Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.
Severe *Legionella* pneumonia: Rapid presumptive clinical diagnosis with Winthrop-University Hospital’s weighted point score system (modified)

Burke A. Cunha, MD

Legionnaires’ disease is a systemic infection involving the lungs and accompanied by a characteristic pattern of extrapulmonary organ involvement. Legionnaires’ disease is one of the non-zoonotic causes of atypical community-acquired pneumonia (CAP). Legionnaires’ disease commonly presents as severe CAP requiring hospitalization and intensive care. Each atypical CAP has its own characteristic pattern of extrapulmonary laboratory clinical findings and abnormalities that are the basis of clinical syndromic diagnosis. Studies have been unsuccessful in identifying individual clinical and laboratory parameters that are specific for *Legionella*. Individually, clinical and laboratory abnormalities lack diagnostic specificity. The diagnostic specificity of clinical and laboratory findings is increased when combined and are the basis of a clinical syndromic diagnosis. The importance of serial nonspecific laboratory abnormalities with Legionnaires’ disease is emphasized. The sensitivity and specificity of a clinical syndromic diagnosis are enhanced if they are based on a weighted point score system. A diagnostic weighted point score system is based on the varying diagnostic importance of clinical and laboratory diagnostic findings. The Winthrop-University Hospital’s Infectious Disease Division’s rapid clinical diagnostic weighted point score system is based on a weighted point score of clinical and laboratory findings. The case presented is that of a 55-year-old man with severe CAP who required hospitalization and intensive care admission. The presumptive clinical diagnosis of *Legionella* CAP was based on the Winthrop-University Hospital Infectious Disease Division’s rapid clinical diagnostic weighted point score system, which permitted early empiric anti-*Legionella* antimicrobial therapy and prompted specific *Legionella* testing. Legionnaires’ disease is definitively diagnosed by serology or a urinary *Legionella* antigen test. This case of severe Legionnaires’ CAP was confirmed by urinary antigen test reported on hospital day 6. The Winthrop-University Hospital’s weighted point score system (modified) permits a rapid clinical presumptive diagnosis of Legionnaires’ disease and is an accurate predictor of *Legionella* CAP. (Heart Lung® 2008;37:311–320.)

Legionnaires’ disease is a systemic infection that involves the lungs and that is one of the causes of non-zoonotic atypical community-acquired pneumonia (CAP). Typical bacterial causes of CAP are usually *Streptococcus pneumoniae*, *Haemophilus influenzae*, or *Moraxella catarrhalis*. With typical bacterial CAP, clinical signs and symptoms are confined to the chest and lungs. In contrast, atypical pneumonias are characterized by extrapulmonary involvement. The atypical CAPs may be differentiated by each pathogen’s characteristic pattern of extrapulmonary findings in patients with CAP. Atypical pneumonias may be classified as zoonotic when they are caused by Q fever, psittacosis, or tularemia, or as non-zoonotic when they are caused by *Clamydia pneumoniae*, *Mycoplasma pneumoniae*, or *Legionella* species. The clinical differentiation of CAP pathogens, whether zoonotic or non-zoonotic, depends on recognizing each of the characteristic patterns of extrapulmonary organ involvement. In atypical CAP, there is some overlap with individual clinical findings, but each has its own pattern of organ involvement.

**CLINICAL DIAGNOSIS OF LEGIONNAIRES’ DISEASE**

Various attempts have been made to identify the distinguishing features of Legionnaires’ disease to permit a presumptive and working clinical diagno-
sis. Many studies have attempted to identify single clinical, radiologic, or laboratory abnormalities that would distinguish Legionnaires’ disease from other atypical and typical causes of CAP. Such attempts have failed largely because isolated findings are nonspecific. Many studies have compared a fever level (eg, >101°F) without taking into account the degree and duration of the fever. As expected, the degree and duration of the fever would vary considerably by pathogen, medications, and cardiopulmonary status. The maximum degree of the temperature, for example, is of some value in differentiating M. pneumoniae or C. pneumoniae.

