endpoint was

to identify factors associated with increased or reduced incidence of fulminant pneumonia.

Statistical methods

Since the primary objective of this study was descriptive, an a priori calculation of sample size was not applicable. Instead, we included all patients who met the inclusion and exclusion criteria at the participating centers during the study period. Data were described using absolute and relative frequencies (percentage) or position indices (mean or median) and relative dispersion indices

Table 1 Baseline characteristics of patients with community-acquired pneumonia

Baseline characteristics	n = 1517
Age (median, IQR)	64 (22)
Male sex (n, %)	974 (64.21%)
Active smoking (n, %)	176 (11.61%)
Corticosteroids within 48 h (n, %)	1025 (67.57%)
Hydrocortisone	74 (4.88%)
Prednisolone	8 (0.52%)
Methylprednisolone	570 (37.57%)
Dexamethasone	372 (24.52%)
PaO ₂ /FiO ₂ , mmHg (median, IQR)	162 (108.25)
PaCO ₂ , mmHg (median, IQR)	37.2 (14)
Respiratory rate, bpm (median, IQR)	25 (11)
APACHE II score first 24 h (median, IQR)	10 (15)
Bacterial infection (n, %)	202 (13.31%)
Viral infection (n, %)	805 (53.06%)
SARS-CoV-2 (n, %)	750 (49.44%)
Any comorbidities	501 (33.03%)
Obesity = BMI > 29 (n, %)	141 (9.29%)
Cardiovascular disease (n, %)	174 (11.47%)
Immunosuppression (n, %)	206 (13.58%)
Chronic pulmonary disease (n, %)	188 (12.39%)
Gastrointestinal disease (n, %)	42 (2.76%)
Diabetes (n, %)	145 (9.56%)
Chronic kidney disease (n, %)	37 (2.44%)
Drug abuse (n, %)	80 (5.27%)
Temperature, °C (median, IQR)	37.3 (1.6)
Systolic blood pressure, mmHg (median, IQR)	125 (30)
Diastolic blood pressure, mmHg (median, IQR)	71 (15)
Heart rate, bpm (median, IQR)	99 (30.75)
C-reactive protein, mg/L (median, IQR)	145.3 (160.99)
Sodium, mEq/L (median, IQR)	137 (6)
Potassium, mEq/L (median, IQR)	3.9 (0.8)
White blood cells/uL (median, IQR)	10,700 (8065)
Serum creatinine, mg/dL (median, IQR)	1.1 (0.56)
Total bilirubin, mg/dL (median, IQR)	0.8 (0.6)
Platelets/uL (median, IQR)	232,100 (163,000)
Hemoglobin, g/dL (median, IQR)	12.2 (2.7)
ALT, U/L (median, IQR)	34 (36)
Albumin, g/dL (median, IQR)	3 (0.85)
Blood urea nitrogen, mg/dl (median, IQR)	34 (31.65)
Blood glucose, mg/dl (median, IQR)	130 (58)

(SD or interquartile range), as appropriate according to the type and distribution of analyzed variables. Differences between study groups in the proportion of patients reaching the primary endpoint were evaluated using a 2-sided chi-square test for categorical variables. The difference in numerical variables between groups was calculated using the Student's *t* test or Wilcoxon rank-sum test, depending on the distribution of the variables. All variables with baseline differences were tested in univariable logistic regression models, and the most significant one were further tested in a multivariable logistic regression model. There was no missing data regarding the primary end point. A *p* value of <0.05 was considered statistically significant.

Results

The study population included 1,517 patients with sCAP, 974 (64.2%) of whom were males. The median ± IQR age was 64 ± 22 years. Table 1 describes the baseline characteristics of the study population. Fulminant pneumonia occurred in 89 (5.9%) patients, with 93.4% of these resulting in death due to ARDS, 70.2% deaths due to severe sepsis, and 72.7% deaths due to multiorgan failure (MOFS).

