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Original Research Article

Correlation between Chest CT Severity Score and Clinical Outcomes in Patients with Community-Acquired Pneumonia

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Abstract

Background: Management of community-acquired pneumonia (CAP) is guided by clinical scores but does not quantify pulmonary involvement; chest CT severity scoring may provide additional prognostic information. There is a lack of evidence regarding the relationship between CAP severity score at admission and clinical outcomes in adults hospitalized for CAP.

Objective: To assess the correlation between the severity score at CAP admission and clinical outcomes in adults hospitalized with CAP.

Methods: A prospective observational study of 186 adults with radiologically confirmed CAP who had clinically indicated chest CT within 24 hours of admission. Five lobes were scored from 0 to 5 based on the percentage of involvement, resulting in a total CT severity score (CTSS) of 0-25. The radiologists were blinded to the follow-up results. CTSS was correlated with Pneumonia Severity Index (PSI), CURB-65, C Reactive Protein (CRP), oxygenation, intensive care unit (ICU) admission, invasive or non-invasive ventilation, length of stay, and 30 day mortality.

Results: Mean CTSS was 10.2±5.1. CTSS correlated with PSI ($r=0.62$; $p<0.001$), CRP ($r=0.49$; $p<0.001$), and inversely with the SpO2/FiO2 ratio ($r=-0.58$; $p<0.001$). Patients with CTSS≥12 had higher ICU admission (43.2% versus 8.9%; $p<0.001$), ventilatory support (24.3% versus 3.6%; $p<0.001$), median hospital stay (10 versus 6 days; $p<0.001$), and 30-day mortality (14.9% versus 2.7%; $p=0.003$). The area under the curve (AUC) for predicting ICU admission was 0.86 at a cutoff of 11 and mortality was 0.82 at a cutoff of 14 with CTSS. For each one-point increase in CTSS, the odds of admission to the ICU were increased by 22% (adjusted OR 1.22; 95% CI 1.10-1.35; $p<0.001$) after adjusting for age, comorbidity, PSI, and oxygenation.

Conclusions: CTSS was highly correlated with severity and adverse outcomes in hospitalized CAP. It can be used in conjunction with existing bedside scores if CT has been performed for clinical indications.

Keywords: Community-Acquired Pneumonia; Chest CT; CT Severity Score; Intensive Care; Mortality; Prognosis.

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Introduction

CAP continues to be a significant cause of hospitalization, respiratory failure, and infection-related death. The clinical course is variable, from mild focal disease that can be treated in an outpatient setting to a rapidly progressive course of multilobar pneumonia that may require ventilatory and circulatory support. Early assessment should detect those patients who are likely to deteriorate and should not lead to unnecessary intensive care or widespread investigations in those who are not likely to deteriorate. The Pneumonia Severity Index (PSI) was created to categorize the risk of mortality based on demographic, comorbidity, examination, laboratory and radiographic variables [1]. CURB-65 is a simpler bedside tool that combines the five elements of confusion, urea, respiratory rate, blood pressure and age [2]. The current guidelines suggest that validated severity assessment be combined with clinical judgement in determining the site of care and assessing severe CAP [3]. These tools can be helpful, but they come with some caveats. Age and comorbidity are significant factors in determining PSI, and CURB-65 may underestimate hypoxemia and/or the severity of parenchymal disease. Both scores do not directly assess the percentage of lung involved, distribution

of opacities, necrosis, pleural disease or complications. X-ray of the chest is the first imaging modality to be used, but the extent of the disease is not always easy to quantify due to overlap, projection and limited sensitivity for subtle or posterior disease. CT helps to visualize consolidation, ground glass opacity, centrilobular nodules, cavitation, pleural effusion, abscess and other diagnoses. CT is not routinely performed on all patients with CAP, but is used when the chest radiograph is unclear, complications are suspected, response is unusual, or the disease is severe and needs to be further characterized. Low-dose CT studies have shown that CT can improve the determination of the extent and diagnostic certainty of pneumonia [4]. This anatomical information could be translated to a useful prognostic parameter by using a semiquantitative lobe-based score.

In outbreaks of viral pneumonia, especially COVID-19, severity scoring of CT has become a popular study. In that context, CTSS has been shown to be correlated with oxygen requirement, inflammatory markers, intensive care and mortality in several cohorts [5]. But the distribution, pathophysiology, host response to and prevalence of focal lobar consolidation differ between bacterial and non-COVID CAP. Direct extrapolation is thus not suitable. Severe CAP also includes aspiration, atypical pathogens and mixed infections, all of which have unique patterns and outcomes [6]. There is limited evidence to support the standardized 0-25 CTSS and outcomes in heterogeneous adult CAP. In the present prospective study, the authors determined the performance of CTSS in patients who were clinically indicated for CT within 24 hours of admission. The main goal was to identify correlations with PSI, CURB-65, inflammatory markers and oxygenation. Secondary endpoints included the prediction of admission to the ICU, ventilatory support, length of hospital stay, and 30-day mortality, and the evaluation of whether CTSS provided prognostic information in addition to the known clinical variables.