A pulse temperature deficit (Faget’s sign) is termed “relative bradycardia.” Relative bradycardia refers to an inappropriate pulse response for a given degree of temperature greater than 102°F in patients without arrhythmias, pacemaker rhythms, or pulse-modifying medications (β-blockers, diltiazem, verapamil). Relative bradycardia is determined by noting a significant discrepancy between the pulse rate and the temperature from what would be expected for any degree of temperature. Physiologically, for each degree of temperature elevation (Fahrenheit) there is a commensurate increase of 10 beats/min in the pulse rate. For example, in a patient with a 104°F fever, the appropriate pulse response should be 130 beats/min. If the pulse response is considerably less than this (ie, <120 beats/min), then relative bradycardia is present.

In the literature on Legionella, the presence or absence of relative bradycardia is either not mentioned or not defined. The presence or absence of relative bradycardia can be a critical diagnostic finding in CAP. None of the typical bacterial causes of CAP are associated with relative bradycardia. Among the atypical CAP pathogens, only Q fever, psittacosis, and Legionnaires’ disease are associated with relative bradycardia. Because relative bradycardia is a cardinal sign of Legionnaires’ disease, studies that do not include pulse and temperature relationships have overlooked an important sign that differentiates Legionella from other non-zoonotic atypical CAPs. Zoonotic atypical pathogens associated with relative bradycardia (eg, psittacosis and Q fever) can readily be eliminated from the differential diagnosis in patients with atypical CAPs on the basis of a negative epidemiologic contact history.

A presumptive diagnosis of Legionnaires’ disease is possible using a clinical syndromic approach that is based on the characteristic pattern of extrapulmonary organ involvement. Since Legionnaires’ disease was first described, a myriad of clinical manifestations of Legionnaires’ disease have been described. The chest x-ray appearance of Legionnaires’ disease is not specific, and there are no particular chest x-ray patterns that are pathognomonic for Legionella CAP. However, Legionella CAP typically presents with rapidly progressive and asymmetric infiltrates on the chest x-ray. The behavior of the infiltrates on the chest x-ray in Legionnaires’ disease, rather than their appearance and location or associated findings (ie, cavitation, pleural effusion), should suggest the possibility of Legionella CAP.

Single clinical findings in CAP have not been helpful diagnostically in differentiating Legionnaires’ disease from other causes of CAP. Studies comparing clinical signs and symptoms in Legionnaires’ disease versus other CAPs have found no single parameter that clearly identifies Legionella CAP. Both Legionella and M. pneumoniae, for example, often present with loose stools and watery diarrhea. Other causes of CAP are not associated with these findings, but gastrointestinal manifestations are not always present in all cases of Legionella or mycoplasma CAP. Other clinical parameters, such as shortness of breath and respiratory rate, are nonspecific and have little differential diagnostic value in CAP. The same approach of trying to identify individual markers has been applied to laboratory abnormalities in CAPs. For example, although hyponatremia is the most commonly recognized laboratory abnormality in Legionella CAP, hyponatremia is nonetheless not specific. It is true that if present, the degree of hyponatremia is likely to be greater in Legionella than in other causes of CAP. Because pneumonia may be associated with hyponatremia, per se, it is not specific for Legionella in patients with CAP.

**CLINICAL SYNDROMIC DIAGNOSTIC APPROACH**

In internal medicine and infectious disease, the differential diagnosis is based on a constellation of presenting signs and symptoms that permit a clinical syndromic diagnosis combining various clinical and laboratory findings to increase the diagnostic specificity, which is not possible when comparing isolated clinical parameters. Similarly, with laboratory abnormalities, diagnostic specificity is increased when laboratory abnormalities are combined, whereas hyponatremia CAP is nonspecific and therefore relatively unhelpful. Hypophosphatemia is more specific diagnostically because Legionnaires’ disease is the only cause of CAP that is associated with hypophosphatemia. Patients with CAP and
extrapulmonary findings who have a negative zoonotic contact history with hyponatremia and hypophosphatemia are more likely to have Legionnaires’ disease than if hyponatremia alone is present.\(^5,7,13\)