Early glucocorticoid treatment (i.e. initiated within 48 h) was administered in 33 (37%) of patients who died within 7 days vs. 992 (69%) of those who survived over 7 days, appearing to be the strongest protective factor associated with fulminant pneumonia (OR 0.26, CI 95% 0.16–0.40, *p* < 0.001). This effect reached significance in patients treated with methylprednisolone (24.7% vs. 41.5%, OR 0.46, CI 95% 0.28–0.76, *p* = 0.002) and dexamethasone (14.6% vs. 25.1%, OR 0.51, CI 95% 0.28–0.92, *p* = 0.03), while the results for treatment with hydrocortisone and prednisolone were likely limited due to low sample size.

The presence of major comorbidities, such as diabetes and cardiovascular diseases, was associated with a higher likelihood of fulminant pneumonia. These conditions were more prevalent among patients who died within 7 days compared to those who survived beyond 7 days (21.3% vs. 8.8%, OR 2.87, CI 95% 1.63–4.84, *p* < 0.001, and 24.7% vs. 10.6%, OR 2.82, CI 95% 1.65–4.68, *p* < 0.001, respectively). Factors associated with fulminant pneumonia also included older age (72 ± 15 vs. 64 ± 21 years, OR 1.03, CI 95% 1.01–1.05, *p* < 0.001), as well as obesity defined as BMI > 29, increased respiratory rate and heart rate, reduced systolic and diastolic blood pressure, a higher APACHE II score, a higher serum creatinine and blood urea nitrogen, lower hemoglobin, platelets and albumin levels. Details on the associations between baseline characteristics and fulminant pneumonia are provided in Table 2; Fig. 1. Of note, baseline respiratory impairment (PaO₂/FiO₂) and systemic inflammation

