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(RESEARCH ARTICLE)



Interest of procalcitonin in the management of bacterial infections

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Abstract

Introduction. Procalcitonin (PCT), a protein of 116 amino acids, is the precursor of calcitonin. Calcitonin is a peptide hormone of 32 amino acids. Most calcitonin is produced in humans by the para-follicular cells (also known as C-cells) of the thyroid gland, and by the ultimo-branchial bodies in many other animal species. Procalcitonin is a biomarker that can be used to rationalize the duration of antibiotic therapy in certain infections, particularly bacterial ones.

Objective. The aim of this study was to evaluate the value of procalcitonin as a prognostic marker and as a tool for guiding the duration of antibiotic therapy in the management of bacterial infections in inpatients and outpatients at the Cliniques Universitaires de Lubumbashi.

Methodology. We conducted a longitudinal descriptive study. A total of 59 patients ranging in age from 0 to over 50 years, regardless of sex, were included in our study. The study was conducted over a six-month period from May to October 2023. We measured procalcitonin in all patients included in this study.

Results. Elevated procalcitonin levels were observed with peaks above 20 ng/ml prior to antibiotic therapy. *Escherichia coli* was the germ most isolated from urine cultures, and the majority of patients included in this study had received cephalosporins and fluoroquinolones. Over 80% of patients had PCT, CRP and ultrasensitive CRP values between 0 - 1 (in ng/ml for PCT and mg/dl for CRP and ultrasensitive CRP) after antibiotic therapy.

Conclusion. The procalcitonin assay, beyond its ability to differentiate bacterial infections from viral or inflammatory pathologies, can therefore be used to identify patients who do or do not require antibiotic treatment

Keywords: Procalcitonin; Management; Bacterial Infections; Antibiotics; CRP; Lubumbashi

1. Introduction

Procalcitonin (PCT), a protein of 116 amino acids, is the precursor of calcitonin. Calcitonin is a peptide hormone of 32 amino acids. Most calcitonin is produced in humans by the para-follicular cells (also known as C cells) of the thyroid gland, and by the ultimo-branchial bodies in many other animal species. Calcitonin is involved in phosphocalcic metabolism, counteracting the effects of parathyroid hormone (PTH), at least in terms of calcemia. It is a hypocalcemic and hypophosphoremic hormone, specifically reducing blood calcium levels [1].

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Procalcitonin is a biomarker that can be used to rationalize the duration of antibiotic therapy in certain infections, particularly bacterial infections [2]. Over the past fifteen years, procalcitonin has attracted considerable clinical interest as an early and sensitive marker of severe bacterial infections. Unlike C-reactive protein (CRP), procalcitonin is specific to infection, as it is either not increased or only modestly increased in inflammatory diseases. It can therefore contribute to the differential diagnosis between a septic state and an inflammatory or viral disease [3].

Indeed, measuring its concentration can be used as a biological marker of the severity of an infection. Procalcitonin concentration decreases very rapidly after eradication of the infectious site, while its production is maintained if the infection persists. Procalcitonin measurement is useful for assessing the evolution of an infectious state or the efficacy of antimicrobial treatment [4].

In this context, procalcitonin is currently a specific biochemical marker of infectious inflammatory reaction, particularly bacterial. This marker is also a prognostic element, as its increase often correlates with the severity of the infection. In addition, PCT better reflects the response to antibiotic treatment than CRP. It is an effective marker for guiding antibiotic therapy, limiting the emergence of bacterial resistance and reducing treatment costs. This compound occurs specifically in cases of microbial infection, and is of particular interest as it is sometimes difficult for clinicians to diagnose the infectious origin of a pathology[5]. PCT measurement is indicated when bacterial, parasitic or fungal infections are suspected. However, PCT concentration is not increased in viral infections or non-infectious inflammatory pathologies. The amount of PCT produced in the blood is often correlated with the amount of infectious agents [6]. This is why the procalcitonin assay is more useful than the C-reactive protein assay, and why it is rapidly elevated in bacterial infections. CRP is a good marker of the acute phase of inflammation, but its use in distinguishing bacterial from viral infection is less obvious. Worldwide, several studies have shown that the use of procalcitonin as a decision-making factor has reduced the use of antibiotics, particularly in the treatment of community-acquired pneumonia, by 40-55%, safely and without impact on the resolution of the infection [7].