Other laboratory tests may be used in the clinical syndromic approach to Legionnaires’ disease, including highly elevated C-reactive protein (CRP) levels (\(\geq 30\)), creatine phosphokinase levels, and serum ferritin levels, or microscopic hematuria. These laboratory abnormalities have diagnostic significance in patients with CAP and extrapulmonary findings with a negative zoonotic contact history if they are otherwise unexplained and temporarily related to the pneumonia. By combining select clinical features with laboratory abnormalities, diagnostic specificity is increased. Diagnostic specificity may be further enhanced by using a weighted point scoring system. It is important to realize that the diagnostic specificity of clinical and laboratory findings varies considerably. In view of the relative diagnostic value of clinical and laboratory findings, by using the weighted point score system and taking into account the diagnostic significance of each clinical and laboratory abnormality, the diagnostic specificity of the clinical syndromic approach can be greatly enhanced. The weighted point system is based on the relative diagnostic importance of various clinical and laboratory findings and was first developed by the Winthrop-University Hospital Infectious Disease Division. By assigning different point scores (weight) to each finding, the probability of Legionnaires’ disease can be rapidly determined.\(^5,7,13-16\)

The Winthrop-University Hospital weighted point score system (modified) for the clinical diagnosis of Legionnaires’ disease contains refined clinical and laboratory parameters that are more sensitive and specific than the original version introduced in 1998.\(^16,17\) The Winthrop-University Hospital weighted point modified system has been used for the presumptive clinical diagnosis of Legionnaires’ disease since 1998 at Winthrop-University Hospital. Properly applied, the Winthrop-University Hospital Infectious Disease Division’s weighted point score system is effective in the rapid presumptive diagnosis of Legionella CAP.\(^16,17\)

The rationale for such a weighted clinical syndromic approach is to predict the probability of Legionnaires’ disease in patients with CAP and prompt clinicians to order specific diagnostic testing to confirm or rule out the diagnosis of Legionella disease. The Winthrop-University Hospital’s Infectious Disease Division’s weighted point score system is not intended to be the basis of empiric therapy but rather should serve as the impetus for further diagnostic testing.\(^16,17\) However, the clinical value of using this system permits a rapid presumptive diagnosis that can explain the patient’s clinical course and alerts the clinician to ensure that the antimicrobial therapy chosen has a high degree of anti-Legionella activity.

Clinicians have needed a reliable and effective rapid presumptive diagnostic clinical method to diagnose Legionella CAP. Various studies have looked at individual clinical and laboratory findings to try to identify Legionella CAP. These have been unsuccessful because they are not based on a clinical syndromic approach and omit characteristic Legionella findings or do not take into consideration the relative importance of different clinical and laboratory findings in Legionnaires’ disease.\(^5-10\)

The most rapid way to confirm Legionella CAP is direct fluorescent antibody testing of the sputum. However, all patients with CAP do not bring up sputum, and direct fluorescent antibody positivity occurs early in a minority of patients with Legionella. Furthermore, anti-Legionella antibiotic therapy rapidly decreases direct fluorescent antibody positivity in sputum. Thus, there is a need for a rapid clinical diagnostic method to presumptively diagnose Legionnaires’ disease, particularly in patients with severe CAP.\(^6,7\)

The usual causes of severe CAP are severe cardiopulmonary disease or decreased splenic function. Organisms, per se, are not determinants of CAP severity. \(M.\) catarrhalis can present as severe CAP in patients with borderline pulmonary function (chronic obstructive pulmonary disease). All other things being equal, Legionella clearly is “more virulent” than \(M.\) pneumonii or \(C.\) pneumoniae among the non-zoonotic atypical causes of CAP.\(^7,18\)

Atypical pathogens predominate in the outpatient setting, whereas typical CAP pathogens predominate in hospitalized patients with severe CAP. Patients hospitalized with severe CAPs often require intensive care and ventilatory support. Legionella and \(S.\) pneumoniae are clearly the two predominant severe CAP pathogens. The diagnostic and therapeutic approach to severe \(S.\) pneumoniae CAP is different than to severe Legionella CAP, underscoring the clinical need for rapid presumptive clinical diagnosis. Rapid clinical differentiation to ensure optimal therapy is best accomplished using a weighted point score system that takes into account the differences in the diagnostic importance of clinical and laboratory findings in Legionnaires’ disease.\(^7,18\)

We present a patient with severe Legionella pneumophila (serotype 01) CAP who was admitted to the
hospital and required intensive care. In this patient, the clinical diagnosis of Legionnaires’ disease was based on the modified Winthrop-University Hospital’s Infectious Disease Division’s weighted point score system (Table I). The rapid presumptive diagnosis of Legionnaires’ CAP in this patient permitted appropriate initial antibiotic therapy that resulted in a successful outcome. The laboratory diagnosis of Legionnaires’ disease was reported positive 6 days later.