Materials and Methods

Study design and participants: This was an 18-month, prospective observational cohort study in the emergency, and respiratory medicine and radiology departments of a tertiary hospital. A screening was performed in consecutive adults admitted with the diagnosis of acute lower respiratory infection and with a new pulmonary infiltrate. CAP was considered to be pneumonia occurring in the community or within 48 hours of admission to hospital without a recent alternative nosocomial source. Patients were not included if they had undergone chest CT within 24 hours for a clinical indication, but not for research purposes. HAP/VAP, active pulmonary TB, primary lung malignancy causing the index opacity, major pulmonary edema without infection, profound neutropenia, and inadequate CT images for scoring were excluded. Viral-bacterial coinfection patients were included in the study when the treating team diagnosed CAP, and patients with confirmed SARS-CoV-2 infection were excluded to ensure a non-COVID cohort. There were 201 eligible patients with complete imaging and 30-day outcome data.

The clinical assessment and management of the patient: Demographic data, smoking, comorbidities, symptoms, vital signs, mental status, and oxygen supplementation were obtained at admission. Laboratory tests performed included complete blood count, urea, creatinine, liver tests, electrolytes, CRP, procalcitonin (if available), lactate and arterial blood gas (ABG) in cases of respiratory compromise. PSI and CURB-65 were computed prior to outcomes. SpO2/FiO2 ratio was calculated by pulse oximetry and estimated FiO2. Microbiological tests were conducted according to clinical procedures and involved blood culture, sputum culture, urinary antigen, respiratory viral testing or bronchoscopy as appropriate.

The treating clinicians who were not given the research CTSS determined antimicrobial therapy, fluid management, oxygen, non-invasive ventilation, invasive ventilation and ICU transfer. Severe CAP care was provided through institutional pathways in line with guideline-based management. The primary outcomes were admission to the Intensive Care Unit (ICU) and all-cause mortality at 30 days. Secondary outcomes included any positive pressure ventilatory support, vasopressor use, hospital length of stay and clinical deterioration within 72 hours.

The acquisition of CT, and the severity scoring: Multidetector scanners with inspiratory volumetric imaging were used to acquire CT examinations. Intravenous contrast was acceptable if there was suspicion of pulmonary embolism, abscess, empyema or other complications, otherwise non-contrast CT was acceptable for parenchymal scoring. Thin section and lung and mediastinal algorithms were used to reconstruct images. The examinations were independently read by two thoracic radiologists blinded to the outcomes and final clinical severity scores.

Acute infectious opacity was scored on each pulmonary lobe as follows: 0, none; 1, less than 5%; 2, 5%-25%; 3, 26%-49%; 4, 50%-75%; and 5, more than 75%. The five lobes were scored and added together to create a CTSS 0-25 score. When these were thought to be part of the acute pneumonia they were included in the consolidation, ground-glass opacity and tree-in-bud or nodular inflammatory change. Old scars, chronic fibrosis,

emphysema, and atelectasis (without infection) were excluded. Pleural effusion, cavitation, abscess, lymphadenopathy and distribution were recorded separately. Disagreements among readers of more than two points were settled by consensus and otherwise the mean reader score was taken.

Statistical Analysis: Patients were divided into groups based on a prespecified CTSS threshold of 12 for descriptive comparisons and based on the results of ROC analysis for optimal cutoffs.

Continuous variables were compared using t tests or Mann-Whitney tests and categorical variables were compared using chi-square or Fisher exact tests. Spearman correlations were used to evaluate the relationships between CTSS and PSI, CURB-65, CRP, procalcitonin and oxygenation. ICU admission and mortality were analyzed using logistic regression. The following model variables were included: age, sex, major comorbidity, PSI class, SpO2/FiO2, lactate, and CTSS; the variable selection was limited by the number of outcomes.

AUCs with 95% confidence intervals were used to express discrimination and compared using DeLong tests. Intraclass correlation was used to quantify interobserver reliability. A p value of < 0.05 was considered significant for two-sided tests.

Results

The cohort had a mean age of 58.4±16.7 years; 111 participants (59.7%) were men. Common comorbidities were diabetes (29.0%), chronic lung disease (24.2%), cardiovascular disease (22.6%), and chronic kidney disease (9.7%).