A study conducted in France by the Cochrane Group analyzed 14 studies involving 4,551 patients between 2012 and 2014. The results of this meta-analysis show that the use of procalcitonin assays to guide the initiation and duration of antibiotic therapy in the treatment of acute respiratory infections is not associated with increased mortality or treatment failure, but significantly reduces antibiotic consumption. However, this meta-analysis highlights the need for more mortality data to ensure the safety of the tool for ICU patients [8].

Several other studies have investigated the use of procalcitonin assays in the treatment of sepsis in ICU patients. The PRORATA study showed a significantly higher number of antibiotic-free days (23% reduction in antibiotic exposure) for patients whose procalcitonin levels were monitored, compared with the control group, with no effect on mortality (death at 28 and 60 days) [9].

In neonates, procalcitonin is superior to C-reactive protein and interleukin-6 as a marker of neonatal bacterial infection, but levels must be carefully assessed against the physiological increase in the first two days of life [15,16]. Its sensitivity is generally equal to or sometimes greater than that of C-reactive protein, and much greater than that of cytokines or interferon alpha, but its specificity is always greater than that of C-reactive protein, interleukin-6 and other cytokines [17,18]. During antibiotic treatment, PCT assays can be used to check the efficacy of the treatment and, if necessary, adapt its duration [19,20].

Despite the routine use of procalcitonin in some of the country's hospitals as a marker of inflammation in certain infections, there are no data on the relevance of this practice and the possible contribution of procalcitonin to the diagnosis of bacterial infections in our study setting.

Following the presentation of this state of affairs, we were able to formulate the following research question.

1.1. Research question

Is procalcitonin of particular interest in the management of bacterial infections, and is there a correlation between CRP and procalcitonin in the diagnosis of bacterial infections?

1.2. Objectives

1.2.1. General objective

The aim of our study is to evaluate the value of procalcitonin as a prognostic marker and as a tool for guiding the duration of antibiotic therapy in the management of bacterial infections in inpatients and outpatients at the University Clinics of Lubumbashi.

1.2.2. Specific objectives

- To achieve our general objective, we set ourselves the following specific objectives:
- Determination of patients' procalcitonin levels before and after antibiotic therapy;
- Determine patients' CRP concentration before and after antibiotic therapy;
- Isolate and identify germs involved in bacterial infections;
- Determine duration of antibiotic therapy.

2. Materials and methods

2.1. Study setting

Our study was carried out in the laboratory of the Cliniques Universitaires de Lubumbashi.

2.1.1. Type of study

This was a longitudinal descriptive study of 59 patients.

2.1.2. Study period

Our study took place over a six-month period from May 1 to October 31, 2023.

2.2. Study population

Patients attending the University Clinics of Lubumbashi.

2.3. Sampling

2.3.1. Type of sampling

This was a non-probability sample of convenience, with patients included according to the inclusion criteria. The sample size was 59 patients.

2.4. Inclusion criteria

Patients attending the Cliniques Universitaires de Lubumbashi, with bacterial infections confirmed by microbial culture on the basis of biochemical characteristics of the germs, and who had received procalcitonin, CRP and antibiotic therapy, were included in this study.

2.4.1. Non-inclusion criteria

All patients who did not meet the inclusion criteria and those whose procalcitonin was not measured after antibiotic treatment were excluded from this study.

2.5. Materials and data collection techniques.

2.5.1. Equipment

Reagents.

Procalcitonin kit, CRP kit, Gram staining kit, culture media.