**ILLUSTRATIVE CASE**

The patient was a 55-year-old man admitted with 1 week’s history of fever (up to 105°F) shortness of breath, and left lower quadrant abdominal pain. A dry cough developed in the patient on the day of admission. The patient’s medical history was significant for diabetes mellitus. Two days before admission, the patient was seen at an urgent care facility where he was told that he had a “viral syndrome,” and no antibiotics were given.

At the time of admission, the patient’s temperature was 105°F with a pulse rate of 118 beats/min, respiratory rate of 32 breaths/min, and blood pressure of 98/63 mm Hg. On physical examination, the patient was hypotensive, hypoxic, lethargic, tachypneic, tachycardic, and dehydrated. Head, ears, eyes, nose, and throat examination results were negative. The cardiac examination results were unremarkable. Auscultation of the lungs revealed bilateral ronchi, with decreased breath sounds in the right middle and lower lobes, and decreased breath sounds over the left lower lobe. The abdomen was soft, but mildly distended, and nontender with bowel sounds present in all four quadrants. Neurologic examination results were unremarkable.

The patient’s admission laboratory test results included the following: The white blood cell count was 11.1 K/mm$^3$ (82% polymorphonuclear cells, 6% lymphocytes, 2% monocytes), hemoglobin was 15.1 g/dL, hematocrit was 46.3%, and platelet count was 15.5 K/mm$^3$. The erythrocyte sedimentation rate was 88 mm/h (n ≤ 20 mm/h), and CRP was 36.4 mg/L (n < 3 mg/L). The sodium level was 131 meq/L, and phosphorus level was 1.5 mg/dL. Blood urea nitrogen was 16 mg/dL, and creatinine was 1 mg/dL. The total bilirubin was 0.5 mg/dL, and direct bilirubin was 0.2 mg/dL. Serum glutamate pyruvate transaminase was 67 IU/L (normal = 4–36 IU/L), serum glutamate oxaloacetate transaminase was 75 IU/L (n = 13–39 IU/L), and serum creatinine phosphokinase was 1005 IU/L (n = 47–422 IU/L). The serum ferritin level was 5990 ng/mL (n ≤ 220 ng/mL). The serum protein electrophoresis was unremarkable. Cold agglutinins were negative. Urinalysis showed microscopic hematuria (90 red blood cells/high-power field). Quantitative urine myoglobin was highly elevated.

Chest x-ray on admission showed a right middle lobe infiltrate and probable left pleural effusion. Repeat chest x-ray performed 7 hours later showed patchy alveolar densities at both lung bases with consolidation on the right middle lobe.

A presumptive clinical diagnosis of legionnaire’s disease was made on the basis of Winthrop University Hospital’s weighted point score system and levofloxacin 500 mg (intravenously) every 24 hours was empirically started. On hospital day 2, the patient became confused and disoriented. A repeat chest x-ray showed an unchanged right middle lobe infiltrate with increased pulmonary vascular congestion, and the patient was admitted to the medical intensive care unit for ventilator support. On hospital day 3, the patient was still confused and disoriented. Because of the patient’s altered mental status, a head computed tomography scan without contrast was performed; the findings were unremarkable. On hospital day 5, the patient showed signs of clinical improvement. Levofoxacin was increased to 750 mg (by mouth) every 24 hours. On hospital day 5, the chest x-ray showed some resolution of the infiltrates in the right middle lobe and lingual segment. The patient’s laboratory abnormalities normalized during his hospitalization (Figs 1–6).

Legionella urinary antigen was reported positive on hospital day 6. The patient was discharged on levofloxacin 750 mg (orally) every 24 hours, completed a 2-week course of therapy.

