

Clinical And Radiological Profile of Acute Respiratory Distress Syndrome and Its Association With In-Hospital Mortality: An Observational Study From A Tertiary Care Intensive Care Unit in North India

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ABSTRACT

Objective: To describe the clinical, radiological, microbiological and physiological profile of acute respiratory distress syndrome in a tertiary intensive care unit and identify features associated with in-hospital mortality.

Methods: Observational study of 50 consecutive patients meeting American–European Consensus Conference criteria between February and July 2014. Clinical findings, arterial blood gases, laboratory parameters, chest radiography, computed tomography, cultures and Sequential Organ Failure Assessment scores on days 1 and 3 were recorded. Those who died were compared with survivors using the Student t test and chi-square test, with Fisher's exact test where expected counts were small.

Results: Thirty-three patients (66.0%) died. Mean age was 44.9 ± 17.3 years; 64.0% were aged 50 or younger. Mortality was higher in women (82.6%) than in men (51.9%; $p = 0.022$). Sepsis and pulmonary infection caused 90% of cases; direct and indirect injury contributed equally. Pallor ($p = 0.008$), inotrope requirement ($p = 0.007$), higher heart rate ($p = 0.044$) and positive end-expiratory pressure ($p = 0.002$), lower oxygen tension ($p = 0.002$) and ratio of oxygen tension to inspired oxygen fraction ($p < 0.001$), and lower haematocrit ($p = 0.028$) were associated with death. Diffuse consolidation on computed tomography predicted death and tracked direct injury ($p < 0.001$). Baseline organ dysfunction scores did not differ, but a rising score over three days did ($p = 0.012$). Age, comorbidity, smoking and culture results did not.

Conclusion: Mortality was high in a young population. The trajectory of organ dysfunction discriminated better than any single admission measurement.

Keywords: Respiratory Distress Syndrome; Intensive Care Units; Hospital Mortality; Tomography, X-Ray Computed; Sepsis; Organ Dysfunction Scores.

INTRODUCTION

Acute respiratory distress syndrome (ARDS) is an acute hypoxaemic respiratory failure that follows diffuse injury to the alveolar–capillary membrane, producing protein-rich non-cardiogenic pulmonary oedema, reduced lung compliance and hypoxaemia refractory to supplemental oxygen.¹ Since the American–European Consensus Conference (AECC) standardised its definition in 1994,² and the Berlin definition refined it in 2012,³ ARDS has been understood as the shared endpoint of many distinct insults rather than as a single disease.

Population-based data from high-income settings place the age-adjusted incidence at 86.2 per 100,000 person-years for a ratio of arterial oxygen tension to inspired oxygen fraction below 300, with in-hospital mortality of 38.5%.⁴ Reported mortality nevertheless varies widely between series, from roughly 24% to over 70%, reflecting differences in case mix, definitions, ventilatory strategy and critical care resources.^{5,6} Whether mortality has genuinely declined over time remains contested; a systematic review of 89 cohorts found a pooled mortality of 44% and no consistent downward trend once study design was accounted for.⁷

Indian data are sparser and generally less favourable. Series from respiratory, medical and surgical intensive care units have reported mortality between 33% and 77%,⁸⁻¹¹ and the burden falls on a comparatively young population: the median age in several Indian cohorts lies below 45 years, in contrast to the predominantly elderly cohorts described from Europe, Australia and North America.^{5,8} ARDS is not confined to older adults. It is well described in adolescents and children, in whom mortality approaching 50% has been reported,^{12,13} so that the syndrome consumes a disproportionate number of potential life-years in settings where its victims are young. Because ARDS is defined clinically and radiologically rather than histologically, the bedside and imaging features available on the day of diagnosis are also the features on which prognostication must rest. We therefore studied the clinical, radiological, microbiological and physiological profile of consecutive patients with ARDS admitted to a tertiary care intensive care unit, and examined which of these features were associated with in-hospital mortality.