The median interval from arrival to CT was 9.2 hours. Mean CTSS was 10.2±5.1, and 74 patients had CTSS≥12. Higher-score patients were older, more frequently hypoxemic, and had higher respiratory rate, CRP, urea, lactate, PSI, and CURB-65 values (Table 1)

Table 1: Admission characteristics according to CT severity score

Variable	CTSS <12 (n=112)	CTSS ≥12 (n=74)	p-value
Age, years	54.3±16.2	64.6±15.5	<0.001
Male sex, n (%)	63 (56.3)	48 (64.9)	0.24
Any major comorbidity, n (%)	65 (58.0)	52 (70.3)	0.09
Respiratory rate, breaths/min	23.1±4.8	29.4±6.2	<0.001
SpO2/FiO2 ratio	365±72	251±79	<0.001
CRP, mg/L	84 (46-142)	176 (104-258)	<0.001
Lactate, mmol/L	1.6±0.7	2.3±1.1	<0.001
PSI score	78±24	112±31	<0.001
CURB-65 ≥3, n (%)	11 (9.8)	29 (39.2)	<0.001
Multilobar involvement, n (%)	28 (25.0)	58 (78.4)	<0.001

CTSS correlated positively with PSI (r=0.62), CURB-65 (r=0.47), CRP (r=0.49), procalcitonin (r=0.32), and respiratory rate (r=0.44), and inversely with SpO2/FiO2 (r=-0.58); all correlations were statistically significant. Multilobar involvement was present in 78.4% of patients with CTSS≥12 versus 25.0% below 12. Pleural effusion and cavitation were associated with longer admission but were less strongly correlated with oxygenation than total parenchymal extent (Table 2).

Table 2: Correlation of CTSS with clinical and laboratory severity

Variable	Correlation with CTSS (r)	p-value
Pneumonia Severity Index	0.62	<0.001
CURB-65 score	0.47	<0.001
C-reactive protein	0.49	<0.001
Procalcitonin	0.32	<0.001
Respiratory rate	0.44	<0.001
SpO2/FiO2 ratio	-0.58	<0.001
Serum urea	0.31	<0.001
Serum lactate	0.38	<0.001
Hospital length of stay	0.52	<0.001

ICU admission occurred in 42 patients, ventilatory support in 22, vasopressor treatment in 15, and 30-day death in 16.

All adverse outcomes were more frequent when CTSS was 12 or higher. Median length of stay was 10 days in the high-score group and 6 days in the lower-score group. CTSS predicted ICU admission with AUC 0.86; a cutoff of 11 provided 81.0% sensitivity and 78.5% specificity.

For mortality, AUC was 0.82 and a cutoff of 14 provided 75.0% sensitivity and 77.1% specificity (Table 3).

Table 3: Clinical outcomes and prognostic performance

Outcome	CTSS <12	CTSS ≥12	p-value / performance
ICU admission, n (%)	10 (8.9)	32 (43.2)	<0.001
Ventilatory support, n (%)	4 (3.6)	18 (24.3)	<0.001
Vasopressor use, n (%)	3 (2.7)	12 (16.2)	0.001
Deterioration within 72 h, n (%)	8 (7.1)	27 (36.5)	<0.001
Length of stay, median (IQR)	6 (4-8) days	10 (7-14) days	<0.001
30-day mortality, n (%)	3 (2.7)	11 (14.9)	0.003
CTSS for ICU admission	Cutoff 11	AUC 0.86	Sensitivity 81.0%; specificity 78.5%
CTSS for mortality	Cutoff 14	AUC 0.82	Sensitivity 75.0%; specificity 77.1%

In multivariable analysis, CTSS remained associated with ICU admission after adjustment for age, major comorbidity, PSI class, and SpO2/FiO2 (adjusted odds ratio per point 1.22; 95% CI 1.10-1.35; p<0.001). Adding CTSS to a model containing PSI and oxygenation increased AUC for ICU admission from 0.84 to 0.90 (p=0.04). The mortality model was limited by 16 events; CTSS remained directionally associated (adjusted odds ratio per point 1.14; 95% CI 1.01-1.29; p=0.04). Interobserver reliability for total CTSS was excellent (intraclass correlation 0.91).

Discussion

Admission CTSS was moderately to strongly correlated with PSI, CURB-65, inflammatory markers, and oxygenation, and was significantly elevated in patients who needed intensive care or ventilatory support or prolonged hospital stay. This score was still linked to ICU admission in the adjusted model, which included the following clinical parameters: age, sex, temperature, blood pressure, respiratory rate, oxygen saturation, and heart rate. These results indicate that the anatomical extent of acute parenchymal involvement is a prognostic factor in addition to the demographic, morbidity and bedside physiology. Despite the fact that there is not always a full set of clinical criteria, early deterioration can be observed, with severe CAP reviews highlighting the need for multidimensional assessment [7].

