SAR Journal of Medicine

Abbreviated Key Title: *SAR J Med*
Home page: https://sarpublication.com/journal/sarjm/home
DOI: https://doi.org/10.36346/sarjm.2026.v07i01.005

OPEN ACCESS

ISSN 2707-773X (P)
ISSN 2709-6920 (O)

Review Article

Pathophysiology of COVID-19 and Autoimmune and Autoinflammatory Diseases: Acute Infectious Systemic Inflammatory Process on Underlying Chronic Non-Infectious Systemic Inflammatory Process. A Literature Review

Keïta Kaly[1*], Dembélé Ibrahima Amadou[1], Sy Djibril[1,9], Cissoko Mamadou[1], Mallé Mamadou[1], Sangaré Moussa[1], Koné Nouhoum[1], Landouré Sékou[1], Djeugoué Stéphane Loique[1], Camara Boua Daoud[2], Doumbia Alassane Alfousséni[3], Dao Karim[4], Traoré Abdramane[5], Cissé Sékou Mamadou[6], Diallo Seydou F[7], Touré Sidi[7], Pamanta Ibrahima Sory[7], Cissé Idrissa Ahamadou[7,9], Dara Charles[3], Dabo Garan[3,9], Traoré Abdoulaye Mamadou[8,9], Minta Daouda Kassoum [8,9], Traoré Djénébou[1,9], Soukho Assétou Kaya[1,9], Dembélé Mamadou[9], Traoré Hamar Alassane[9]

[1]Department of Internal Medicine, University Hospital Center of the Point G, Bamako, Mali
[2]Department of Medicine, Nianakoro Fomba Regional Hospital of Segou, Segou, Mali
[3]Department of Medicine, University Hospital Center of "Hopital du Mali", Bamako, Mali
[4]Department of Internal Medicine, University Hospital Center of Gabriel Touré, Bamako, Mali
[5]Department of Medicine, Kati University Hospital Center, Bamako, Mali
[6]Department of Medicine and Medical Specialties, Fousseyni Daou Regional Hospital of Kayes, Kayes, Mali
[7]Department of Rheumatology, University Hospital Center of the Point G, Bamako, Mali
[8]Department of Infectious Diseases, University Hospital Center of the Point G, Bamako, Mali
[9]Faculty of Medicine and Odonto-stomatology (FMOS) - University of Sciences, Techniques and Technologies of Bamako (USTTB), Mali

***Corresponding Author:** Keïta Kaly
Department of Internal Medicine, University Hospital Center of the Point G, Bamako, Mali, Email: keitakaly@gmail.com

Article History: | Received: 06.12.2025 | Accepted: 04.02.2026 | Published: 07.02.2026 |

Abstract: The occurrence of COVID-19 in the context of autoimmune or autoinflammatory disease prompts practitioners involved in the management of these conditions to investigate the pathophysiological, clinical, therapeutic, and evolutionary implications. The patients with autoimmune and autoinflammatory diseases that contract COVID-19 present both acute infectious systemic inflammatory process and chronic non-infectious systemic inflammatory process, which raise the interesting research question: is there an over-risk for severe exacerbation? In-deepth understanding of these pathophysiological implications, may allow practitioners involved in the management of these conditions to update and refine their knowledge, enable researchers to pursue the therapeutic targets research, and help health policy makers to adjust and improve prevention and treatment strategies. To provide a relevant synthesis on the interface and the overlap that can exist between these two conditions: acute infectious systemic inflammatory process (COVID-19) and chronic non-infectious systemic inflammatory process (autoimmune and autoinflammatory diseases), we conducted a literature review of the literature. Therefore, this literature review has six objectives. First, to present the etiological factor of COVID-19, the etiopathogenic factors of COVID-19, the pathophyiological factors of COVID-19 and the subsequent clinical expressions. Second, to expose the etiological factor of autoimmune and autoinflammatory diseases, the etiopathogenic factors of autoimmune and autoinflammatory diseases, the pathophyiological factors of autoimmune and autoinflammatory diseases, and the resulting clinical expressions. Third, to analyze the relationship between acute infectious systemic inflammatory process (COVID-19) and chronic non-infectious systemic inflammatory process (autoimmune and autoinflammatory diseases).