METHODS

Design, setting and participants

This observational, descriptive study was carried out in the Department of Medicine in collaboration with the Department of Radiology at Vivekananda Polyclinic and Institute of Medical Sciences, Lucknow, India, between February and July 2014. The study was approved by the Institutional Ethical Committee of Vivekananda Polyclinic and Institute of Medical Sciences, Lucknow. Written informed consent was obtained from all participants or their next of kin.

Fifty consecutive patients admitted to the intensive care unit who met the AECC criteria for ARDS were enrolled by purposive sampling.² The criteria applied were acute onset; bilateral infiltrates on the frontal chest radiograph; a ratio of arterial oxygen tension to inspired oxygen fraction of 200 or less; and absence of left atrial hypertension on clinical or echocardiographic assessment. Patients aged under 18 or over 75 years, those with clinical or echocardiographic evidence of heart failure, and those for whom consent was not obtained were excluded. The sample size of 50 was fixed pragmatically; no formal power calculation was performed, as the study was exploratory and tested no pre-specified hypothesis.

Procedures

Demographic details, medical history, history of smoking and alcohol use, and details of the presenting illness were recorded, followed by general and systemic examination. Investigations included complete blood count with haematocrit, serum bilirubin, transaminases, alkaline phosphatase, creatinine and albumin, and arterial blood gas analysis. C-reactive protein and procalcitonin were measured within 48 hours of diagnosis. Bronchoalveolar lavage, endotracheal aspirate, urine, throat swab or serological specimens were obtained as clinically indicated and cultured. Chest radiography was performed in all patients on the day of diagnosis and at intervals thereafter; computed tomography of the thorax was obtained wherever the patient's condition permitted transfer. Organ dysfunction was quantified using the Sequential Organ Failure Assessment (SOFA) score on days 1 and 3.¹⁴ Patients were reviewed every 24 hours for vital parameters, new organ dysfunction, arterial blood gases and inotrope requirement; where a parameter was measured more than once in 24 hours, the most abnormal value was taken. The precipitating disorder identified within the first 24 hours was recorded as the aetiology and classified as direct or indirect lung injury.

Outcome definition

The primary outcome was in-hospital mortality. Patients were classified as having died (Group A) or survived (Group B). Seven patients were transferred to other institutions for continuing care before a final outcome could be ascertained; these were analysed within the survivor group, which makes the reported mortality a conservative estimate. Restricting the analysis to the 43 patients with a definitively ascertained in-hospital outcome yields a mortality of 76.7%.

Statistical analysis

Analyses were performed in SPSS version 15.0. Continuous variables are presented as mean $\pm$ standard deviation and compared using the unpaired Student t test; within-group change in SOFA score was compared using the paired t test. Categorical variables are presented as number (percentage) and compared using the chi-square test, with Fisher's exact test used to confirm associations in tables containing expected cell counts below five. A two-sided p value below 0.05 was taken as significant. Permission was obtained from the Institutional Ethical Committee, and written informed consent was obtained from every participant or, where the patient was unable to consent, from a family member.

RESULTS

Outcome and demographic profile

Of the 50 patients, 33 (66.0%) died in hospital and 17 (34.0%) survived, comprising 10 (20.0%) who recovered and 7 (14.0%) who were referred elsewhere. Mean age was 44.9 ± 17.3 years (range 18–75); 32 patients (64.0%) were aged 50 years or younger, and 2 were adolescents aged 18–19 years. Age did not differ between those who died and those who survived (45.3 ± 18.5 versus 44.1 ± 15.2 years; $p = 0.817$), and the distribution across age bands was not associated with outcome ($p = 0.504$). Mortality was 59.4% in patients aged 50 years or under and 77.8% in those older, a difference that did not reach significance ($p = 0.187$).

Men formed the slight majority of the cohort (27; 54.0%), but mortality was markedly higher among women (19 of 23; 82.6%) than among men (14 of 27; 51.9%; $p = 0.022$; Fisher's exact $p = 0.036$). Demographic findings are shown in Table 1.