2.5.2. Equipment.

Finecare and RaFIA semi-automated Procalcitonin and CRP assays, microscopes, data-encoding computer.

2.5.3. Techniques

Procalcitonin was measured by immunofluorescence in all patients with bacterial infections confirmed by culture before and after antibiotic therapy. Germ cultures to confirm bacterial infection were performed in the bacteriology unit of the Cliniques Universitaires de Lubumbashi laboratory. Samples were inoculated on CLED, Mac Conkey, cooked blood agar and SS culture media.

2.6. Study variables

- Age
- Sex
- Patient category
- Residence
- Procalcitonin levels before and after antibiotic therapy
- CRP levels
- Types of infection
- Duration of antibiotic therapy
- Germs isolated.

2.7. Data management and analysis

Data analysis was performed using IBM SPSS version 20.0 software.

2.8. Ethical considerations

Authorization from the ethics committee of the University of Lubumbashi was granted. Approval: UNILU/CEM/034/2014.

3. Results

The results of our study are shown in the tables and figures below:

Table 1 Breakdown of patient frequency by month.

Month /year	Frequency	Percentage	Cumulative percentage
May 2023	7	11.9%	11.9%
June 2023	3	5.1%	17.0%
July 2023	15	25.4%	42.4%
August 2023	10	16.9%	59.4%
September 2023	6	10.2%	69.6%
October 2023	18	30.5%	100.0%
Total	59	100,0%	

The table show that October recorded the hightest number of patients, at 30.5%, followed by July at 25.4%.

Table 2 Distribution of patients by municipality of residence.

Municipality of residence	Frequency	Percentage	Cumulative percentage
LUBUMBASHI	29	49.2%	49.2%
KENYA	8	13.2%	62.4%
KAMALONDO	6	10.2%	72.6%
КАМРЕМВА	5	8.5%	81.1%

ANNEXE	5	8.5%	89.6%
RUASHI	4	6.8%	96.4%
KATUBA	2	3.4%	100.0%
Total	59	100,0%	

The results of this table show that 49,2% of patients came from the commune of Lubumbashi.

Table 3 Patient distribution by category.

Patient category	Frequency	Percentage	Cumulative percentage
SUBSCRIBERS	31	52.5%	52.5%
UNILU	12	20.3%	72.8%
PRIVATE	9	15.3%	88.1%
PRODEO	7	11.9%	100.0%
Total	59	100,0%	

The results of table show that 52,5% were subscribers.

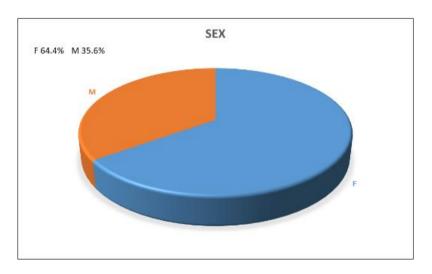


Figure 1 Patient distribution by gender

This figure shows that 64,4% of patients were female.

Table 4 Patient Distribution by aged group

Aged tench	Frequency	Percentage	Cumulative percentage
0 – 10 years	29	49.1%	49.1%
11 - 20 years	6	10.2%	59.3%
21 - 30 years	9	15.2%	74.5%
31 - 40 years	4	6.8%	81.3%
41 – 50 years	7	11.9%	93.2%
> 50 years	4	6.8%	100.0%
Total	59	100,0%	

The results in this table show that the 0 -10 group was the most represented.

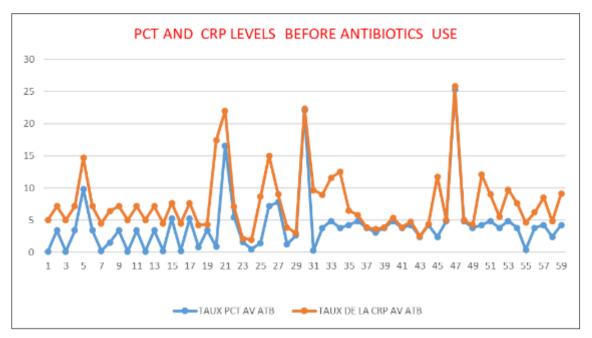


Figure 2 PCT and CRP levels before antibiotics in ng/ml and mg/dl.