Keywords: Pathophysiology, COVID-19, Autoimmune Disease, Autoinflammatory Disease, Literature Review.

1. BACKGROUND

Coronavirus infectious disease 2019 (COVID-19) is a systemic infection caused by SARS-CoV2 [1]. Autoimmune diseases result from a dysfunction of the specific immune system and are subdivided into two groups, systemic autoimmune disease (example: systemic lupus erythematous, rheumatoid arthritis) and organ-specific autoimmune disease (example: diabetes

mellitus autoimmune type 1, Graves' disease) [2, 3]. As for auto-inflammatory diseases, they are the result of a dysregulation of the innate immune system. Thus, two groups are distinguished, monogenic auto-inflammatory disease (example: familial Mediterranean fever, IgD deficiency) and polygenic auto-inflammatory disease (example: spondyloarthropathies, sarcoidosis, Still's disease) [2-4]. Broadly, autoinflammatory diseases reflect disorders of innate immunity while autoimmune and allergic diseases represent disorders of adaptive immunity [5].

Increasingly, case series studies are describing cases of COVID-19 in patients with autoimmune and autoinflammatory diseases. These include systemic lupus erythematosus, antiphospholipid syndrome, rheumatoid arthritis, psoriatic arthritis, etc [6-9]. The occurrence of COVID-19 in the context of autoimmune or autoinflammatory disease prompts practitioners involved in the management of these conditions to investigate the pathophysiological, clinical, therapeutic, and evolutionary implications.

The patients with autoimmune and autoinflammatory diseases that contract COVID-19 present both infectious systemic inflammatory process and non-infectious systemic inflammatory process, which raise the interesting research question: is there an over-risk for severe exacerbation? In-deepth understanding of these pathophysiological implications, may allow practitioners involved in the management of these conditions to update and refine their knowledge, enable researchers to pursue the therapeutic targets research, and help health policy makers to adjust and improve prevention and treatment strategies. To provide a relevant synthesis on the interface and the overlap that can exist between these two conditions: acute infectious systemic inflammatory process (COVID-19) and chronic non-infectious systemic inflammatory process (autoimmune and autoinflammatory diseases), we conducted a literature review of the literature.

Therefore, this literature review has six objectives. First, to present the etiological factor of COVID-19, the etiopathogenic factors of COVID-19, the pathophyiological factors of COVID-19 and the subsequent clinical expressions. Second, to expose the etiological factor of autoimmune and autoinflammatory diseases, the etiopathogenic factors of autoimmune and autoinflammatory diseases, the pathophyiological factors of autoimmune and autoinflammatory diseases, and the resulting clinical expressions. Third, to analyze the relationship between acute infectious systemic inflammatory process (COVID-19) and chronic non-infectious systemic inflammatory process (autoimmune and autoinflammatory diseases).

2. METHODS

An in-depth literature search was conducted over a two-month period to extract articles relevant to this narrative review study. Research articles in English and French dating from December 2019 to May 2020 were reviewed using databases such as PubMed, Google Scholar, and press articles. A combination of keywords such as “SARS-CoV2,” “etiopathogenic factors of COVID-19,” “pathophysiology of COVID-19,” “etiopathogenic factors of each autoimmune,” “etiopathogenic factors of each autoinflammatory diseases,” “pathophysiology of each autoimmune,” “pathophysiology of each autoinflammatory diseases,” was searched. The extracted articles were then reviewed to verify their relevance to the objectives of this work. A total of 33 articles were read for the final review after discarding 38 irrelevant articles.

