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Kinetic Evolution of C-Reactive Protein as a Function of Drug Molecule Intake in Patients with Tuberculosis in the Sédhiou Health District (Sénégal)

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Abstract

C-Reactive protein (CRP) is a protein of the inflammatory reaction whose name is linked to its property of being precipitable by a pneumococcal polysaccharide of type C in the presence of ionized calcium. In this prospective study carried out in the health district of Sédhiou, we try to evaluate the impact of taking the anti-tuberculosis treatment molecule in patients with tuberculosis combined with the evolutionary monitoring over time of CRP. The values of the indicator of inflammation or infection were carried out by an A15 biochemical automaton on 30 sick subjects and 20 healthy carriers as a comparative control. The results obtained in patient input data on average give in CRP a value of 93.56 ± 5.71 mg/l, which shows a real infection in the subjects studied then the weight as an illustrative element of the body mass deficit is translated on average with a value of 49.55 ± 1.81 kg and finally the temperature which oscillates with a value of 39.95˚C ± 0.58˚C, which actually shows the feverish state of the patients involved. Moreover, the taking of medication carried out in accordance with the established treatment gives quite encouraging results through the drastic drop in the entry CRP with a value turning to 6.30 ± 1.56 mg/l, leading to a parallel increase in weight with an average value of 60.40 ± 2.76 kg and suddenly a significant drop in temperature with an average of 36.19˚C ± 0.43˚C. Analysis of variance according to the Kruskal-Wallis test (P = <0.001) shows a significant difference among the mean values of the studied data.

Keywords

CRP, Tuberculosis, Sédhiou Health District, Sénégal

1. Introduction

C-reactive protein (CRP) is an inflammatory response protein whose name is linked to its property of being precipitable by a pneumococcal type C polysaccharide in the presence of ionized calcium. It is a protein with a molecular weight of 118 kDa, synthesized under the dependence of interleukin 6 by hepatocytes mainly, but also by T lymphocytes and NK cells. The physiological roles of CRP are the activation of the classical complement pathway, the mobilization and activation of leukocytes and the stimulation of phagocytosis.

Its kinetics are rapid, with a half-life of 18 hours; it increases 6 to 7 hours after an attack to reach a maximum after 72 hours, and its level returns to normal after 1 week [1].

In the inflammatory profile, CRP measurement is often combined with slow-onset inflammatory proteins (orosomucoid or haptoglobin) to quantify and date the inflammatory response. It does not cross the placenta, hence the importance of its determination in neonatal pathology. It can be measured by radial immunodiffusion, nephelometry, or turbidimetry, on clear serum or heparinized plasma (fasting sample). CRP is relatively stable; samples can be stored refrigerated for 1 week and frozen for several months. Normal values are less than 6 mg/L in both men and women. There is no known cause of decreased CRP. However, in cases of severe hepatocellular insufficiency, the response to inflammation is diminished since the liver can no longer synthesize CRP [1].

The only cause of increased CRP is inflammation. Its basal level can be multiplied by 100, or even 1000 in the case of severe infection. A clear elevation of CRP is observed in bacterial infections (especially in neonatal and post-surgical infections), in rheumatic pathologies (such as rheumatoid arthritis, ankylosing spondylitis, vasculitis), in digestive pathologies (Crohn's disease), in malignant diseases (lymphomas, sarcomas, carcinomas), in ischemic necrosis (infarction regardless of the location), and in trauma (surgery, burns).

A slight elevation of CRP is observed in viral infections, in certain connective tissue diseases (systemic lupus erythematosus, scleroderma), in leukemias and in certain digestive pathologies (ulcerative colitis). Another early marker of inflammation, serum procalcitonin increases within the same timeframe as CRP, but differs from it by its elevation in systemic infections of purely bacterial origin. In the neonatal period, where infection represents the main cause of inflammation, CRP proves to be a more reliable marker than procalcitonin in the detection of early infections, because the latter has a physiological peak making it difficult to define its significance threshold [1].

C-reactive protein (CRP) is a glycoprotein secreted during the acute reaction phase, the level of which increases following the inflammatory process, particularly in bacterial infections (pneumococcus), histolytic diseases and in many other pathological conditions [2].

CRP is part of the pentraxin family, a set of very ancient proteins that are highly conserved between species, including CRP, SAP (serum amyloid component) and

APP (amyloid precursor protein), a precursor of the β-amyloid peptide [3].