The inverse correlation with SpO2/FiO2 was the strongest physiological correlation. The greater the percentage of lung involved, the more likely shunt, ventilation-perfusion mismatch, reduced aerated volume and work of breathing will get worse. The score is not directly related to the recruitability, the perfusion or the microbiological virulence, but it reflects a visible part of the gas-exchange impairment. There was a strong correlation between PSI and the other factors that tended to be associated with extensive disease, namely, the age of the patient, systemic inflammation and physiologic abnormalities. The increased AUC gain indicates that CTSS was not just replicating PSI, but further validation is needed. The 0-25 method is easy to perform and repeatable and is based on lobes. The wide percentage ranges and thin-section CT may account for the excellent interobserver agreement. In COVID-19, the use of similar scoring systems gained traction, with correlation between CT extent and clinical severity and short-term outcome [5]. The current cohort however did not include COVID-19 and included focal bacterial consolidation, atypical patterns, aspiration and mixed etiologies. The direction of the prognosis is similar and suggests a generic association between parenchymal burden and respiratory risk, although there may be pathogen-specific variations.

CT should not be routinely used to determine a severity score in all patients with CAP. Incidental findings, cost, transport, and risk of radiation and contrast should be taken into account. Guidelines recommend clinical assessment and chest radiography, and only consider CT for diagnostic uncertainty, suspected complication, treatment failure, or severe presentation [3]. The role of CT is therefore opportunistic: If CT has already been acquired for a legitimate clinical indication, then structured scoring can be used to gain prognostic information at little extra cost. Low dose protocols can be used if clinically necessary to reduce exposure.

CTSS also has some limitations as a measure of anatomy. A small lesion in a vulnerable patient may result in significant hypoxemia while a large lesion may be well tolerated in a younger patient with high cardiopulmonary reserve. Chronic emphysema or fibrosis should not be considered as acute pneumonia but should be counted as a decrease in reserve. The presence of pleural effusion, empyema, cavitation, airway obstruction and pulmonary embolism may have a significant impact on outcome without a corresponding increase in parenchymal score. Therefore, the use of CTSS should be combined with, rather than replacing, oxygenation, hemodynamics, organ dysfunction, comorbidity and microbiological assessment.

The 14-death cut-off had reasonable discrimination but should be used with caution as there were only 16 deaths. Mortality in CAP is due to treatment delay, goals of care, frailty, pathogen, resistance, immune status and complications. There have been systematic comparisons between PSI and CURB-

65, which have demonstrated that the two prognostic tools have different sensitivities and are also different in terms of operational simplicity [8]. CTSS might be most helpful for decisions on escalation rather than as a standalone mortality prediction tool. Repeat CT might also be useful for serial change but should be limited to clinical indications due to the extra radiation exposure.

Prospective enrollment, early imaging, blinded independent scoring, validated clinical comparators, complete 30-day follow-up, and multivariable analysis are strengths. A major limitation is selection bias as only patients with a clinical indication for CT were included, which makes for a more complex and/or more severe clinical population and therefore limits the generalizability to all CAP. The study was conducted at a single center and some patients were not definitively diagnosed with the etiology, and treatment could have been affected by the CT images even though the clinicians were not given the research score. The threshold was derived from within and the mortality model had few events. The cutoffs should be validated in a multicenter study, pathogen and phenotype subgroups should be explored, and the CTSS should be compared to quantitative automated lung-volume methods.

There are a number of prognostic models that can be used in conjunction with anatomical scoring. Short- and long-term CAP mortality can be predicted by biomarkers of cardiovascular stress and vasopressin activation [9].

The delay in the recognition of patients requiring intensive care has been associated with poor outcomes in severe-CAP prediction rules [10] and the PIRO model offers structured risk estimation in patients in the intensive care unit (ICU) [11]. However, CT is also useful for detecting pneumonia that is not detected on concurrent radiography but does not necessarily suggest a different pathogen spectrum or a more benign clinical course [12].

Prospective diagnostic studies have shown that early CT can significantly reclassify suspected CAP [13] and can provide information about complications or alternative diagnoses in severe disease [14]. Other cohorts have shown that CT is associated with less underdiagnosis and overdiagnosis when the radiography is indeterminate [15].

The use of CT extent in existing clinical scores could enhance the ability to discriminate prognosis [16] and CT characteristics of pleural infection can help predict prolonged hospitalisation [17]. A recent study that integrated CT severity with inflammatory or nutritional biomarkers further reinforces multimodal risk assessment [18].

Conclusion

The severity score of chest CT was associated with existing pneumonia severity indices, inflammation, oxygenation impairment, intensive care unit (ICU) admission, ventilatory support, length of stay, and 30-day mortality. A higher CTSS independently identified patients who were at high risk of admission to the ICU, and when combined with clinical severity and oxygenation, enhanced discrimination. The score should not be used as an indication to use a routine CT in uncomplicated CAP, but rather as a structured score to complement clinical indications for CT.

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