3. Pathophysiology of COVID-19 and Autoimmune and Autoinflammatory Diseases

3.1. Acute Infectious Systemic Inflammatory Process

3.1.1. What is the Etiological Factor of COVID-19?

COVID-19 is characterized by an acute infectious systemic inflammatory process, which is to emphasize the infectious inducers role in inflammatory process triggering.

SARS-CoV-2 was first isolated in bronchoalveolar lavage fluid from three COVID-19-positive patients at Wuhan Jinyintan Hospital on December 30, 2019. After sequencing and genealogical analysis, SARS-CoV-2 was considered a member of the Beta-CoV family. The CoV family is a class of enveloped, positive-sense, single-stranded RNA viruses [10].

Studies have shown that wearing an N95 mask reduces the risk of SARS-CoV-1 infection by 91%. Wearing an apron/gown reduces this risk by 77%, wearing a surgical mask by 68%, and frequent hand washing by 55%. These observations strongly suggested human-to-human transmission via airborne droplets or aerosols, fecal-oral transmission, or contact [11].

SARS-CoV-2 has been detected in type II alveolar epithelial cells, monocytes, digestive tract epithelial cells, distal renal tubular cells, skin sweat gland cells, parathyroid and pituitary eosinophils, adrenal cortex cells, gastric parietal cells, pancreatic cells, acinar cells, glial cells, and tracheal serous gland cells [12].

3.1.2. What are the Etiopathogenic Factors of COVID 19?

Since COVID-19 was discovered in China in December 2019, several studies on its etiopathogenesis have been conducted, focusing in particular on the interaction between SARS-CoV-2 and several cells with ACE 2 (angiotensin-converting enzyme 2) receptors and the interaction between SARS-CoV-2 and red blood cells [12, 13]. Several types of elementary lesions resulting from these interactions have also been described [10-14].

3.1.3. What are the Pathophyiological Factors of COVID-19? And what are the Clinical Expressions of These Pathophysiological Consequences?

The pathophysiological consequences and their clinical manifestations are also numerous and sometimes severe, such as macrophage activation syndrome resulting from cytokine storm and microvascular thrombosis resulting from coagulation disorders [10-15]. A summary of data on the main interactions between SARS-CoV-2 and target cells in the body, as well as their pathophysiological consequences and clinical manifestations, is provided in table 1 [1-24].

Table 1: The main interactions between SARS-CoV-2 and the body's target cells with ACE2 receptors, their pathophysiological consequences, and their clinical manifestations.

Etiopathogenics	Pathophysiological consequences	Clinical manifestations	References
Interaction between SARS-CoV-2 and ACE2 receptor-expressing cells (type 2 alveolar epithelial cells, digestive tract epithelial cells, hepatobiliary cells, pancreatic cells, etc.) via their respective receptors: spike glycoprotein and ACE2-R, leading to their internalization into the cells.	Massive viral replication with necrosis of R-ACE2-expressing cells such as destruction of type 2 pneumocytes with production of hyaline membrane plus damage to the alveolar-capillary barrier, disruption of the absorptive functions of the intestine, destruction of hepatobiliary cells, destruction of Langerhans Beta cells of the pancreas.	Cough, dyspnea, diarrhea, cytolysis and/or cholestasis, hyperglycemia.	[10, 16, 17, 18]
	Activation of the inflammatory process: vasodilation, vascular hyperpermeability, infiltration of inflammatory cells leading to the release of pyrogenic cytokines, or even a cytokine storm (IL6, IL8, IL10, TNF, VEGF).	Fever, COVID-19–related macrophage activation syndrome.	
	Activation of the immune system characterized by activation and production of effectors (NK cells, TCD8 lymphocytes, antibodies) and the release of pro-inflammatory cytokines.	Healing occurs if the immune response is appropriate, or if inappropriate the inflammatory process will persist and worsen.	
	Accumulation of angiotensin II by competitive inhibition of its ACE2 receptor leading to the activation of the specific receptor present on CD8 T lymphocytes.	Enhancement of the cell-mediated immune response (CD8 T cells).	
Interaction between SARS-CoV-2 surface spike glycoproteins and the CD147 of the red blood cell.	Reduction in hemoglobin's ability to transport and efficiently exchange CO2 and O2 across the alveolar-capillary barrier, which over time leads to deterioration of the alveolar-capillary barrier and may eventually progress to pulmonary fibrosis.	Acute respiratory distress syndrome.	[13, 10]
Interaction between SARS-CoV-2 and gut microbiota.	Modification de microbiote qui favoriserait l'apparition de l'orage cytokinique.	COVID-19–associated macrophage activation syndrome	[19]
Interaction between SARS-CoV-2 and endothelial cells.	Endothelial cell damages lead to the destruction of endothelial cells and fragilisation of blood vessels.	Hemorrhage from pulmonary microvessels (hemoptysis).	[15]
	Increased activation of the inflammatory process (vasodilation, vascular hyperpermeability, and release of pro-inflammatory cytokines), leading to inflammation of the walls of small blood vessels.	Systemic vasculitis, particularly of small vessels (skin, lungs, etc.).	