Table 2 Association between baseline characteristics and mortality at 7 days

Characteristics	Survival >7 d (n = 1428)	Death ≤ 7 d (n = 89)	OR ¹	95% CI ¹	p-value
Age (median, IQR)	64 (21)	72 (15)	1.03	1.01–1.05	< 0.001
Male sex (n, %)	917 (64.2%)	57 (64%)	0.99	0.64–1.55	1
Active smoking (n, %)	164 (11.5%)	12 (13.5%)	1.20	0.65–2.23	0.61
Corticosteroids within 48 h (n, %)	992 (69.5%)	33 (37.1%)	0.26	0.17–0.40	< 0.001
Hydrocortisone	72 (5%)	2 (2.2%)	0.43	0.12–1.56	0.31
Prednisolone	8 (0.6%)	0 (0%)	n/a	n/a	n/a
Methylprednisolone	552 (38.6%)	18 (20.2%)	0.40	0.24–0.68	< 0.001
Dexamethasone	359 (25.1%)	13 (14.6%)	0.51	0.28–0.92	0.03
PaO ₂ /FI _O ₂ , mmHg (median, IQR)	162 (109)	153 (94)	1.00	0.99–1.00	0.09
PaCO ₂ , mmHg (median, IQR)	37 (14)	40 (12)	1.00	0.98–1.02	0.7
Respiratory rate, bpm (median, IQR)	25 (11)	29 (12)	1.06	1.03–1.09	< 0.001
APACHE II score first 24 h (median, IQR)	9 (14)	20 (12)	1.09	1.05–1.13	< 0.001
Bacterial infection (n, %)	189 (13.2%)	13 (14.6%)	1.13	0.59–2.00	0.75
Viral infection (n, %)	765 (52.9%)	40 (44.9%)	0.71	0.46–1.09	0.12
SARS-CoV-2 (n, %)	715 (50.1%)	35 (39.3%)	0.65	0.41–1.00	0.05
Any comorbidities	462 (32.3%)	39 (43.8%)	1.63	1.06–2.51	0.03
Obesity = BMI > 29 (n, %)	123 (8.6%)	18 (20.2%)	2.69	1.56–4.63	< 0.001
Cardiovascular disease (n, %)	152 (10.6%)	22 (24.7%)	2.75	1.66–4.57	< 0.001
Immunosuppression (n, %)	194 (13.9%)	12 (13.5%)	0.99	0.53–1.83	1
Chronic pulmonary disease (n, %)	173 (12.1%)	15 (16.8%)	1.47	0.83–2.59	0.18
Gastrointestinal disease (n, %)	37 (2.6%)	5 (5.6%)	2.23	0.89–5.62	0.09
Diabetes (n, %)	126 (8.8%)	19 (21.3%)	2.80	1.64–4.78	< 0.001
Chronic kidney disease (n, %)	33 (2.3%)	4 (4.5%)	1.98	0.72–5.45	0.27
Drug abuse (n, %)	72 (5%)	8 (9%)	1.86	0.88–3.91	0.13
Temperature, °C (median, IQR)	37.3 (1.5)	37.75 (1.9)	1.00	0.80–1.26	1
Systolic blood pressure, mmHg (median, IQR)	137 (6)	135.5 (9)	0.98	0.97–0.99	< 0.001
Diastolic blood pressure, mmHg (median, IQR)	73 (15)	70 (20)	0.98	0.96–1.00	0.03
Heart rate, bpm (median, IQR)	98 (31)	102 (30)	1.01	1.00–1.02	0.03
C-reactive protein, mg/L (median, IQR)	144 (160)	165 (182)	1.00	1.00–1.00	0.2
Sodium, mEq/L (median, IQR)	137 (6)	135.5 (9)	0.97	0.92–1.03	0.3
Potassium, mEq/L (median, IQR)	3.9 (0.8)	4 (1)	1.20	0.79–1.80	0.4
White blood cells/uL (median, IQR)	10,755 (7,990)	9,745 (9,100)	1.00	1.00–1.00	0.5
Serum creatinine, mg/dL (median, IQR)	1.1 (0.5)	1.4 (1.1)	1.53	1.24–1.92	< 0.001
Total bilirubin, mg/dL (median, IQR)	0.8 (0.6)	0.8 (0.8)	1.16	0.76–1.64	0.4
Platelets/uL (median, IQR)	234,000 (161,000)	182,650 (173,000)	1.00	1.00–1.00	0.02
Hemoglobin, g/dL (median, IQR)	12.2 (2.6)	11.55 (2.8)	0.83	0.73–0.95	0.005
ALT, U/L (median, IQR)	33 (35)	39 (44)	1.00	1.00–1.01	0.4
Albumin, g/dL (median, IQR)	3 (0.8)	2.9 (0.6)	0.57	0.31–1.03	0.06
Blood urea nitrogen, mg/dl (median, IQR)	34 (28)	59 (71)	1.01	1.01–1.02	< 0.001
Blood glucose, mg/dl (median, IQR)	130 (58)	140 (52)	1.00	1.00–1.01	0.3

¹OR Odds Ratio, CI Confidence Interval

(C-reactive protein levels) were not associated with increased risk of death within 7 days, neither the viral or bacterial origin of pneumonia.

The protective effect of glucocorticoids on mortality within 7 days was confirmed to be markedly significant (OR 0.22, CI 95% 0.12–0.38, $p < 0.001$) in a multivariate model that included age, SARS-CoV-2 infection, diabetes and cardiovascular diseases. In this model, diabetes remained associated with fulminant pneumonia (OR 1.95, CI 95% 0.99–3.73, $p = 0.046$), whereas cardiovascular diseases (OR 1.28, CI 95% 0.67–2.36, $p = 0.4$)

and SARS-CoV-2 infection (OR 1.70, CI 95% 0.95–3.06, $p = 0.074$) were associated with higher odds fulminant pneumonia but did not reach significance.

