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Table 1: Rigor Adherence Table

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<u>Inclusion and Exclusion Criteria</u>
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<u>Attrition</u>
No data were reused or generated in this study.
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not required.
<u>Subject Demographics</u>
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Weight: not required.
<u>Randomization</u>
not detected.
<u>Blinding</u>
not detected.
<u>Power Analysis</u>
not detected.
<u>Replication</u>
not required.

Table 2: Key Resources Table

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Access Microbiology

Clinical Value of P² calcitonin as a Biomarker for Detecting a Primary Tuberculous Spinal Infection: Case Report and Review of the Literature.

--Manuscript Draft--

CONFIDENTIAL

1 **Clinical Value of Procalcitonin as a Biomarker for Detecting a Primary Tuberculous**
2 **Spinal Infection: Case Report and Review of the Literature**

3
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16
17 **ABSTRACT:**

18 Procalcitonin is a biomarker, potentially ¹⁶ useful in the diagnosis of sepsis and severe bacterial
19 infections. Few studies have examined the usefulness of this biological marker in the diagnosis
20 of tuberculous spondylodiscitis.

21 ⁷ We report the case of a 37-year-old woman with an unremarkable medical history who was
22 newly diagnosed with tuberculous spondylodiscitis while the procalcitonin test was negative.

23 This case confirms the hypothesis that procalcitonin ¹¹ is not a useful parameter to support the
24 diagnosis of primary tuberculous spondylodiscitis.

25
26 **KEYWORDS:** Procalcitonin biomarker, spinal infection, tuberculosis.

27

28

29 **INTRODUCTION:**

30 Spinal infections are ¹¹ diagnosed through physical examination, laboratory tests, and radiological
31 analysis. Commonly used inflammatory biomarkers in laboratories include ⁴ white blood cell
32 count, C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR). Procalcitonin
33 (PCT) has recently ¹¹ been used to differentiate between bacterial and nonbacterial infections.
34 Infections should be recognized as quickly as possible for physiological and clinical reasons.
35 Through the early detection of damaged tissue, therapeutic and preventive measures can be
36 initiated earlier [1-3].

37 A useful marker for diagnosing bacterial infection is PCT, a prohormone for calcitonin. After
38 infection, PCT secretion begins 4 hours later and peaks after 8 hours, whereas CRP secretion
39 begins 4-6 hours later and peaks only 36 hours later. Serum PCT levels are still useful for
40 clinical purposes and are currently used for four common purposes. To determine ⁵ the likelihood
41 of death in critically ill patients with sepsis. ⁵ Second, PCT values were used to guide patients
42 receiving empirical antibiotic therapy for sepsis, pneumonia, and bronchitis. Third, PCT value,
43 along with other conventional biomarkers, can help assess the effectiveness of empirical
44 antibacterial therapy for the patient. The fourth and most practical ⁵ application is to use
45 sequential PCT values to assess when antibacterial therapy is no longer necessary [4-6].

46 This biomarker has attracted great interest in recent years, particularly ² in the differential
47 diagnosis between bacterial and viral infections. Nevertheless, few ⁴ studies have examined the
48 utility of this biological marker in the diagnosis of infectious spondylodiscitis, particularly
49 tuberculous spondylodiscitis.

50 ² We report a case of tuberculous spondylodiscitis in a 37-year-old woman with negative
51 procalcitonin.

52

53

54 **CASE PRESENTATION:**

55 **Patient information:** A 37-year-old woman with no medical history presented with progressive
56 upper back pain for six months. She reported progressive dorsalgia that became resistant to
57 standard pain medications, followed by rapid fatigue of the lower limbs and intermittent
58 claudication complicated by bladder and bowel dysfunction on admission. She denied any
59 trauma or other triggering events.

60 **Clinical findings:** On physical examination, the patient was fully conscious, had no fever, and
61 was both respiratory and hemodynamically stable. There was D4 level hypoesthesia and normal
62 muscle tone with a Medical Research Council (MRC) muscle strength scale of 3/5 in both lower
63 limbs. The reflexes of the right lower extremity were reduced with right-sided Babinsky. Mild
64 abdominal distension with pollakiuria was noted.

65 **Diagnostic assessment:** Magnetic resonance imaging showed spinal cord compression at the
66 D3-D4 level with epidural collection. The initial laboratory evaluation revealed WB 10.4G/l;
67 CRP 138.7 mg l⁻¹; PCT 0.04 ng/ml. ECG and Chest X-Ray were unremarkable. Analysis of
68 sputum using molecular methods (GeneXpert MTB/RIF®) and classical methods (direct
69 examination and culture on Löwenstein-Jensen solid media) yielded negative results.