The figure shows that procalcitonin peaked at 25 ng/ml and CRP at 20 mg/dl.

Table 5 Distribution of patients accordind to germs isolated

Isolated germs	Frequency	Percentage	Cumulative percentage
Escherichia coli	13	20.03%	20.03%
Klebsiella spp	12	18.64%	38.67%
PFPA	10	16.95%	55.62%
Citrobacter diversus	8	13.56%	69.18%
Morganella morganii	6	10.17%	79.35%
Staphylocoque spp	5	8.47%	87.82%
Salmonella typhi	2	3.39%	91.21%
Sérratia spp	1	1.69%	92.90%
Klebsiella pneumoniae	1	1.69%	94.59%
Proteus spp	1	1.69%	96.28%
Enterobacter spp	1	1.69%	100.0%
Total	59	100,0%	

This table shows that that *Escherichia coli* was the germ most isolated germ, at 20.03%.

Table 6 Distribution of patients according to the type of primary samples deposited at the Laboratory.

Primary sample	Frequency	Percentage	Cumulative percentage
Urine	29	49.15%	49.15%
Blood	9	15.25%	64.40%

Stool	6	10.17%	74.57%
Vaginal smear	5	8.47%	83.04%
Sputum	5	8.47%	91.51%
Pus	5	8.47%	100.0%
Total	59	100,0%	

The table shows that over 49% of germs were isolated from urine.

Table 7 Distribution of patients according to antibiotics received.

Antibiotics	Frequency	Percentage	Cumulative percentage
Ceftriaxone	12	20.34%	20.34%
Ofloxacin	10	16.95%	37.29%
Cefixime	8	13.56%	50.85%
Nitrofurantoin	7	11.86%	62.71%
Imipenem	6	10.17%	72.88%
Kanamycin	4	6.78%	79.66%
Azythromycin	4	6.78%	86.44%
Tétracycline	3	5.08%	91.52%
Ampicillin	3	5.08%	96.60%
Amoxicillin	2	3.39%	100.0%
Total	59	100,0%	

The results of this table show that 20.43% and 16.95% of patients had received cephalosporins and ofloxacin respectively.

 Table 8 PCT, CRP and ultrasensitive CRP levels after antibiotic therapy

Interval	PCT rate in ng/ml		CRP rate in mg/dl		Ultra sensitive CRP in mg/dl	
	Frequency	%	Frequency	%	Frequency	%
[0 - 1]	46	77.97%	50	84.74%	38	64.41%
]1 - 2]	1	1.69%	2	3.39%	18	30.51%
]2 - 3]	3	5.08%	5	8.47%	0	0.0%
]3 - 4]	4	6.78%	0	0.0%	0	0.0%
]4 - 5]	4	6.78%	0	0.0%	0	0.0%
> 5	0	0.0%	2	3.38%	3	5.08%
Total	59	100%	59	100%	59	100%

The results of table show that 77.97%, 84.74% and 64.41% of patients had PCT, CRP and ultrasensitive CRP values values between 0 - 1 mg/dl.

Table 9 Duration antibiotic therapy

Duration of antibiotic therapy	Frequency	Percentage	Cumulative percentage
2 days	7	11.9%	11.9%
3 days	13	22.0%	33.9%
4 days	3	5.1%	39.0%
5 days	25	42.4%	81.4%
≥ 6 days	11	18.6%	100.0%
Total	59	100,0%	

The results of this table show that 42.4% of patients had received antibiotic treatment for five days.

Table 10 Correlation between PCT and CRP.