Tuberculosis is a contagious, bacterial infectious disease that usually attacks the lungs. Tuberculosis can cause significant damage if not treated properly. Active tuberculosis in the lungs is called pulmonary tuberculosis, which is often contagious.

Extrapulmonary forms of tuberculosis (e.g., lymph node tuberculosis, bone tuberculosis, meningitis, etc.) are not contagious [4].

One-third of the world's population is exposed to Mycobacterium tuberculosis at some point. An infected person can infect their loved ones at anytime.

To guide the diagnosis and the therapeutic course to be adopted, it is important for the physician to have an early, rapid, reliable and inexpensive biological test. Some studies validate the use of CRP as a diagnostic biomarker for sepsis, due to its reproducibility, low cost and availability. Studies validate the use of CRP as a diagnostic biomarker for sepsis, due to its reproducibility, low cost and availability [5].

Its interest as a prognostic marker, CRP kinetics has been described as a predictive marker of survival in the first days of antibiotic treatment in patients hospitalized in intensive care for sepsis [6].

However, during an inflammatory reaction, it is a mode of response of the organism to an aggression, which can be physical, infectious, chemical, immunological, tumoral or traumatic. This mechanism is controlled locally by a complex network of cellular and humoral interactions, among the main participants of which are interleukin 6 (IL-6) produced by fibroblasts and endothelial cells, interleukin 1 (IL-I) and tumor necrosis factor (TNF), produced by macrophages and/or neutrophils, at the origin of a sophisticated control of the inflammatory response [7].

There are three distinct phases in the course of the inflammatory reaction:

1) An initiation phase, which depends on the nature of the triggering factor and involves primary factors:

- Activation of platelets, endothelial cells, fibrinolysis, and complement with the release of anaphylatoxins C3a, C5a, etc.
- The release of vasoactive amines such as histamine and serotonin, which promote vasodilation, increase capillary permeability, and induce the expression of adhesion molecules.

2) An amplification phase which mobilizes and activates secondary actors by: the expression of adhesion molecules, cytokine receptors, chemokines, an influx of cells at the inflammatory focus under the effect of chemotactic factors and an activation of these cells which produce pro-inflammatory factors (IL-1, IL-6, TNF-α, etc.), the rapid recruitment of neutrophil polymorphonuclear cells which will be able to ensure the phagocytosis of exogenous pathogenic agents or infected cells at the inflammatory site. The macrophages will release vasoactive substances, participate in phagocytosis and initiate the specific type of immune response.

3) A resolution and repair phase which allows reconstruction of the damaged cell so the amplification phase is limited in time by the implementation of control

systems such as anti-proteases, anti-inflammatory cytokines, anti-radicular systems, the secretion of growth factors, cytokines and neovascularization facilitated by chemokines will participate in the reconstruction of damaged tissues. More than 30 proteins undergo an increase in their circulating concentration during an inflammatory syndrome [8].

CRP would therefore appear to be a good indicator of bacteremia from a certain level, in the same way as certain clinical criteria, but its normality alone does not allow us to exclude an infection or bacteremia. The ideal marker of inflammation should have a rapid kinetics of evolution, an exclusive dependence on the inflammatory reaction, be independent of the clinical etiology of inflammation, have a precise, rapid, easy, standard-compliant and inexpensive dosage, and have a significant increase during a moderate reaction proportional to the degree of inflammation [2].

Infection is a microbial phenomenon characterized by an inflammatory response linked to the presence of microorganisms or the invasion of normally sterile host tissue by these organisms [9].

Pneumonia (tuberculosis) is based on the presence of a new radiological infiltrate and the sudden onset of at least one confirmed clinical element, suggestive of pneumonia, including: cough, dyspnea, pleuritic pain, sputum production, fever, and newly altered mental status [10].

The hypothesis of this work is that the severity of patients admitted to intensive care is related to whether the serum CRP level is less than 10 or between 10 and 100 and greater than 100 [11].

CRP testing in patients with tuberculosis allows for rapid diagnostic hypothesis development and immediate initiation of treatment. It allows for monitoring treatment effectiveness, diagnosing complications, and preventing further progression of the disease.

For practical and economic reasons, it is preferable to choose among these molecules in order to select the most reliable marker [12].