Etiopathogenics	Pathophysiological consequences	Clinical manifestations	References
	Activation of the coagulation system and fibrinolytic system, particularly at the microvascular level (resulting from systemic inflammation, endothelial dysfunction, severe hypoxemia, and the production of antiphospholipid antibodies), leads to clot formation.	Thrombosis of pulmonary microvessels (furthermore pulmonary embolism and pulmonary arterial hypertension) and renal microvessels, deep vein thrombosis, disseminated Intravascular Coagulation (DIC).	
Interaction between SARS-CoV-2 and cardiomyocytes	Direct cytotoxicity of SARS-CoV-2 associated with hypoxia resulting from gas exchange obstruction, leading to acidosis and an increase in intracellular free radicals damaging phospholipid membranes, resulting in the destruction of cardiomyocytes.	Myocarditis linked to SARS Cov2.	[1, 10]
	Activation of the inflammatory process (lymphocytic infiltration and vascular hyperpermeability) leading to inflammation of the myocardium.		
Interaction between SARS-CoV-2 and renal epithelial cells.	Direct cytotoxic effect of SARS-CoV-2, resulting in the destruction of renal epithelial cells with disruption of water-electrolyte and acid-base homeostasis and glomerular filtration.	Acute tubular necrosis and/or acute glomerulonephritis with kidney failure.	[20, 21]
Interaction between SARS-CoV-2 and glial cells.	Glial cells are affected via the bloodstream (blood-brain barrier) or the synaptic pathway, followed by viral replication leading to respiratory center dysfunction, meningeal irritation, and inflammation of the brain (hippocampus).	Central dyspnea or even Acute respiratory distress syndrome, meningeal and encephalitic manifestations.	[22, 23]
Interaction between SARS-CoV-2 and skeletal muscle cells.	Direct cytopathogenic effect of SARS-CoV-2 on skeletal muscle cells, resulting in damage to these cells and the release of myoglobin, lactate dehydrogenase, and creatine phosphokinase.	Rhabdomyolysis, acute tubular necrosis.	[23]
Other etiopathogenic aspects without direct cytopathogenic effect.	Dehydration.	Acute tubular necrosis and/or acute glomerulonephritis with renal failure.	[21, 1, 24]
	Release of catecholamines secondary to anxiety related to COVID-19.	Myocarditis.	
	Microvesicular steatosis.	Non-alcoholic fatty liver disease.	
ACE-2 receptor: Angiotensin II converting enzyme receptor ; IL6 : interleukin 6 ; IL8 : interleukin 8 ; IL10 : interleukin 10 TNF : tumor necrosis factor, VEGF : vascular endothelial growth factor			

3.2. Chronic Non-Infectious Systemic Inflammatory Process

Autoimmune and autoinflammatory diseases are characterized by chronic non-infectious systemic inflammatory process, which is to underline the non-infectious inducers role in inflammatory process triggering. Although, some of these autoimmune and autoinflammatory diseases present the local inflammatoire process at the early disease onset but the systemic inflammatory process may be observed at the late disease course or during the disease flares.