**DISCUSSION**

In patients with severe CAP, it is important to make a rapid clinical presumptive diagnosis of Legionnaires’ disease to initiate therapy early in the course of infection. Legionella CAP, like other atypical CAPs, is characterized by its own characteristic pattern of extrapulmonary organ involvement and is distinctive for Legionella. Attempts to identify unique clinical and laboratory abnormalities that would be predictive of Legionella CAP have been unsuccessful because clinical and laboratory findings considered alone are nonspecific. However, when clinical findings are assessed in concert, the diagnostic specificity of the clinical findings is increased. Similarly, laboratory abnormalities when considered in con-
### Table I
Winthrop-University Hospital Infectious Disease Division’s weighted point system for diagnosing Legionnaires’ disease† (modified)

| Qualifying conditions                                      | Point score | Illustrative case’s point score |
|------------------------------------------------------------|-------------|---------------------------------|
| **Clinical features**                                       |             |                                 |
| Temperature >102°F*                                        | +5          | +5                              |
| Headache                                                   | +2          |                                 |
| Mental confusion/lethargy*                                 | +4          | +4                              |
| Ear pain                                                   | −3          |                                 |
| Non-exudative pharyngitis                                  | −3          |                                 |
| Hoarseness                                                 | −3          |                                 |
| Sputum (purulent)                                          | −3          |                                 |
| Hemoptysis*                                                | −3          |                                 |
| Chest pain (pleuritic)                                    | −3          |                                 |
| Loose stools/watery diarrhea*                              | +3          |                                 |
| Abdominal pain*                                            | +5          |                                 |
| Renal failure*                                             | +3          |                                 |
| Shock/hypotension*                                         | −5          | −5                              |
| Splenomegaly                                               | −5          |                                 |
| Lack of response to β-lactam antibiotics                   | +5          |                                 |
| **Laboratory features**                                    |             |                                 |
| Chest x-ray                                                | +3          | +3                              |
| ↓ pO₂ with ↑ A-a gradient (>35)*                           | −5          |                                 |
| ↓ Na⁺                                                      | +1          | +1                              |
| Hypophosphatemia                                           | +5          | +5                              |
| ↑ SGOT/SGPT (early, mild/transient)*                       | +2          | +2                              |
| ↑ Total bilirubin                                          | +1          |                                 |
| ↑ LDH (>400)*                                              | −5          |                                 |
| ↑ CPK                                                      | +4          | +4                              |
| ↑ CRP (>30)                                                | +5          | +5                              |
| ↑ Cold agglutinins (≥1:64)                                 | −5          |                                 |
| Severe relative lymphopenia (<10%)*                        | +5          |                                 |
| ↑ Ferritin (>2×n)                                          | +5          |                                 |
| Microscopic hematuria*                                     | +2          | +2                              |

**Likelihood of Legionella**
- >15 Legionella very likely
- 5–15 Legionella likely
- <5 Legionella unlikely

**Patient’s point score**
- 41 (Legionella very likely)

---

*AECB, Acute exacerbation of chronic bronchitis; BPH, benign prostatic hyperplasia; SARS, severe acute respiratory syndrome; SGOT/SGPT, serum glutamate oxaloacetate transaminase/serum glutamate pyruvate transaminase; LDH, lactate dehydrogenase; CPK, creatine phosphokinase; CRP, C-reactive protein.

Adapted with permission from Cunha BA. Pneumonia Essentials (2nd ed.). Royal Oak, MI: Physicians Press; 2008.

*Otherwise unexplained (acute and associated with the pneumonia).
†In adults, otherwise unexplained, acute and associated with the pneumonia.
Fig 1 Serial CRP levels in patient with Legionella CAP.

Fig 2 Serial erythrocyte sedimentation rates in a patient with Legionella CAP. ESR, Erythrocyte sedimentation rate.
Fig 3 Serial serum phosphorus levels in a patient with *Legionella* CAP.

Fig 4 Serial serum transaminase levels in a patient with *Legionella* CAP. *SGOT*, Serum glutamate oxaloacetate transaminase; *SGPT*, serum glutamate pyruvate transaminase.
Fig 5 Serial creatine phosphokinase levels in patient with *Legionella* CAP. CPK, Creatine phosphokinase.

Fig 6 Serial serum ferritin levels in a patient with *Legionella* CAP.
cert have increased diagnostic specificity than when evaluated individually. The clinical syndromic diagnosis of Legionnaires’ disease based on the characteristic pattern of extrapulmonary organ involvement is based on the presence or absence of key clinical signs, symptoms, and laboratory abnormalities.