2. Method and Materials

Serum C-reactive protein (CRP) causes agglutination of latex particles coated with anti-human C-reactive protein antibodies. Agglutination of latex particles is proportional to the CRP concentration and can be quantified by turbidimetry.

2.1. Materials

- A15 Biochemical Automated Analyzer
- Cups
- 200 µL Micropipette
- 200 µL Tips or Tips
- Wash Bottle
- Pure H_2O
- NaCl for analysis

- Cup or Petrie dish (glass)
- Precision Balance 0.01 g (Ohaus 10-4 Pres)
- 1000 ml System Fluid Code BO 11524 Biosystems S.A. Costa Brava 30, Barcelona (Spain)
- 100 ml Washing Solution Code BO 13416 Biosystems S.A. Costa Brava 30, Barcelona (Spain)
- CRP Reagent Ref 13921 2 × 20 ml Biosystems Biosystems S.A. Costa Brava 30, Barcelona (Spain)
- Reagent A CRP (2 × 16 ml) 0.1 mol/L glycine buffer, 0.95 g/L sodium azide, pH 8.6.
- Reagent B CRP (2 × 4 ml) suspension of latex particles sensitized with anti-human CRP antibodies, 0.95 g/L sodium azide
- Calibrator S CRP Ref 31113 (1 × 1 ml) Biosystems S.A. Costa Brava 30, Barcelona (Spain)
- Rheumatic Control Level I code 31213 Biosystems S.A. Costa Brava 30, Barcelona (Spain)
- Rheumatic Control Level II code 31214 Biosystems S.A. Costa Brava 30, Barcelona (Spain)
- Bathroom scale
- Thermometer

2.2. Method

Treatment Dosage

Tuberculosis is certainly a curable infectious disease today, and patients receive free treatment, which lasts an average of six months. The protocol follows this path for 15 days, the patient is no longer contagious. In the intensive phase, which lasts two months, four molecules are prescribed, and the patient consumes the following:

1) Rifampicin 150 mg (R)
2) Isoniazid 75 mg (H)
3) Pyrazinamide 400 mg (Z)
4) Ethambutol Hydrochloride 275 mg (E)

This molecule intake constitutes the combined fixed dose, according to the patient following his assiduity and his motivation to heal according to the regular intake of the prescribed medication according to his weight. From the 2nd month of treatment an assessment is carried out in order to evaluate the reduction in the spread of the lesions affected by the organism this assessment consists of a spit bar test followed by the patient's weight gain. If the diagnosis situation is positive correlated with the negative spit test after 5 months of consumption of the fixed dose molecule another assessment is carried out in order to free the patient from taking medication by noting a healing of the damaged lesions. The data in the following Table 1 give rise to the treatment to be followed for the patient while respecting the respective weight ranges [13].

Table 1. Treatment in adult patients.

Processing phase	Weight kg			
	30 - 39	40 - 54	55 - 70	70
Intensive initial phase 2 months	2 cp	3 cp	4 cp	5 cp
Continuation phase 4 months	2 cp	3 cp	4 cp	5 cp

Anti-tuberculosis medication should be taken regularly every day at the same time, very early in the morning, often 1 hour before meals.

The data results were analyzed by Excel 10 and Sigma plot 11 software.

3. Results Obtained

Table 2 illustrates the values of the standard parameters recorded upon admission of the patient to the health district in question, the CRP, the weight and temperature of the interned patient.

Table 2. Standard input and output parameter values.

	CRP 1	CRP 2	Weight 1	Weight 2	T˚C 1	T˚C 2
P1	95	10	51	63	40	36
P2	90	8	49	65	39.7	35.8
P3	88	6	52	70	40.2	35.7
P4	98	9	48	60	39.8	36.2
P5	89	5	45	61	39.6	35.9
P6	86	7.7	53	65	40.1	36.2
P7	92	9.5	50	59	39	37
P8	85	6.8	47	58	38.9	36.1
P9	81	4.2	51	61	39.5	35.8
P10	82	3.8	48	59	40.9	36
P11	97	7	49	61	39.6	37
P12	100	6.8	50	60	40.2	36.8
P13	96	7	48	58	39.8	36.9
P14	94	6	51	60	39.7	35.8
P15	101	7	49	57	41.1	37
P16	98	6.6	50	62	39.6	36.7
P17	95	5.4	48	59	39.8	36.1
P18	103	6.9	49	60	39.7	37
P19	97	4.5	50	61	41.1	36.1