Exploratory univariable analyses on death within 48 h, which can be considered as a stricter criterion for fulminant pneumonia, revealed that 19 (1.3%) patients died within this time frame. These patients died due to ARDS, with 80.0% also experiencing sepsis and 66.7% multiorgan failure. Early glucocorticoid treatment was associated with mortality reduction within 48 h, as 3 (15.8%) deaths occurred among patients receiving early glucocorticoid

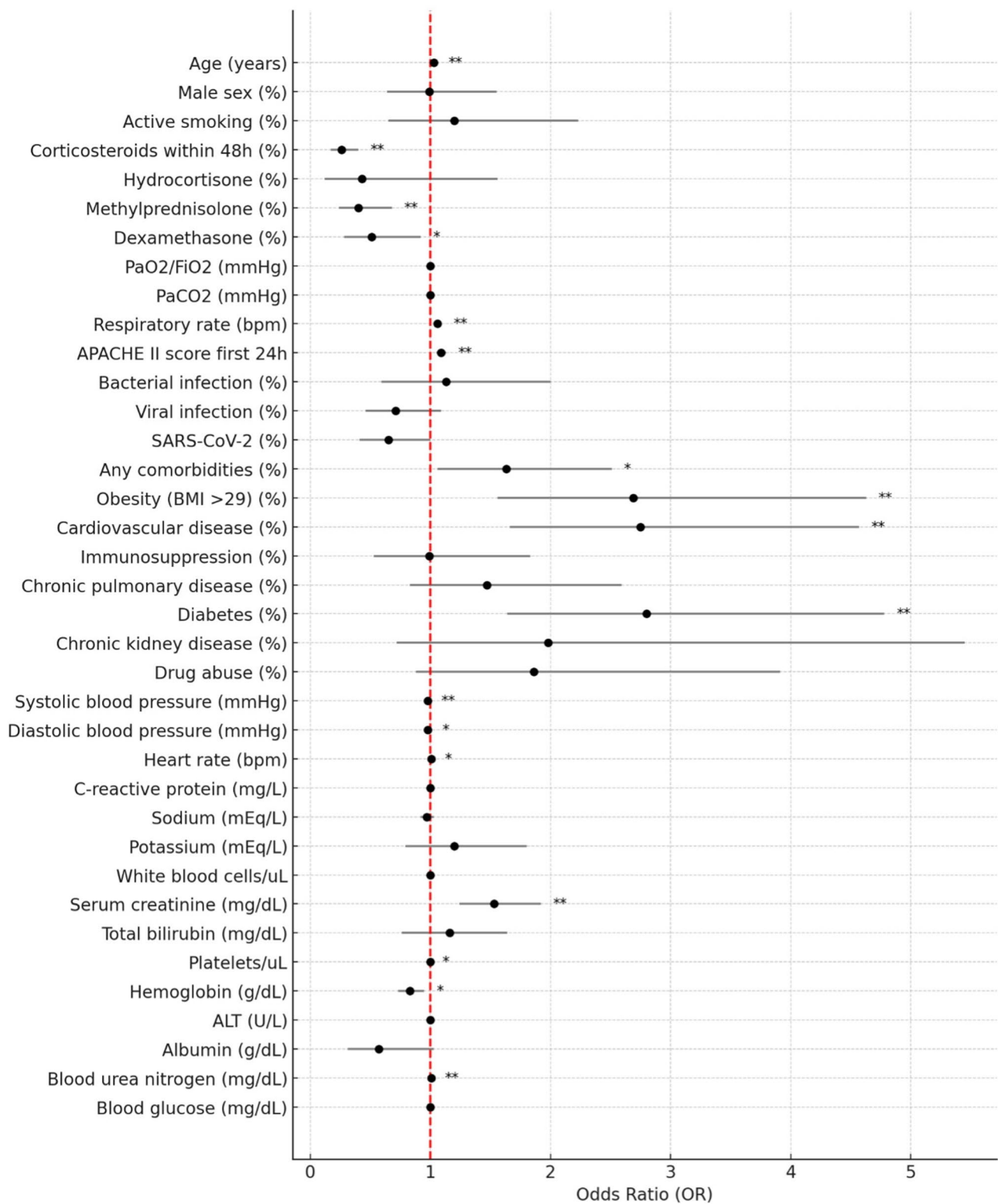


Fig. 1 Forest plot illustrating the association between baseline characteristics and fulminant pneumonia. $*=p \leq 0.05$; $**=p \leq 0.001$

therapy vs. 16 (84.2%) deaths among those who did not, $p < 0.001$.