For example, the likelihood of Legionella in patients with CAP with extrapulmonary findings is unlikely if the patient does not have relative bradycardia, mental confusion, loose stools/watery diarrhea, hypophosphatemia, or early/mild transient elevations of serum transaminases. Patients with atypical CAP without relative bradycardia, liver involvement, or hypophosphatemia are more likely to have M. pneumoniae CAP rather than Legionnaires’ disease. The relative importance of diagnostic findings should be the basis of a clinical syndromic approach.

There are several key clinical abnormalities characteristic of Legionella CAP. Relative bradycardia is a cardinal sign of Legionella CAP. Relative bradycardia occurs with any Legionella species. Headache is common with many acute infectious diseases, but mental confusion in a patient with CAP that is not drug induced or related to acute fever or hypoxemia indicates Legionella. In patients with CAP, although loose stools and diarrhea limit the diagnostic possibilities in M. pneumoniae or Legionella, abdominal pain with or without diarrhea is highly indicative of Legionnaires’ disease.

Among nonspecific laboratory tests, hypophosphatemia, if present, is a helpful marker in pointing to the possibility of Legionnaires’ disease in a patient with CAP. Other causes of hypophosphatemia are excluded. Hypophosphatemia in Legionnaires’ disease occurs early and is easily missed if not ordered on admission day 1 of hospital. Hypophosphatemia is usually overlooked as an important laboratory finding in the Legionella literature. Because the hypophosphatemia in Legionnaires’ disease occurs early and may be absent after the first 24 hours of hospital admission, hypophosphatemia is easily overlooked. Transient hypophosphatemia will develop in some patients without initial hypophosphatemia, and then normal phosphorus levels will rapidly return. More frequently, however, hypophosphatemia occurs in the first 24 hours and is easily missed if the serum phosphorus levels are not obtained until the second day of hospitalization.

Transient elevations of the serum transaminases also occur early, but being mild and easily overlooked, they are regarded as nonspecific or unimportant. Although elevated CRP is nonspecific and increased in many acute infectious and inflammatory processes, creatine phosphokinase is another important indicator of Legionnaires’ disease. In CAP, if CRP is highly elevated (>30), Legionnaires’ disease is likely if accompanied by other characteristic clinical and laboratory findings. Otherwise unexplained microscopic hematuria is another important laboratory marker for Legionnaires’ disease.

The serum ferritin levels seem to be another important marker for Legionnaires’ disease. Serum ferritin is an acute phase reactant in patients with many noninfectious and infectious disorders. As an acute phase reactant, ferritin elevations are slightly above normal, that is, 220 to 280 ng/mL (n ≤ 220 ng/mL). In my experience, patients with Legionnaires’ disease have serum ferritin levels > 2 × n ng/mL. Before ascribing an increase in ferritin levels to Legionnaires’ disease, other causes of increased serum ferritin levels should be excluded (eg, myeloproliferative, neoplastic, and rheumatic disorders) in patients with CAP. However, otherwise unexplained highly elevated serum ferritin levels > 2 × n point to the diagnosis of Legionnaires’ disease. Serum ferritin levels, as with hypophosphatemia, should be obtained on admission and obtained serially. Serum ferritin levels optimally should be obtained on admission. Elevations of serum ferritin levels are more sustained than low serum phosphorus levels and remain elevated for several days before decreasing.

These nonspecific laboratory tests when considered individually are not, per se, indicative of Legionnaires’ disease. However, taken together they are important clinical indicators of Legionnaires’ disease in patients with CAP.

The Winthrop-University Hospital is weighted point score system for the presumptive clinical diagnosis of Legionnaires’ disease is interpreted in terms of Legionella CAP probability. In the Winthrop-University Hospital is weighted point score system, more than 15 points indicates that Legionella is very likely, 5 to 10 points indicate that Legionella is likely, and less than 5 points indicates that Legionella is an unlikely cause of CAP. The Winthrop-University Hospital is weighted point score system assumes that the clinical and laboratory findings being scored are otherwise unexplained, acute, and associated with the patient’s CAP.