Continued

P20	102	7.2	49	57	39.6	35.8
P21	98	6.4	51	60	39.8	36.2
P22	99	6.8	49	58	41.5	35.9
P23	89	5.2	48	57	39.9	36.1
P24	97	4.8	47	60	40.5	36
P25	89	4.6	50	62	39.7	35.9
P26	93	4.1	49	58	40.1	36.1
P27	95	5	52	62	39.6	36
P28	90	5.2	51	61	40.2	35.9
P29	92	5.5	53	60	39.5	35.7
P30	96	7	49.5	58	39.8	36.2

The data in the table reported in the group of CRP intake at the patient's admission denotes a mean value of 93.56 ± 5.7 mg/l, manifesting an increase in the infection rate in the 30 patients with tuberculosis (P = 0.416). There is a significant difference between the values of the group (P = <0.001). In view of these results, the hypothesis does not rule out the presence of sepsis [8]. However, the average CRP value after treatment after fifteen days, correlated with the control CRP value, shows a satisfactory improvement compared to the sick subjects with a value of 6.30 ± 1.56 mg/l (P = 0.206) compared to the control value which is 5.65 ± 2.06 mg/l. Note that the decrease in weight clearly reveals the degree of inflammation or infection of the sick subject characterized by a weight loss whose average of the group the weight is 49.55 ± 1.81 kg (P = 0.474) compared to the control value of 68.30 ± 7.56 kg (P = 0.600), which reflects a very significant decrease. At the same time, a gradual return is observed through sudden weight gain with an average of 60.40 ± 2.762 kg compared to the control, which is very remarkable and positive. Regarding the temperature delivered on average in all the patients studied, it gives a value of 39.95˚C ± 0.584˚C compared to the control whose average is 36.495˚C ± 0.742˚C with P = 0.002 this shows in an illustrative manner the persistent presence of fever in people suffering from tuberculosis disease, but with the support of the care and the monitoring of the assessment of the treatment carried out in the patients we note a drastic drop in fever demonstrated by a significant decrease in temperature with an average of 36.19˚C ± 0.43˚C substantially equal to the control value. We note a fairly significant difference among all the subjects in the study group with (P = <0.001).

The control values in the following **Table 3** were carried out on a sample of twenty healthy individuals who had not contracted tuberculosis disease. Here is the data of the results of taking CRP and their respective weight as well as the temperature.

Table 3. The control values of the standard parameters.

Patient witness	CRP mg/L	Weight Kg	T˚C
P1	5	56	36.7
P2	7	62	35.9
P3	5	65	36.5
P4	4	68	36
P5	10	69	38.4
P6	8	58	37
P7	9	60	37.8
P8	5.5	67	36.1
P9	6.2	71	35.8
P10	3	72	36
P11	2.7	75	36.6
P12	3.3	80	36.2
P13	5.2	65	35.8
P14	3.8	60	36
P15	4.7	70	36.7
P16	5.1	85	36
P17	4.9	80	35.8
P18	5.2	69	36.5
P19	9.2	66	37.8
P20	6.2	68	36.3

As shown in **Figure 1** below, the control gives an arbitrary value of CRP with 5.65 ± 2.06 mg/l (P = 0.128) which illustrates an acceptable threshold compared to the accepted reference rate, *i.e.* the value of 5 mg/l (https://www.fiches-ide.fr/).

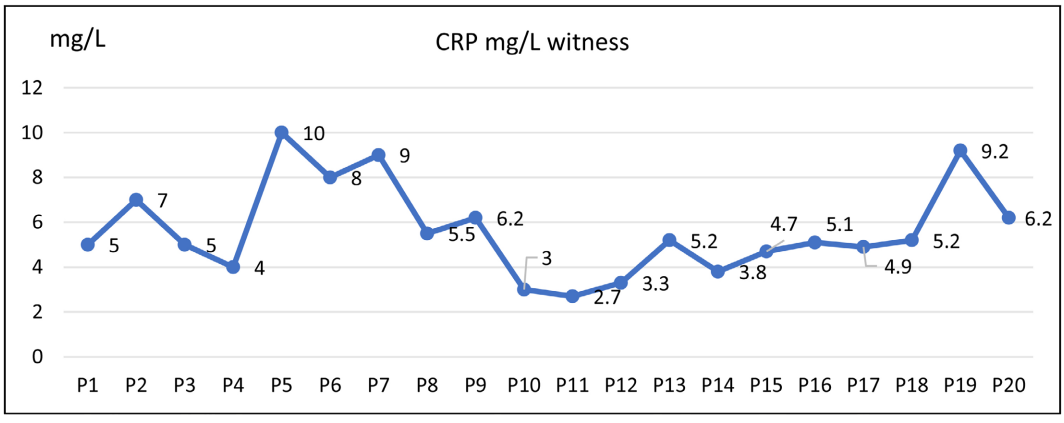


Figure 1. CRP value in control subjects.