PEARSON CORRELATION		RATE PCT BEFORE ATB	RATE CRP BEFORE ATB
RATE CRP BEFORE ATB	Pearson Correlation	1	-0.224
	Meaning		0.089
	N	59	59
RATE CRP BEFORE ATB	Pearson Correlation	-0.224	1
	Meaning	0.089	
	N	59	59

In fact, the Pearson correlation coefficient r varies between +1 and -1. If the coefficient correlation coefficient r is greater than 0.6, there's a strong correlation, between 0 and 0.3, there's a low correlation, and between 0.3 and 0.6, there's a medium correlation.

In our series, the correlation coefficient between PCT and CRP is equal to - 0.224 and the p value is equal to 0.089 > 0.05. The increase in PCT does not affect the increase in CRP. (Strength of Correlation low, difference not significant).

4. Discussion

Our study is based on 59 patients with culture-confirmed bacterial infections, at the Laboratory of the University Clinics of the Faculty of Medicine of the University of Lubumbashi.

Figure 2 shows elevated procalcitonin levels, with peaks above 25 ng/ml before antibiotic therapy. Our results are similar to those found by Hoffmann G and colleagues, who reported PCT results (measured by immunofluorescence) in the follow-up of patients with severe infections. To categorize infections according to the severity of systemic involvement, these authors showed that high PCT (threshold value between 0.1 and 0.5 ng/ml) distinguishes patients with sepsis, severe sepsis or septic shock at high risk of mortality from those with localized infections, with a sensitivity of 56% to 91%, a specificity of 87% to 100%, a positive predictive value of 69 to 100% and a negative predictive value of 80 to 95%. They observed that, unlike CRP, PCT is primarily a marker of the severity of bacterial infection[10].

Another study showed that PCT continued to rise in patients with severe sepsis unresponsive to treatment, with a rapid fall in PCT observed as soon as effective treatment was instituted (60% reduction in median PCT value, from 9 to 3 ng/ml, after three days). Ferrière F. and colleagues have also shown that, in patients with bacterial infections in which PCT had crossed the threshold of 0.5 ng/ml, a fall in PCT levels was associated in 85% of cases with a fall in temperature, clinical improvement and/or eradication of the pathogen, irrespective of the site of infection [16].

The majority of patients included in our study had received cephalosporins and fluoroquinolones as treatment, and *Escherichia coli* was the most isolated germ, justified by the fact that most cultures were taken from patients' urine. And *Escherichia coli* is a germ responsible for community-acquired urinary tract infections. A study by Gendrel D. showed

that ongoing antibiotic therapy at the time of fever could have prevented documentation of bacteremia. Overlapping PCT values in patients with documented infections explain the limited diagnostic performance of PCT in distinguishing them from other infections of viral, fungal or parasitic origin (13). PCT values < 0.5 ng/ml have been described in coagulase-negative staphylococcal bacteremia (median 0.2 to 0.4 ng/ml). On the other hand, PCT levels > 0.5 ng/ml observed in some patients demonstrate the presence of a bacterial infection that could not be documented either by clinical examination or by cultures. This illustrates the significant limitations in assessing the diagnostic performance of PCT. Several factors may have contributed to the variability of the results of these studies, making their comparison tricky. In particular, immediate administration of empirical antibiotic therapy at the time of onset of febrile neutropenia may explain the poor response of PCT in certain cases of documented infection [20].

Indeed, with the current upsurge in bacterial resistance becoming a real public health problem, PCT appears to be a marker that can help us decide whether or not to put the patient on antibiotics while awaiting the results of isolations of the bacteria responsible for bacterial infections. These antibiogram results last an average of 48 hours. However, samples must be taken before any antibiotic treatment is started. Antibiotic therapy can be adapted according to the results of microbial cultures during treatment [14].