Discussion

We have found that among 1,517 patients with sCAP, 89 (5.9%) developed fulminant pneumonia, defined as death within seven days from hospital admission. This finding aligns with previous literature [15]. Key baseline risk factors for fulminant pneumonia included older age, obesity, diabetes, cardiovascular comorbidities and elevated serum creatinine levels. Conversely, an early corticosteroid treatment appeared to have a protective effect. The leading cause of death was ARDS, followed closely by sepsis and MOFS, with a significant overlap in their occurrences.

Beyond the identification of factors associated with fulminant pneumonia, a key objective of this study was to highlight its existence and incidence among severe CAP patients. We strongly believe that fulminant pneumonia remains an under-recognized condition in medical practice and it is often overlooked in major medical textbooks, unlike well-documented conditions such as fulminant hepatitis or other rapidly progressive inflammatory diseases [16]. Even current CAP and sCAP guidelines fail to acknowledge its existence and clinical significance [17, 18].

According to the published reports, about 40% of CAP patients require hospitalization, with 5–10% admitted to the intensive care unit (ICU), primarily due to shock or respiratory failure requiring invasive or noninvasive mechanical ventilation. The recent ERS/ESICM/ESCMID/ALAT guidelines for the management of sCAP emphasize ICU admission as the primary criterion when defining severe cases [18]. However, this approach can be inconsistent in the absence of organ support needs and lacks precise criteria for emergency room triage. This is particularly relevant, as research indicates that patients initially triaged to general wards but later transferred to the ICU face a higher risk of progressive pneumonia and a twofold increase in early mortality [15, 19]. Therefore, early identification of patients at risk for rapid deterioration is crucial and should be actively pursued. Fulminant pneumonia represents one of the most severe manifestations of acute respiratory infections, characterized by rapid clinical deterioration and high mortality rates despite intensive care. Although its incidence is relatively low, its clinical impact in outcome is profound.

Our retrospective analysis underscores the importance of identifying specific patient characteristics at the triage level to detect those patients at higher risk for rapidly progressive pneumonia.

Even if this study was not designed to assess the efficacy of pharmacological interventions, we observed that corticosteroid therapy administered within 48 h from

admission was associated with a reduced risk of early death. This finding aligns with previous evidence supporting the effectiveness of early corticosteroid use in reducing mortality rates in sCAP cases [10, 20]. Recently, the ESCAPE and REMAP-CAP trials did not demonstrate a mortality benefit with corticosteroids [21, 22]. However, some methodological aspects may have influenced this result. In the ESCAPE trial, approximately 34% of participants met criteria for healthcare-associated pneumonia (HCAP), the analyses may have been underpowered, and delayed initiation of anti-inflammatory therapy may have attenuated the differences between the treatment groups. In the REMAP-CAP, the exclusion of patients with “imminent death” may have led to the underrepresentation of fulminant pneumonia cases.

Approximately half of the study population was SARS-CoV-2 positive, but this did not affect the incidence of fulminant pneumonia or clinical outcomes. Although there was initial hesitation to equate COVID-19 pneumonia with other severe cases of CAP/ARDS, a comparison of clinical and physiological characteristics and outcomes between COVID-19 and non-COVID-19 cases revealed no conclusive evidence to support deviation from evidence-based management of severe CAP/ARDS [23, 24]. However, the intent of this study was not to equate COVID-19 with classical CAP, but to highlight shared clinical trajectories leading to fulminant presentations.