While Figure 2 shows the weight variation in the control which gives a value of 68.30 ± 7.56 Kg with (P = 0.600).

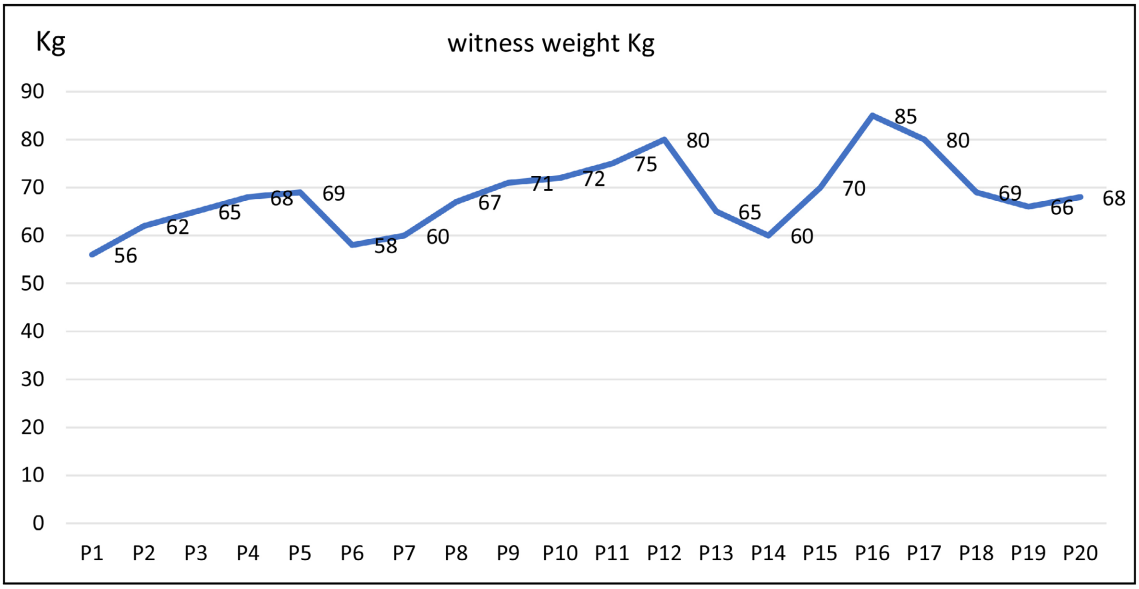


Figure 2. Variation in body mass content in the control (Kg).

Figure 3 below shows the temperature reading of the control data giving an average value of 36.495˚C ± 0.742˚C with P = 0.002.