70 **Therapeutic intervention:** The patient underwent decompressive surgery, which included a
71 laminectomy of D4 and osteosynthesis of D2-D5. A molecular test using real-time PCR
72 (GeneXpert MTB/RIF®) performed on the surgical specimen revealed the presence of
73 Mycobacterium tuberculosis complex (MTBC) without detecting rifampicin resistance.
74 Anatomopathological examination of the surgical specimen confirmed the diagnosis by
75 revealing a caseating necrotizing epithelioid and giant cell granuloma.

76 **Follow-up and outcome:** After detection of MTBC, treatment included antitubercular therapy
77 according to the national protocol. The treatment was successful and led to resolution of the
78 inflammatory syndrome. CRP normalized 8 weeks after the initiation of treatment.

79 **DISCUSSION:**

80 Tuberculosis ¹² is a significant public health problem worldwide. According to the World Health
81 Organization (WHO), more than 10 million people develop an active tuberculosis infection (2).
82 This disease ¹⁵ is the third most common cause of death from infectious diseases worldwide. The
83 frequency of tuberculous spondylodiscitis among all infectious spondylodiscitis varies
84 considerably depending on the endemic status. It ranges from 20–40% in France, Spain or
85 Sweden (3–5) to over 70% in North African countries (6). The dorsolumbar spine is more
86 commonly affected (more than 95% of cases), as was the case in our patient, who had
87 compression between D3 and D4 (7). The diagnosis of tuberculous spondylodiscitis ¹³ is based on
88 clinical, radiological and biological arguments. The gold standard for diagnosing tuberculosis
89 is either isolating the bacterium through conventional culture or detecting its specific nucleic
90 acid sequence using molecular testing. In our case, the diagnosis was confirmed using the real-
91 time PCR technique, which, with its specificity and sensitivity as well as its rapid results, has
92 revolutionized diagnostics and allows timely treatment of this chronic disease. Therefore, it
93 serves as a valuable tool in an endemic country like ours (8). As for the laboratory results, our
94 patient had a negative PCT and a positive CRP. Several ¹⁴ studies have concluded that PCT is a
95 potentially useful biomarker for identifying traumatic spinal cord lesions (9,10). However,
96 many other studies suggest that PCT is not helpful in diagnosing tuberculosis spondylodiscitis
97 (1). This last statement is confirmed in our case. ¹⁰ On the other hand, Jeong et al. showed that
98 high PCT serum levels were observed, particularly in associated systemic infections (9). Yoon
99 et al. observed that the serum level of PCT is lower in tuberculous spondylodiscitis compared
100 to pyogenic spondylodiscitis (11).
101 According to the recommendations ² of the French Society of Infectious Diseases, the
102 investigation of an inflammatory syndrome using CRP measurement should be the first

103 biological test carried out. On the contrary, PCT is not a useful marker for the diagnosis of
104 spondylodiscitis (12).

105 PCT is a precursor of calcitonin, a hypocalcemic peptide hormone secreted by C cells of the
106 thyroid. This protein is encoded by the CALC-I gene. Under normal conditions, PCT undergoes
107 intracellular maturation and is then secreted as calcitonin. This means that PCT is not released
108 as a prohormone and its measurement is negative. However, in some pathological situations
109 (sepsis, severe bacterial infections), the expression of the CALC-I gene is enhanced in other
110 tissues, leading to PCT synthesis in other organs such as liver, lungs, kidneys, intestines,
111 adipose tissue, etc not by leukocytes [13,14]. This atypical expression in unusual cell types is
112 not followed by the maturation step that normally converts the prohormone into active
113 calcitonin. Thus, it is the PCT that is excreted into the bloodstream. Initial treatment of
114 tuberculous spondylodiscitis includes antitubercular drugs, starting with two-month
115 quadritherapy based on rifampicin-isoniazid-pyrazinamide-ethambutol, followed by seven-
116 month bitherapy with rifampicin-isoniazid. The patient was treated with the same regimen and
117 showed significant clinical and biological improvement.

118 **Conclusion**

119 PCT measurement remains challenging, time-consuming, and costly. Therefore, in this clinical
120 scenario, there is a need for optimization and the need for simultaneous measurement of CRP.
121 Therefore, it is advisable to prioritize CRP and control PCT measurement based on the criteria
122 described previously.

123 **Declarations**

124 ***Ethics approval and consent to participate:*** Informed consent was obtained from the patient
125 before the submission of this article. Also, this article respects both the Consensus-based
126 Clinical Case Reporting Guideline and the Recommendations for the Conducting, Reporting,
127 Editing, and Publication of Scholarly Work in Medical Journals.

128 **Consent for publication:** Informed consent was obtained from the patient to publish his case

129 **Availability of data and material:** No data were reused or generated in this study.

130 **Competing interests:** The authors declare that they have no competing interests

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132 **Authors' contributions:** **YBL:** Conceptualization, writing draft, reviewing and editing,
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135 Supervision, Validation, & review.

136 **Acknowledgments:** N/A

137

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