3.2.1. What Are the Etiological Factors of Autoimmune and Autoinflammatory Diseases?

As for autoimmune and autoinflammatory diseases, these are noninfectious conditions resulting from dysregulation of the innate (autoinflammatory diseases) and adaptive (autoimmune diseases) immune systems of unknown etiology. In addition, according to

dysregulated immunity leads to human disease: autoinflammation (aberrant antigen-independent immune activation), autoimmunity / allergy (aberrant antigen-dependent immune activation), and immunodeficiency (defects in innate or adaptive immunity resulting in inadequate defense against pathogens) [5].

3.2.2. What Are the Etiopathogenic Factors of Autoimmune and Autoinflammatory Diseases?

Unlike COVID-19, in systemic lupus erythematosus, described as the prototype of systemic diseases, the elementary lesions (tissue inflammation) observed result from the interaction between discrete genetic factors that predispose the immune system and environmental factors, in particular, leading to excess production and/or defective clearance of apoptotic cells, inducing the accumulation of cellular debris and, consequently, an abnormal immune response with tissue deposition of immune complexes, complement activation, cytokine secretion, and lymphocyte cytotoxicity, inducing tissue inflammation [25].

3.2.3. What are the Pathophyiological Factors of Autoimmune and Autoinflammatory Diseases? And what are the Clinical Expressions of These Pathophysiological Consequences?

The pathophyiological factors of autoimmune diseases mainly include autoantibody activation, defective apoptosis clearance, innate immune activation, complement activation and those of autoinflammatory diseases include inflammasomopathies, interferonopathies, IL1 related manifestations, NFκbopathies, which broadly result in the chronic inflammation causing tissue damage, most commonly manifesting as nephritis (kidney), arthritis (joints), dermatitis (skin), fever, abdominal pain and vasculitis [26].

3.3. Acute Infectious Systemic Inflammatory Process on Chronic Non-Infectious Systemic Inflammatory Process

Admittedly, the inflammatory process is the main pathological process both in COVID-19 and in autommune and autoinflammatory diseases. Thus, the elementary lesions encountered in COVID-19 have the same characteristics as those found in certain autoimmune and autoinflammatory diseases, such as an increase in antiphospholipid antibodies responsible for clot formation (antiphospholipid syndrome, systemic lupus erythematosus) [27]. Similar pathophysiological consequences are also found in certain autoimmune and autoinflammatory diseases as well as in COVID-19, such as pulmonary fibrosis (systemic lupus erythematosus, rheumatoid arthritis, sarcoidosis, etc.) [28, 29, 30], and the release of a cytokine storm (Still's disease) [31].

Is acute infectious systemic inflammatory process on chronic non-infectious systemic inflammatory process occuring lead to over-risk for severe exacerbation? At the end of our review of the literature, none of these studies allowed us to conclude that the onset of COVID-19 in autoimmune and autoinflammatory diseases promoted the worsening or resumption of disease activity. However, the impact of COVID-19 on pre-existing conditions that can complicate autoimmune and autoinflammatory diseases, such as heart disease and chronic kidney failure, is increasingly being described. As demonstrated in his study, myocardial infarction is possible when there is underlying ischemic heart disease or comorbidities in patients who test positive for COVID-19 [32]. In addition, COVID-19 worsens the prognosis of pre-existing chronic kidney disease (CKD). Based on a meta-analysis of available data, CKD appears to be associated with an increased risk of severe COVID-19 infection [33].

4. CONCLUSION

COVID-19 occurring in autoimmune and autoinflammatory diseases is increasingly being reported. Autoimmune and autoinflammatory diseases are characterized by chronic systemic non-infectious inflammatory process, while COVID-19 by acute systemic infectious inflammatory process.