Aetiology

Sepsis (24; 48.0%) and pulmonary infection (21; 42.0%) accounted for 90% of cases, with aspiration (3), intestinal perforation (1) and non-thoracic trauma (1) making up the remainder. Direct and indirect lung injury contributed equally, 25 patients (50.0%) each. Mortality was higher after direct injury (18 of 25; 72.0%) than after indirect injury (15 of 25; 60.0%), but the difference was not significant ($p = 0.370$). No individual aetiological category was significantly associated with outcome (Table 2).

Clinical features

Dyspnoea and crepitations were universal, and fever was present in 48 patients (96.0%). Agitation (35; 70.0%), cough (23; 46.0%), rhonchi (18; 36.0%), pallor (15; 30.0%) and disorientation (13; 26.0%) were the other common findings. Oedema (8; 16.0%), icterus (7; 14.0%), cyanosis (4; 8.0%), clubbing (2; 4.0%) and lymphadenopathy (2; 4.0%) were uncommon. Of these, only pallor was significantly associated with death, being present in 14 of 33 patients who died (42.4%) compared with 1 of 17 survivors (5.9%; $p = 0.008$) (Table 3).

Diabetes mellitus (14; 28.0%) and hypertension (7; 14.0%) were the only comorbidities recorded, and neither was associated with outcome ($p = 0.242$ and $p = 0.594$). Smoking (10; 20.0%) and alcohol use (6; 12.0%) were likewise unrelated to mortality. Inotropic support was required by 40 patients (80.0%) and was strongly associated with death, being needed by 30 of 33 non-survivors (90.9%) against 10 of 17 survivors (58.8%; $p = 0.007$).

Physiological and laboratory parameters

Patients who died had a higher heart rate (115.7 ± 16.3 versus 103.7 ± 24.7 beats per minute; $p = 0.044$) and required higher positive end-expiratory pressure (13.4 ± 2.6 versus 10.5 ± 3.5 cm H₂O; $p = 0.002$). Oxygenation was substantially worse in non-survivors, whose arterial oxygen tension averaged 69.8 ± 17.6 mmHg against 92.7 ± 33.3 mmHg in survivors ($p = 0.002$) and whose ratio of arterial oxygen tension to inspired oxygen fraction was 78.5 ± 24.7 against 115.1 ± 41.3 ($p < 0.001$). Haematocrit was lower in those who died ($31.1 \pm 7.2\%$ versus $35.5 \pm 4.6\%$; $p = 0.028$). Blood pressure, pH, bicarbonate, carbon dioxide tension, leucocyte and platelet counts, creatinine, bilirubin, albumin, C-reactive protein and procalcitonin did not differ between groups (Table 4).

Radiology, microbiology and organ dysfunction

All 50 patients showed diffuse bilateral infiltrates on chest radiography; 7 (14.0%) had an accompanying mild pleural effusion, a finding unrelated to outcome ($p = 0.163$). Computed tomography was obtained in 45 patients (29 who died, 16 who survived). Ground-glass opacity was the dominant pattern in 24 (53.3%), diffuse consolidation in 18 (40.0%) and multifocal patchy involvement in 3 (6.7%). The pattern was associated with outcome ($p = 0.013$): diffuse consolidation was present in 15 of 29 non-survivors (51.7%) but only 3 of 16 survivors (18.8%), while all 3 patients with multifocal patchy involvement survived. The pattern also tracked the mechanism of injury closely ($p < 0.001$), with diffuse consolidation in 16 of 23 patients with direct injury and ground-glass opacity in 19 of 22 with indirect injury.

Cultures were positive in 25 patients (50.0%), most often from endotracheal aspirate (12) or bronchoalveolar lavage (8). *Acinetobacter* species (9) and extended-spectrum beta-lactamase-producing *Klebsiella* (8) were the commonest isolates; influenza A(H1N1) was identified in 2 patients, both of whom died. Neither culture positivity ($p = 0.370$) nor the organism isolated ($p = 0.441$) was associated with mortality.