Furthermore, our findings indicated that sCAP should be classified as a time-dependent condition, as timely diagnosis and treatment are essential for improving outcomes and reducing mortality. Time-dependent conditions require immediate recognition and prompt therapeutic intervention to prevent irreversible damage or death, even at the prehospital care stage [25]. In Italy, only cardiovascular and neurovascular clinical conditions are officially recognized as time-dependent diseases, although paradoxically sepsis is occasionally included. The German CAPNETZ medical competence network for CAP identified a subgroup comprising 5% of cases, defined “emergency CAP”; characterized by signs of acute organ dysfunction and associated with a worse prognosis [15]. However, despite being the leading cause of community-acquired sepsis, sCAP is frequently addressed as a separate issue in the scientific literature. This distinction has resulted in inconsistencies in international guidelines concerning corticosteroid use in sCAP, except in cases of refractory septic shock or confirmed bacterial infection [26]. Although recent European guidelines define sCAP as CAP requiring ICU admission, some patients who are not admitted to the ICU may still develop severe sepsis or ARDS and benefit from corticosteroid treatment [17].

We acknowledge that this study has major limitations. First, it is retrospective in nature. Second, due to the descriptive nature of the primary endpoint, the sample

size was not predetermined. Third, data regarding the dose, duration and administration route of glucocorticoid therapy were missing for some patients and a comparison of different corticosteroid types was not a prespecified objective of this retrospective study. These factors hinder drawing conclusions about the optimal treatment strategy to prevent fulminant pneumonia. Lastly, the analysis of risk factors associated with fulminant pneumonia was restricted to baseline characteristics for avoiding the inclusion of “immortal time bias,” given the lack of information on the timing of subsequent events. Nevertheless, we believe that the relatively large sample size of this cohort of sCAP patients provides valuable clinical insights into a rare but prognostically significant condition that deserves recognition.

Conclusion

In conclusion, our findings suggest that sCAP can be a rapidly fatal condition and requires heightened attention from the earliest stages of care. We emphasize the critical importance of early recognition and management of fulminant CAP, even before hospital admission. Large prospective and rigorously designed studies are essential to better understand the optimal management of fulminant pneumonia.

Abbreviations

sCAP	Severe community-acquired pneumonia
ARDS	Acute respiratory distress syndrome
RCTs	Randomized controlled trials
CRP	C-reactive protein
CPAP	Continuous positive airway pressure
HFNC	High-flow nasal cannula
PaO ₂ /FiO ₂	Ratio of partial pressure arterial oxygen and fraction of inspired oxygen
MOFS	Multiorgan failure

Authors' contributions

FS conceived and designed the study, analyzed and interpreted the data, drafted, revised and approved the work; JV designed the study, collected and interpreted the data, revised and approved the work; MM collected and interpreted the data, revised and approved the work; SH collected and interpreted the data, revised and approved the work; AS conceived and designed the study, collected the data, revised and approved the work; AA collected and interpreted the data, revised and approved the work; FA collected and interpreted the data, revised and approved the work; CF collected and interpreted the data, revised and approved the work; TM collected and interpreted the data, revised and approved the work; JAS collected and interpreted the data, revised and approved the work; LPM designed the study, analyzed and interpreted the data, drafted, revised and approved the work; CF analyzed and interpreted the data, drafted, revised and approved the work; DM conceived and designed the study, interpreted the data, revised and approved the work; SG collected and interpreted the data, revised and approved the work; SC conceived and designed the study, interpreted the data, drafted, revised and approved the work; UZ collected and interpreted the data, revised and approved the work; GB collected and interpreted the data, revised and approved the work; AR collected and interpreted the data, revised and approved the work; BR collected and interpreted the data, revised and approved the work; MC collected and interpreted the data, revised and approved the work; PC conceived and designed the study, interpreted the data, drafted, revised and approved the work. All Authors agree to be accountable for all aspects of the work in

ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Data availability

The data that support the findings of this study are available upon reasonable request to the corresponding author.

Declarations

Ethics approval and consent to participate

The study was registered on ClinicalTrials.gov (NCT06516601), and received approval from the referral ethics committee at the coordinating center, the University Hospital of Trieste (CEUR-2024-OS-6). The protocol and trial were conducted following the Declaration of Helsinki, International Council for Harmonisation E6 Guideline for Good Clinical Practice and European regulations. Signed informed written consent was obtained from the patient or her/his next of kin if the patient was unable, and all recorded data were anonymized.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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