Although, the different underlying etiological factors: SARS-CoV-2 for COVID-19, autoantigens for autoimmune diseases, and dysregulation of the innate immune system for autoinflammatory diseases, similar pathophysiological consequences are also found in certain autoimmune and autoinflammatory diseases as well as in COVID-19, such as pulmonary fibrosis, the release of a cytokine storm, and so on.

Competing Interests: The authors declare no competing interests.

REFERENCES

1. Li B, Yang J, Zhao F, Zhi L, Wang X, Liu L, Bi Z, ZhaoY. Prevalence and impact of cardiovascular metabolic diseases on COVID-19 in China. *Clinical Research in Cardiology.* 2020 ; 109 : 531–538.
2. Guillevin L, Meyer O, Hachulla E, Sibilia J. Traité des Maladies et syndromes systémiques. In : TRON Fet Jean-François BACH JF, Eds. Concepts actuels de l'auto-immunité. 6ème édition. Paris, Lavoisier, 2015.
3. Bouchekout R. Etude épidémio-clinique et biologique des maladies auto-immunes exprimant des facteurs anti nucléaires : particularité du lupus. *Mémoire,* Algérie, 2016, 90p.
4. Collège Français des Enseignants en Rhumatologie (COFER). Item 112 : Réaction inflammatoire. Aspects biologiques et cliniques, conduite à tenir. *Retrieved* 06 april 2019.
5. Nigrovic PA, Lee PY, Hoffman HM. Monogenic autoinflammatory disorders: conceptual overview, phenotype, and clinical approach. *J Allergy Clin Immunol.* 2020 November; 146(5): 925–937. doi: 10.1016/j.jaci.2020.08.017