Day 1 SOFA scores were identical in the two groups (7.82 ± 2.07 versus 7.82 ± 3.17 ; $p = 0.994$), as were day 3 scores (9.12 ± 2.68 versus 8.24 ± 3.77 ; $p = 0.341$). The trajectory, however, separated them: the score rose significantly between days 1 and 3 among those who died (mean change $+1.30$; $p < 0.001$) but not among survivors ($+0.41$; $p = 0.234$), and the between-group difference in change was significant ($p = 0.012$). An increase in score was seen in 24 of 33 non-survivors (72.7%) against 6 of 17 survivors (35.3%), and a fall occurred only in survivors ($p = 0.014$). Neither duration of intensive care stay (9.0 ± 4.9 versus 9.3 ± 4.0 days; $p = 0.831$) nor duration of mechanical ventilation (7.1 ± 4.4 versus 6.7 ± 3.3 days; $p = 0.752$) differed (Table 5).

DISCUSSION

In-hospital mortality of 66% in this cohort sits at the upper end of the published range and well above the 38.5% reported from population-based North American surveillance⁴ or the 34% recorded across three Australian states.⁵ It is closer to the 58% described from Argentina⁶ and to the Indian experience, where series have reported 57% from a Mumbai medical intensive care unit,⁹ 68% to 77% in patients with and without influenza A(H1N1),¹⁰ and 41.8% from a surgical intensive care unit.¹¹ Agarwal and colleagues, reporting from a respiratory intensive care unit in North India, recorded considerably lower mortality,⁸ a difference plausibly attributable to case mix: a respiratory unit admits patients earlier and with fewer extrapulmonary failures than a general medical unit. The claim that ARDS mortality has fallen steadily over time⁷ is not supported by Indian data, and our figure should in any case be read as a floor, since seven patients transferred elsewhere were counted as survivors.

The age profile deserves emphasis in the context of this journal. Mean age was 44.9 years and almost two-thirds of patients were 50 or younger, a decade or more below the cohorts underlying most Western prognostic models. Although age above 65 years predicted mortality in one North American medical intensive care unit series,¹⁵ and older age predicted one-year mortality among hospital survivors elsewhere,¹⁶ we found no association between age and in-hospital death, consistent with the relatively young distribution of our patients. Two patients were adolescents, and paediatric and adolescent series report mortality of comparable magnitude,^{12,13} which argues against treating ARDS as a disease whose prognosis is governed principally by chronological age. Where the syndrome strikes a young population, as it does across much of South Asia, the loss of life-years is correspondingly greater and the case for early recognition correspondingly stronger.

Mortality was significantly higher in women despite their forming a minority of the cohort. A similar pattern has been reported from North India,⁸ and experimental work offers a plausible mechanism in the immunostimulatory effect of female sex hormones compared with the immunosuppressive effect of androgens following haemorrhage and sepsis.¹⁷ The finding is nonetheless inconsistent across the literature, with other series reporting higher mortality in men or no difference at all,¹⁵ and with only 23 women in our cohort the observation should be regarded as hypothesis-generating.

Sepsis and pulmonary infection together caused 90% of cases, and direct and indirect injury contributed equally. The slightly higher mortality after direct injury was not significant, in keeping with the comparative Indian analysis of pulmonary and extrapulmonary ARDS.⁸ Of the bedside findings, only pallor predicted death. Taken together with the significantly lower haematocrit among non-survivors, this suggests that pallor was a visible marker of anaemia rather than an independent sign, and that reduced oxygen-carrying capacity compounded the impairment of oxygen transfer. Inotrope requirement, identified in four-fifths of the cohort, was the single strongest clinical predictor, reflecting the circulatory failure that so often accompanies severe hypoxaemia.