In the patient reported, Legionella was rapidly presumptive diagnosed clinically using the Winthrop-University Hospital weighted point score system after the patient was admitted. The patient had severe CAP that required intensive care. A rapid presumptive diagnosis of Legionnaires’ disease was made on the basis of his
clinical and laboratory findings using the modified Winthrop-University Hospital is weighted point score system, which ensured early anti-Legionella therapy.

Winthrop-University Hospital is weighted point score system is intended to provide a rapid presumptive clinical diagnosis and anti-Legionella coverage, and prompt specific testing for Legionnaires’ disease. In this case, as in many others, laboratory confirmation of Legionella disease was reported after the patient had responded to therapy and was discharged from the intensive care unit on hospital day 6.

REFERENCES

1. Kirby BD, Synder KM, Meyer RD, et al. Legionnaire’s disease: clinical features of 24 cases. Ann Intern Med 1978;89:297-309.
2. Cunha BA, Quintiliani R. The atypical pneumonias, a diagnostic and therapeutic approach. Postgrad Med 1979;66:95-102.
3. Cunha BA, Ortega AM. Atypical pneumonia. Extrapulmonary clues guide the way to diagnosis. Postgrad Med 1966;99:123-8.
4. Johnson DH, Cunha BA. Clinical and extrapulmonary features of Chlamydia, mycoplasmas, and Legionella infections. Postgrad Med 1992;93:69-72.
5. Cunha BA. The atypical pneumonias: clinical diagnosis and importance. Clin Microbiol Infect 2006;12:12-24.
6. Cunha BA. Atypical Pneumonia: Clinical Features, Diagnosis, and Management. Current Opinion in Pulmonary Medicine 2008;14:183-194.
7. Cunha BA. Pneumonia essentials. 2nd ed. Royal Oaks, MI: Physicians Press; 2008:55-63.
8. Sugihara E, Dambara T, Aiba M, et al. Clinical characteristics of 8 sporadic cases of community-acquired Legionella pneumonia in advanced age. Intern Med 2007;46:461-5.
9. Sopena N, Pedro-Botet L, Mateu L, et al. Community-acquired Legionella pneumonia in elderly patients: characteristics and outcome. J Am Geriatr Soc 2007;55:114-9.
10. Ben-Dror G, Mizerisky Y, Vier G, et al. The epidemiology and clinical features of Legionella pneumonia (LP) in patients older than 60 years old who were hospitalized with pneumonia in Northern Israel. Harefuah 2002;141:680-2.
11. Cunha BA. Diagnostic Significance of relative bradycardia. Clin Microbiol & Infect Dis 2009;6:633-634.
12. Franzini L, Cabodi D. Legionella pneumonia and serum procalcitonin. Curr Microbiol 2005;50:43-46.
13. Cunha BA. Hypophosphatemia: diagnostic significance in Legionnaire’s disease. Am J Med 2006;119:5-6.
14. Cunha BA. Diagnostic Significance of Non-Specific Laboratory Tests in Infectious Diseases. In: Gorbach SL, Bartlett JB, Blacklow NR (Eds.). Infectious Diseases in Medicine and Surgery (3rd ed.). W.B. Saunders Co., Philadelphia, 2002, pp 116-127.
15. Cunha BA. The Clinical Diagnosis of Legionnaires’ Disease: Diagnostic Value of Combing Non-Specific Laboratory Tests. Journal of Infection 2008;6:395-397.
16. Cunha BA. Clinical diagnosis of Legionnaire’s disease. Semin Respir Infect 1998;13:116-27.
17. Gupta SK, Imperiale TF, Sarosi GA. Evaluation of the Winthrop-University Hospital criteria to identify Legionella pneumonia. Chest 2001;120:1064-1071.
18. Cunha BA. Severe community acquired pneumonia in the critical care unit. In Cunha BA, editor. Infectious diseases in critical care medicine (2nd ed.). New York: Informa Healthcare; 2007:157-68.
19. Cunha BA. Legionella pneumonia: the value of clinical and laboratory findings. J Respir Dis 2005;26:515-16.
20. Cunha BA. Legionnaire’s disease. In: Rackel ER, Bope ED, editors. Conn’s current therapy–2005 (57th ed.). Philadelphia, PA: WB Saunders; 2005.
21. Cunha BA. Serum Ferritin Levels in Legionella Community-acquired Pneumonia. Clinical Infectious Disease 46:1789-1791, 2008.