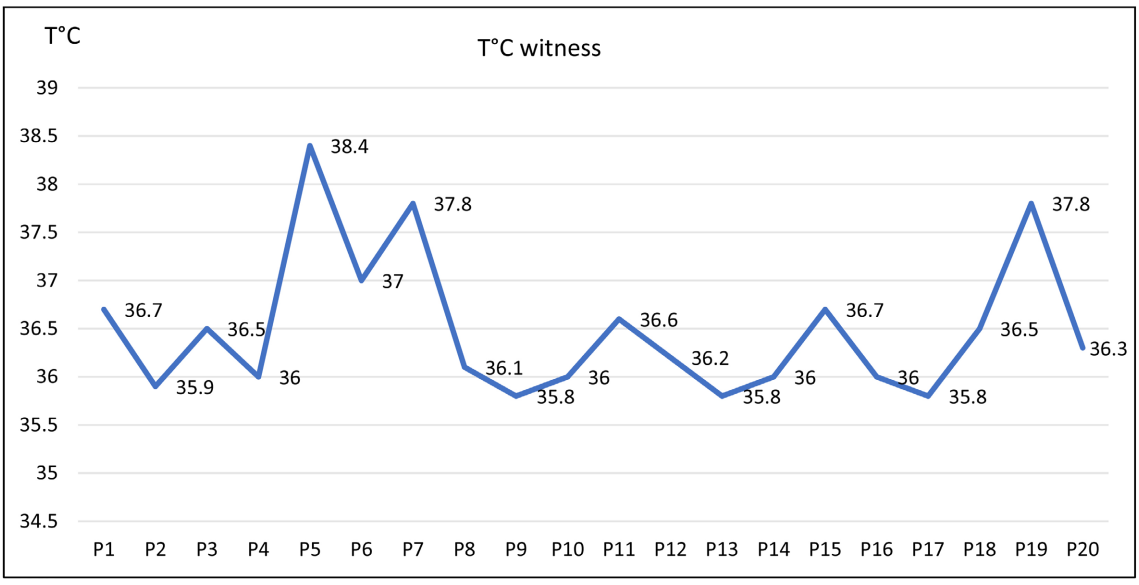
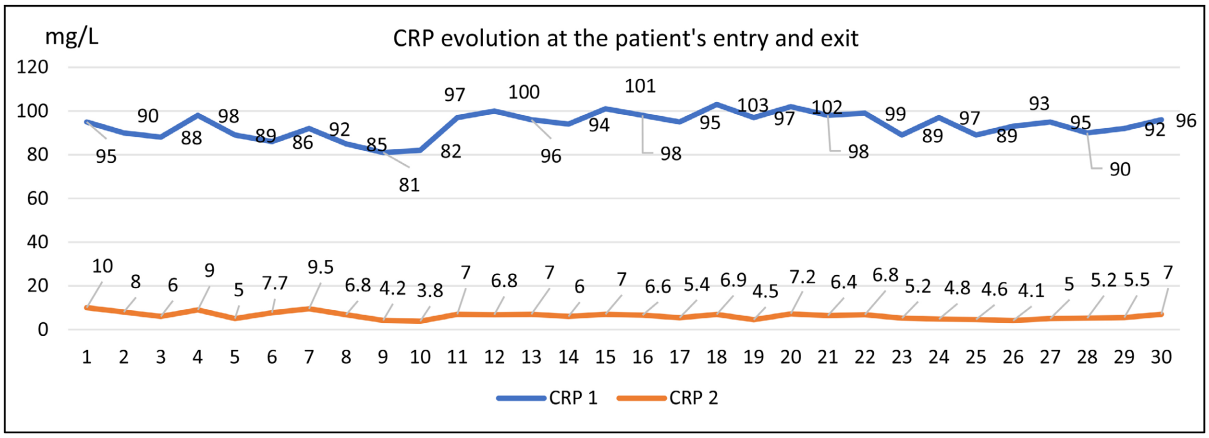


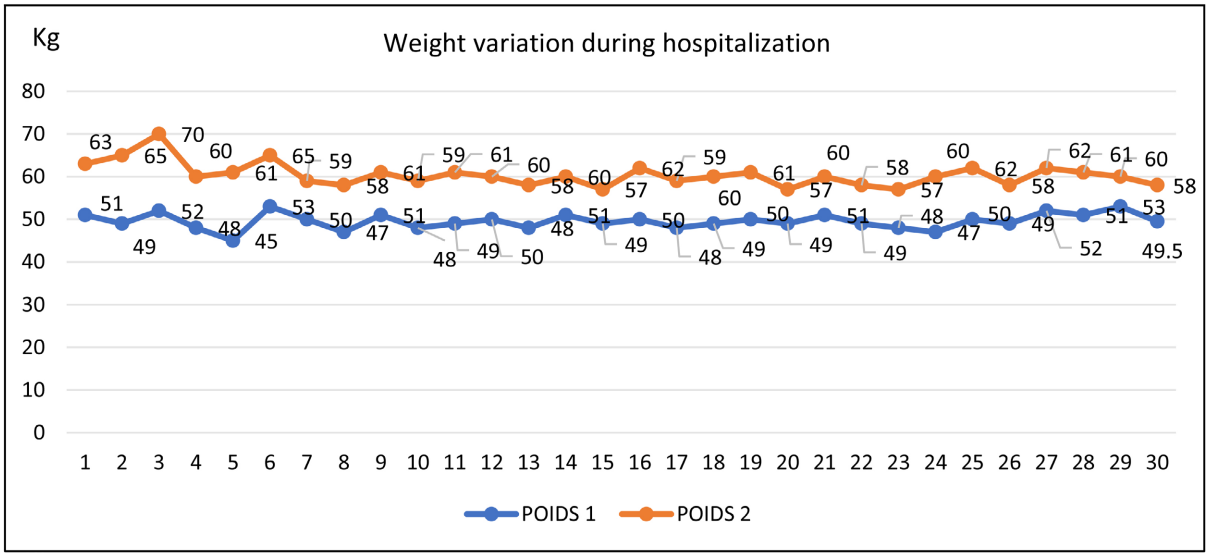
Figure 3. The witness temperature variation.