The computed tomography findings reproduce a well-described distinction. Diffuse consolidation clustered in direct injury and ground-glass opacity in indirect injury, matching the asymmetric consolidation of pulmonary ARDS and the more homogeneous pattern of extrapulmonary ARDS described by Goodman and Desai and their colleagues.^{18,19} Consolidation also carried the worse prognosis, as thin-section computed tomography studies of prognostication have found.²⁰ That a pattern obtainable on the day of diagnosis distinguishes outcome is of practical value, although transfer of an unstable patient to the scanner carries its own risk, and the five patients not scanned here were among the sickest.

The most informative prognostic signal was dynamic rather than static. Baseline and day 3 SOFA scores were indistinguishable between groups, yet the change between them separated survivors from non-survivors cleanly, and no patient who died showed a falling score. A single admission score describes how ill a patient is; the trajectory describes whether treatment is working, and it is the latter that discriminated here.

This study has clear limitations. It is a single-centre observational series of 50 patients, which restricts power — several associations of plausible clinical importance, including diabetes and alcohol use, moved in a consistent direction without reaching significance. No multivariable analysis was attempted, so the associations reported are unadjusted and may be confounded. The AECC rather than the Berlin definition was applied, which limits comparability with contemporary series. Outcome was not ascertained in the seven transferred patients. Finally, as a tertiary referral unit receiving patients already ventilated elsewhere, our measurements of intensive care stay and ventilation duration do not capture the full course of illness, which may explain why neither was associated with outcome.

CONCLUSION

Acute respiratory distress syndrome carried 66% in-hospital mortality in this tertiary care cohort, and affected a population whose mean age was below 45 years. Female sex, pallor with low haematocrit, inotrope requirement, higher heart rate and positive end-expiratory pressure, worse oxygenation, a diffuse consolidative pattern on computed tomography and a rising SOFA score over the first three days were each associated with death, while age, comorbidity, smoking, alcohol use, culture positivity and the organism isolated were not. The prognostic value lay in the trajectory of organ dysfunction rather than in any single admission measurement. That so severe a syndrome falls predominantly on young adults and, at times, on adolescents in this setting strengthens the argument for early recognition and for prospective multicentre study using contemporary diagnostic criteria.

Table 1. Demographic profile by outcome (n = 50)

Variable	Died (n = 33)	Survived (n = 17)	Total (n = 50)	p
Age group (years)				
18–20	2 (6.1)	0 (0.0)	2 (4.0)	0.504
21–30	7 (21.2)	4 (23.5)	11 (22.0)	
31–40	7 (21.2)	4 (23.5)	11 (22.0)	
41–50	3 (9.1)	5 (29.4)	8 (16.0)	
51–60	5 (15.2)	1 (5.9)	6 (12.0)	
61–70	5 (15.2)	2 (11.8)	7 (14.0)	
71–75	4 (12.1)	1 (5.9)	5 (10.0)	
Mean age, years	45.3 ± 18.5	44.1 ± 15.2	44.9 ± 17.3	0.817
Sex				
Female	19 (57.6)	4 (23.5)	23 (46.0)	0.022
Male	14 (42.4)	13 (76.5)	27 (54.0)	
Outcome				
Died in hospital	33 (100.0)	—	33 (66.0)	—
Recovered	—	10 (58.8)	10 (20.0)	
Referred elsewhere	—	7 (41.2)	7 (14.0)	

Values are n (%) or mean ± standard deviation. Age groups compared by chi-square test; mean age by unpaired t test. Sex: chi-square p = 0.022, Fisher's exact p = 0.036. Patients referred elsewhere had no ascertained final outcome and were analysed as survivors.