6. Mathian A, Mahevas M, Rohmer J et al. Clinical course of coronavirus disease 2019 (COVID-19) in a series of 17 patients with systemic lupus erythematosus under long-term treatment with hydroxychloroquine. *Ann Rheum Dis.* 2020 Vol 0 No 0.
7. Zhang Y, Xiao M, Zhang S et al. Coagulopathy and Antiphospholipid Antibodies in Patients with Covid-19. *N Engl J Med* 2020; 382: e38.
8. Monti S, Balduzzi S, DelvinoP, Bellis E, Quadrelli VS, Montecucco C. Clinical course of COVID-19 in a series of patients with chronic arthritis treated with immunosuppressive targeted therapies. *Ann Rheum Dis.* 2020; 79: 667–668.
9. Manalo IF, Smith MK, Cheeley J, Jacobs R. A Dermatologic Manifestation of COVID-19: Transient Livedo Reticularis. *Journal of the American Academy of Dermatology.* 2020.
10. Yuefei J, Haiyan Y , Wangquan J , Weidong W , Shuaiyin C , Weiguo Z, Guangcai D. Virology, Epidemiology, Pathogenesis, and Control of COVID-19. *Viruses.* 2020 ; 12, 372.
11. Placais L, Richier Q. COVID-19: caractéristiques cliniques, biologiques et radiologiques chez l'adulte, la femme enceinte et l'enfant. Une mise au point au cœur de la pandémie. *La Revue de médecine interne.* 2020 (41) 308–318.
12. Wenling W, Yanli X, Ruqin G, Roujian L, Kai H, Guizhen W, Wenjie T . Detection of SARS-CoV-2 in Different Types of Clinical Specimens. *JAMA.* 2020; 323(18):1843-1844.
13. Wenzhong L and Hualan L. COVID-19: Attacks the 1-Beta Chain of Hemoglobin and Captures the Porphyrin to Inhibit Human Heme Metabolism. 2020. Retrieved 15 mai 2020.
14. Hanley B, Lucas SB, Youd E, Swift B, Osborn M. Autopsy in suspected COVID-19 cases. *J Clin Pathol.* 2020; 73: 239–242.
15. McGonagle D, O'Donnell JS, Sharif K, Emery P, Bridgewood C. Immune mechanisms of pulmonary intravascular coagulopathy in COVID-19 pneumonia. *Lancet Rheumatol.* 2020.
16. Silva-Filho J, Caruso-Neves C, Pinheiro A. Angiotensin II type-1 receptor (AT1R) regulates expansion, differentiation, and functional capacity of antigen-specific CD8+ T cells. *Sci Rep.* 2016; 6, 35997.
17. Vivier E, Elena E, Baratin M, Walzer T. « Functions of natural killer cells ». *Nature Immunology* 2008; 9 (5): 503–510.
18. Meijuan Z, Yong G, Gang W, Guobin S Siyu L, Dandan S, Yuanhong X, Zhigang T . « Functional exhaustion of antiviral lymphocytes in COVID-19 patients ». *Cellular & Molecular Immunology.* 2020; 17: 533–535. DOI 10.1038/s41423-020-0402-2.
19. Lei P, Mi M, Hong GR, and Lei T et al. Clinical characteristics of COVID-19 patients with digestive symptoms in Hubei, China: a descriptive, cross-sectional, multicenter study. *Am J Gastroenterol.* 2020; 115(5):766-773.
20. Hamid Rabb. Kidney diseases in the time of COVID-19: major challenges to patient care. *J Clin Invest.* 2020; 1 – 3.
21. Valizadeh R, Baradaran A, Mirzazadeh A, Bhaskar LV. Coronavirus-nephropathy; renal involvement in COVID-19. *J Renal Inj Prev.* 2020; 9(2): e18.
22. Moriguchi T, Harii N, Goto J, and Shimada S et al. A first Case of Meningitis/Encephalitis associated with SARS-Coronavirus-2. *International Journal of Infectious Diseases* 2020; 94: 55-58
23. Ling M, Mengdie W, Shengcai C. Neurological Manifestations of Hospitalized Patients with COVID-19 in Wuhan, China: a retrospective case series study. *medRxiv.* 2020 preprint.
24. Dong J, Enqiang Q, Jing X et al. Non-alcoholic fatty liver diseases in patients with COVID-19: A retrospective study. *J Hepatol.* 2020; S0168-8278(20)30206-3.
25. Mathian A, Arnaud L, Amoura Z. Physiopathologie du lupus systémique: le point en 2014 Etiopathogenesis of systemic lupus erythematous: A 2014 update. *La Revue de Médecine Interne* 2014; 35(8): 503-511.
26. José C Crispín JC, S-NC, Katalin Kis-Toth [1], Lieberman LA, et al. Pathogenesis of human systemic lupus erythematosus: recent advances. *Trends Mol Med.* 2010 Feb 4;16(2):47–57. doi: 10.1016/j.molmed.2009.12.005
27. Encyclopédie Orphanet Grand Public. Le syndrome des antiphospholipides. Juillet 2008. *Retrieved* 15 may 2020.
28. Arnaud I, Amoura Z. Lupus érythémateux systémique. EMC-Traité de Médecine AKOS 2012 ; 7(2) : 1 – 9
29. Falgarone G, Marie-Christophe Boissier MC. Polyarthrite rhumatoïde. *La Revue du Praticien.* 2011 ; (61) : 119 – 126.
30. Chapelon-Abric C. Sarcoïdose. EMC-Traité de méecine AKOS 2015; 10(3): 1 – 4.
31. Gerfaud-Valentin M, Sève P, Hot A, Brousselle C, Jamilloux Y. Données actualisées sur la physipathologie, les phénotypes et es traitements de la maladie de Still de l'adulte. *La Revue de Médecine interne.* 2015 ; 36 : 319 – 327.
32. Chapman AR Bularga A, Mill NL. High-Sensitivity Cardiac Troponin Can Be an Ally in the Fight Against COVID-19. *Circulation.* 2020.
33. Brandon M H, Giuseppe L. Chronic kidney disease is associated with severe coronavirus disease 2019 (COVID-19) infection. *Int Urol Nephrol.* 2020 ; 1–2.