Analysis of variance according to the Kruskal-Wallis test (P = <0.001) shows a significant difference among the mean values of the studied data.

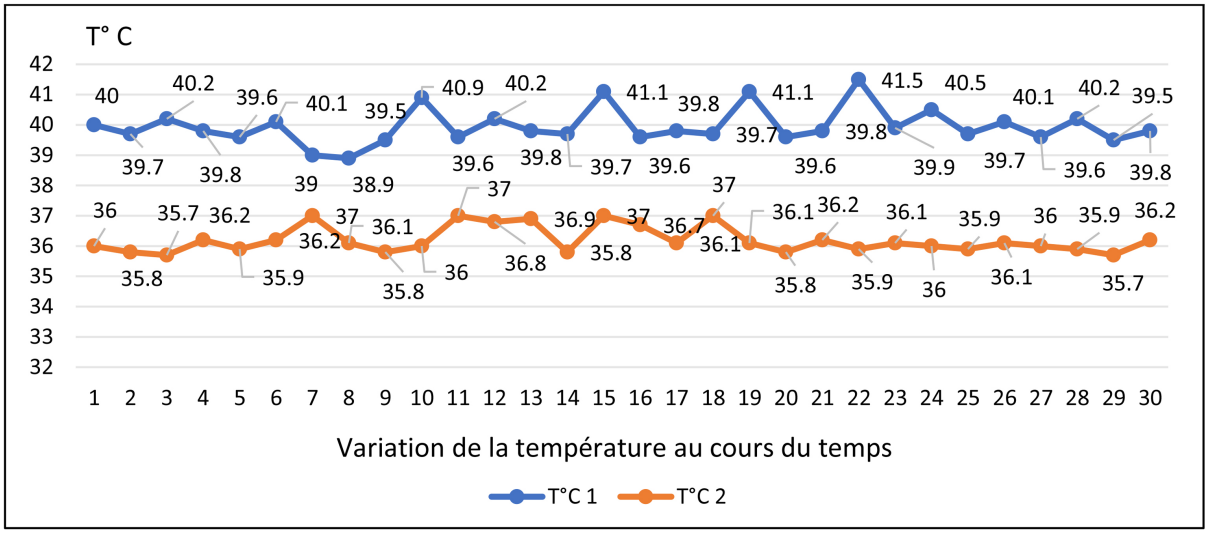
Graph 1 shows the CRP evolution from time hospitalization of the patient as an entry indicator and when subjected to the consumption of the medication.



Graph 1. CRP evolution at the patient's entry and exit.



Graph 2. Weight variation at patient entry and exit.



Graph 3. Temperature variation at patient entry and exit.

While Graph 2 shows the variation in the subject's weight during the patient's internship in order to appreciate the gain in body mass volume giving a comforting quality of satisfaction to the sick patient.

Finally, Graph 3 translates the temperature measurement in order to correlate it with the increase or decrease in fever. We can clearly see a clear difference characterized by decreasing the patient's temperature with an average of 36.05˚C ± 0.262˚C. This situation illustrates the proper functioning of the associated antibiotics, having their effects or having a positive impact on the patient's state of health.

4. Discussions

Anti-tuberculosis drugs act on the different bacillary populations of a patient given that there are several bacillary populations: namely the metabolically active bacilli that multiply rapidly and continuously, they reside most often in the pulmonary caverns, as opposed to the bacilli whose multiplication is slow, located in the macrophages which are slowed down by the absence of oxygen with the acidic pH of the macrophage cytoplasm, or the quiescent or persistent bacilli in the tissues with slow multiplication in bursts, their multiplication is stopped, but remain alive, capable of resuming their activity and multiplying as soon as the immune defenses decrease.