Table 2. Aetiology and category of lung injury by outcome (n = 50)

Aetiology	Died (n = 33)	Survived (n = 17)	Total (n = 50)	p
Precipitating disorder				
Sepsis	15 (45.5)	9 (52.9)	24 (48.0)	0.581
Pulmonary infection	15 (45.5)	6 (35.3)	21 (42.0)	
Aspiration	2 (6.1)	1 (5.9)	3 (6.0)	
Intestinal perforation	0 (0.0)	1 (5.9)	1 (2.0)	
Non-thoracic trauma	1 (3.0)	0 (0.0)	1 (2.0)	
Category of lung injury				
Direct	18 (54.5)	7 (41.2)	25 (50.0)	0.370
Indirect	15 (45.5)	10 (58.8)	25 (50.0)	

Values are n (%). Chi-square test. Direct versus indirect injury: Fisher's exact p = 0.551.

Table 3. Presenting clinical features, comorbidity and inotrope requirement by outcome (n = 50)

Feature	Died (n = 33)	Survived (n = 17)	Total (n = 50)	p
Symptoms and signs				
Dyspnoea	33 (100.0)	17 (100.0)	50 (100.0)	—
Crepitations	33 (100.0)	17 (100.0)	50 (100.0)	—
Fever	31 (93.9)	17 (100.0)	48 (96.0)	0.300
Agitation	24 (72.7)	11 (64.7)	35 (70.0)	0.558

Feature	Died (n = 33)	Survived (n = 17)	Total (n = 50)	p
Cough	18 (54.5)	5 (29.4)	23 (46.0)	0.091
Rhonchi	12 (36.4)	6 (35.3)	18 (36.0)	0.941
Pallor	14 (42.4)	1 (5.9)	15 (30.0)	0.008
Disorientation	9 (27.3)	4 (23.5)	13 (26.0)	0.775
Oedema	7 (21.2)	1 (5.9)	8 (16.0)	0.161
Icterus	5 (15.2)	2 (11.8)	7 (14.0)	0.744
Cyanosis	2 (6.1)	2 (11.8)	4 (8.0)	0.481
Clubbing	1 (3.0)	1 (5.9)	2 (4.0)	0.626
Lymphadenopathy	0 (0.0)	2 (11.8)	2 (4.0)	0.044
Comorbidity and habits				
Diabetes mellitus	11 (33.3)	3 (17.6)	14 (28.0)	0.242
Hypertension	4 (12.1)	3 (17.6)	7 (14.0)	0.594
Smoking	6 (18.2)	4 (23.5)	10 (20.0)	0.654
Alcohol use	2 (6.1)	4 (23.5)	6 (12.0)	0.072
Support				
Inotrope requirement	30 (90.9)	10 (58.8)	40 (80.0)	0.007

Values are n (%). Chi-square test. Confirmatory Fisher's exact p values: pallor 0.009; inotrope requirement 0.021; lymphadenopathy 0.111.

Table 4. Haemodynamic, respiratory and laboratory parameters by outcome (n = 50)

Parameter	Died (n = 33)	Survived (n = 17)	t	p
Heart rate, beats/min	115.70 ± 16.30	103.65 ± 24.74	2.067	0.044
Systolic blood pressure, mmHg	110.79 ± 22.57	107.29 ± 15.52	0.571	0.571
Diastolic blood pressure, mmHg	69.09 ± 13.31	63.88 ± 9.68	1.427	0.160
Mean arterial pressure, mmHg	82.95 ± 15.77	78.34 ± 10.62	1.085	0.283
Positive end-expiratory pressure, cm H ₂ O	13.39 ± 2.62	10.47 ± 3.52	3.318	0.002
Arterial oxygen tension, mmHg	69.79 ± 17.59	92.66 ± 33.30	-3.192	0.002
Inspired oxygen fraction	0.90 ± 0.13	0.82 ± 0.15	1.903	0.063
Oxygen tension / inspired fraction	78.54 ± 24.65	115.10 ± 41.30	-3.925	<0.001
pH	7.32 ± 0.15	7.38 ± 0.19	-1.150	0.256
Bicarbonate, mmol/L	22.45 ± 5.80	23.52 ± 5.59	-0.628	0.533
Carbon dioxide tension, mmHg	35.99 ± 11.59	38.49 ± 9.08	-0.774	0.443
Haematocrit, %	31.14 ± 7.22	35.50 ± 4.59	-2.259	0.028
Leucocyte count, /mm ³	16069 ± 13079	12130 ± 5710	1.180	0.244
Platelet count, ×10 ³ /mm ³	173.6 ± 129.7	217.7 ± 238.2	-0.852	0.399
Serum creatinine, mg/dL	1.44 ± 0.73	1.47 ± 0.86	-0.140	0.890
Serum bilirubin, mg/dL	2.31 ± 1.89	2.09 ± 1.96	0.382	0.704
Serum albumin, g/dL	2.48 ± 0.62	2.63 ± 0.41	-0.865	0.391