In our study, over 80% of patients had PCT, CRP and ultrasensitive CRP values between 0 - 1 (in ng/ml for PCT and mg/dl for CRP and ultrasensitive CRP) after antibiotic therapy. CRP is one of the so-called acute-phase proteins, whose level in serum or plasma increases during a non-specific systemic response to infectious and non-infectious inflammatory processes. CRP is synthesized in the liver and is normally present in trace amounts in serum or plasma. In various pathological conditions involving tissue damage, infection or acute inflammation, CRP values may be higher than normal, reaching between 2 and 50 mg/dl [15,16].

As elevated CRP levels are always associated with pathological changes, the CRP method provides useful information on the diagnosis, treatment and monitoring of inflammatory processes and associated diseases. In contrast to PCT, which is specific to bacterial infection, increased CRP values are non-specific and should not be interpreted without the patient's full clinical history. Studies have shown that high-sensitivity CRP measurement (Ultrasensitive CRP) is a powerful independent predictor of the risk of cardiovascular and peripheral vascular disease. Ultrasensitive CRP measurements have been shown to enhance the predictive value of other markers used to assess the risk of cardiovascular and peripheral vascular disease[17]. Elevated CRP values measured by high-sensitivity CRP methods may indicate the prognosis of patients suffering from acute coronary syndromes, and may be useful in treating these patients.

Over 40% of patients had received antibiotic treatment for five days. Indeed, the next important step in the management of a patient with a bacterial infection is between 24-48 hours after the start of empirical antibiotic therapy. This is the time for clinical reassessment and receipt of culture results. At this stage, it is possible to classify the etiology of the fever and adapt or change the antibiotic after obtaining an appropriate antibiogram. However, neither the type of infection nor its severity really influences the duration of antibiotic therapy: in fact, it is recommended to continue until the procalcitonin level has fallen below 1 ng/ml [18].

Adaptations to antimicrobial therapy are often necessary if the febrile state persists beyond 48 hours. In the absence of rapid, high-performance diagnostics, these changes (modification of current antibiotic therapy, addition of antibacterial and/or antifungal agents) are made empirically in the majority of cases. However, persistent fever is a poor parameter for distinguishing an uncontrolled infection from one that is progressing favorably (median defervescence between 2 and 7 days). PCT is a marker that could help identify clinical situations requiring such adaptations, and would therefore be very useful [19].

In terms of disease prognosis, the course of PCT after day 4 of the onset of systemic inflammation was able to distinguish survivors from non-survivors. Up to day 4, PCT concentrations were not statistically different between the groups. Similarly, the initial height of PCT concentrations did not correlate with further disease progression. The results of this analysis should not be over-interpreted. The number of patients is too small and too heterogeneous for a general conclusion concerning the absolute height of PCT concentrations and the estimation of disease prognosis by PCT [20].

Additional diagnostic tools would be useful to identify patients in whom early discontinuation of antibiotic therapy could be considered. These markers will be the focus of future studies.

5. Conclusion

In addition to its ability to differentiate between bacterial infections and viral or inflammatory pathologies, procalcitonin measurement can be used to identify patients who may or may not require antibiotic treatment. Our study and other studies carried out in populations of patients suffering from severe bacterial infections, particularly respiratory infections, show that monitoring serum PCT levels significantly reduces the duration of antibiotic treatment.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