It should be noted that the action of anti-tuberculosis drugs varies depending on their bactericidal, bacteriostatic and/or sterilizing activity on bacillary populations. These drugs have three main properties to remember: one is early bactericidal, capable of rapidly destroying bacilli with an accelerated metabolism, while the sterilizing ones have the capacity to exterminate persistent bacilli with a slow metabolism and finally the bacteriostatic ones have the ability to block or slow the multiplication of bacillary populations, so for effective treatment it is essential to combine these three drugs [13].

It should be noted that the treatment carried out after 15 days allows the patient to no longer be able to transmit or even contaminate another person. Our results demonstrate a drastic reduction in fever and a return to normal temperature, which means that the medication has acted effectively on the body, which is reflected in a significant increase in weight after 15 days. Sputum examination checks are carried out at the end of the second month of treatment, at the fifth month, and at the end of treatment, *i.e.*, at the eighth month.

The results of this prospective study are in line with those of the research work on the comparison of the level of C-reactive protein (CRP) in pulmonary tuberculosis and community-acquired pneumonia by K. Bouytse in Morocco [14].

However, it should be noted that Ethambutol is the subject of withdrawal from patients because it presents several adverse effects due to the toxicity of the drugs used in the treatment of multidrug-resistant tuberculosis.

In the continuation phase, for a duration of 4 months, two molecules are administered to the patient: Rifampicin (R) and Isoniazid (H) [4].

Note that patients who do not adhere to the treatment as prescribed undergo shock treatment lasting 6 months until complete recovery.

The prospective study generally shows a statistically significant difference between the different parameters studied (P = <0.001).

Furthermore, the diagnosis of a bacterial infection in the emergency department is completely different depending on the point of call. Often patients with suspected sepsis are presented in the emergency department, and while it is relatively easy to quickly obtain bacteriological proof from urine, the diagnosis of community-acquired acute pneumonia is more difficult. A study could assess the effectiveness of CRP in the diagnosis of community-acquired acute pneumonia [15].

It should be added that despite hepatic synthesis, CRP is also produced within the atheromatous plaque by macrophages and smooth muscle fibers. Through its ability to bind to numerous ligands (lipids, phospholipids, nucleic acids), CRP forms complexes that cause plaque progression. CRP activates complement, and the resulting cell lysis worsens tissue damage. In this inflammatory environment, the secretion of metalloproteinases (PAPP-A) also contributes to plaque rupture. In addition, the pro-coagulant action of CRP promotes thrombus formation. In this context, it is necessary to use an "ultrasensitive" assay method, *i.e.*, one with a lower detection limit than conventional CRP assays…

The CRPus dosage should be able to reach 0.1 or 0.2 mg/l, and should have a coefficient of variation lower than 10% in low values. The CRPus dosage is proposed mainly in screening and assessment of cardiovascular risk in primary prevention (healthy subjects) a CRPus level lower than 1 mg/l is associated with a low risk of developing a heart problem a level between 1 and 3 mg/l is associated with a moderate risk a level higher than 3 mg/l is associated with a high risk. It must be confirmed on a second sample due to intra-individual biological variations of CRPus. The association of this dosage with other traditional risk factors (lipid profile, smoking, systolic pressure) increases its predictive value [1]. It would be absurd not to be able to mention that several studies have been interested in the same subject given that CRP is an early marker of inflammation (6 hours after the start of the process), that it has a constant clearance rate and a simple and available dosage technique. The results of these studies are controversial but it is currently accepted that even if from a certain value of positivity (variable according to the studies), CRP can indicate the presence of sepsis, a negative value of CRP does not allow to exclude with certainty a bacterial infection in particular in elderly subjects hence the interest in testing new markers such as procalcitonin [16].

5. Conclusion

The results are quite satisfactory given the evolution of the situation of the data received following this study among these patients. However, populations at risk of tuberculosis are defined as populations with high rates of tuberculosis with limited access to tuberculosis care services. It will therefore be necessary to set up an

active screening system in order to improve the early detection of the disease to improve the treatment success rate in order to reduce the duration of the contagious period. This study also shows the scope and quality of implementation of tuberculosis control activities, particularly with regard to the effectiveness of treatment in patients. Despite these results, CRP is a biochemical marker of inflammation and infection that varies with leukocytes. It provides much more in terms of diagnosis of infection or inflammation compared to hyperleukocytosis while remaining correlated with the clinical context. This analytical approach remains preliminary and requires further in-depth studies. [12].

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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