Parameter	Died (n = 33)	Survived (n = 17)	t	p
C-reactive protein, mg/L	178.42 ± 80.45	210.18 ± 43.32	-1.511	0.138
Procalcitonin, ng/mL	10.27 ± 31.57	4.80 ± 7.79	0.700	0.487

Values are mean ± standard deviation; unpaired Student t test. C-reactive protein and serum albumin were each unavailable in one patient.

Table 5. Radiological, microbiological and organ dysfunction findings by outcome

Finding	Died	Survived	Total	p
Chest radiograph (n = 50; 33 died, 17 survived)				
Diffuse bilateral infiltrate	30 (90.9)	13 (76.5)	43 (86.0)	0.163
With mild pleural effusion	3 (9.1)	4 (23.5)	7 (14.0)	
Computed tomography (n = 45; 29 died, 16 survived)				
Ground-glass opacity	14 (48.3)	10 (62.5)	24 (53.3)	0.013
Diffuse consolidation	15 (51.7)	3 (18.8)	18 (40.0)	
Multifocal patchy involvement	0 (0.0)	3 (18.8)	3 (6.7)	
Culture (n = 50)				
No growth	15 (45.5)	10 (58.8)	25 (50.0)	0.479
Endotracheal aspirate	8 (24.2)	4 (23.5)	12 (24.0)	
Bronchoalveolar lavage	7 (21.2)	1 (5.9)	8 (16.0)	
Urine	2 (6.1)	1 (5.9)	3 (6.0)	
Serology	0 (0.0)	1 (5.9)	1 (2.0)	
Throat swab	1 (3.0)	0 (0.0)	1 (2.0)	
Organ dysfunction and support (n = 50)				
SOFA score, day 1	7.82 ± 2.07	7.82 ± 3.17	7.82 ± 2.46	0.994
SOFA score, day 3	9.12 ± 2.68	8.24 ± 3.77	8.82 ± 3.08	0.341
Change in SOFA score	+1.30 ± 1.02	+0.41 ± 1.37	+1.00 ± 1.21	0.012
Score increased	24 (72.7)	6 (35.3)	30 (60.0)	0.014
Score unchanged	9 (27.3)	9 (52.9)	18 (36.0)	
Score decreased	0 (0.0)	2 (11.8)	2 (4.0)	
Intensive care stay, days	9.00 ± 4.87	9.29 ± 4.01	9.10 ± 4.56	0.831
Mechanical ventilation, days	7.09 ± 4.38	6.71 ± 3.29	6.96 ± 4.02	0.752

Values are n (%) or mean ± standard deviation. SOFA = Sequential Organ Failure Assessment. Computed tomography pattern by category of lung injury: chi-square $p < 0.001$. Organisms isolated (n = 25): Acinetobacter species 9, extended-spectrum beta-lactamase-producing Klebsiella 8, Pseudomonas 4, Escherichia coli 3, methicillin-resistant Staphylococcus aureus 3, influenza A(H1N1) 2, dengue 1 (some patients had more than one isolate); organism isolated versus outcome $p = 0.441$.

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