References

- [1] Matera G, Quirino A, Giancotti A, Pulicari M, Rametti L, Rodríguez M, et al. Procalcitonin neutralizes bacterial LPS and reduces LPS-induced cytokine release in human peripheral blood mononuclear cells. BMC Microbiol. 2012;12(1):68.
- [2] Wiedermann FJ, Kaneider N, Egger P, Tiefenthaler W, Wiedermann CJ, Lindner KH, et al. Migration of human monocytes in response to procalcitonin. Crit Care Med. 2002 May;30(5):1112-7.
- [3] Monneret G, Arpin M, Venet F, Maghni K, Debard A-L, Pachot A, et al. Calcitonin gene related peptide and N-procalcitonin modulate CD11b upregulation in lipopolysaccharide activated monocytes and neutrophils. Intensive Care Med. 2003 Jun;29(6):923-8.
- [4] Clec'h C, Ferriere F, Karoubi P, Fosse JP, Cupa M, Hoang P, et al. Diagnostic and prognostic value of procalcitonin in patients with septic shock. Crit Care Med. 2004 May;32(5):1166-9.
- [5] Boussekey N, Leroy O, Georges H, Devos P, d'Escrivan T, Guery B. Diagnostic and Prognostic Values of Admission Procalcitonin Levels in Community-Acquired Pneumonia in an Intensive Care Unit. Infection. 2005 Aug;33(4):257-63.
- [6] Nylen ES, Whang KT, Snider RH, Steinwald PM, White JC, Becker KL. Mortality is increased by procalcitonin and decreased by an antiserum reactive to procalcitonin in experimental sepsis. Crit Care Med. 1998 Jun;26(6):1001-6
- [7] Pouly O., Intérêt de la procalcitonine dans la prise en charge des infections sévères des tissus mous en réanimation. Présentée et soutenue publiquement le 15 IUIN 2017 à 18h. Lille. 2017.
- [8] Becker KL, Nylén ES, Snider RH, Müller B, White JC. Procalcitonin immunoneutralization as a therapy for sepsis. J Endotoxin Res. 2003;9 (6):367-74.
- [9] Tavares E, Miñano FJ. Aminoprocalcitonin peptide immunoneutralization of procalcitonin protects rats from lethal endotoxemia: neuroendocrine and systemic studies. Clin Sci. 2010 Dec 1;119(12):519-34.
- [10] Hoffmann G, Czechowski M, Schloesser M, Schobersberger W. Procalcitonin amplifies inducible nitric oxide synthase gene expression and nitric oxide production in vascular smooth muscle cells. Crit Care Med. 2002 Sep;30(9):2091-5.
- [11] Luong Ba K, Harbarth S, Carballo S. Procalcitonin: to dose or not to dose? [Procalcitonin: should it be measured systemically?] Rev Med Suisse. 2013 Oct16;9(402):1881-2, 1884-5. In French. PMID: 24298711.
- [12] Herbomez M. Calcitonin assays: indications and interpretation. Presse Med. 2011 Dec;40(12 Pt1):1141-6.
- [13] Gendrel D, Moulin F. Community-acquired pneumonia in children. Rev Prat. 2007 Nov 15;57(17):1883-94. French.PMID: 18095624.
- [14] Hausfater P. Le dosage de la procalcitonine en pratique clinique chezl'adulte. Rev Med Interne. 2007 Mai;28(5):296-305.

- [15] Bohuon C, Gendrel D. Procalcitonin: a new indicator of bacterial infection. Intérêt et perspectives [Procalcitonine: a new marker of bacterialinfection. Importance and prospects]. Arch Pediatr. 1999 Feb;6(2):141-4.
- [16] Ferrière F. Interest of procalcitonin, a new marker of bacterial infection. Ann BiolClin (Paris). 2000 Janeb;58(1):49-59.
- [17] Mary R, Veinberg F, Couderc R. Acute meningitis, inflammatory proteins and procalcitonin. Ann Biol Clin (Paris). 2003 Mar-Apr;61(2):127-37.
- [18] Green Y, Petignat PA, Perrier A. Proper use of procalcitonin. Rev Med Suisse. 2007 Oct 17;3(129):2330-2, 2334.
- [19] Le Goff C, Ladang A, Gothot A, Cavalier E. Les marqueurs biologiques de l'inflammation: faisons le point. Rev Med Liege. 2022 May;77(5-6):258-264.
- [20] Gendrel D. Infection urinaire et marqueurs biologiques: protéine C réactive,interleukines et procalcitonine. Arch Pediatr. 1998;5 Suppl 3:269S-273S.