

Individuals afflicted with concurrent anemia, tuberculosis, and *Candida albicans* infection manifest: A pronounced state of immunosuppression

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Abstract

This study delved into the immune functionality of individuals grappling with anemia, tuberculosis, and *Candida albicans* infection. The findings conclusively revealed that this cohort manifested a pronounced state of immunosuppression, thereby emphasizing the intricate interplay of these concurrent maladies on the immune landscape. Subsequent investigations should aim to unravel the underlying mechanisms orchestrating this immunosuppressive milieu and to contemplate potential interventions aimed at alleviating its impact on immune response dynamics.

Keywords: Anemia; Tuberculosis; *Candida albicans* infection; Immunosuppression; Immune function; Co-presence; Health effects; Combined effects of disease.

Introduction

The intricate nexus between infectious diseases and the immune system has, over an extended period, commanded the persistent attention of both researchers and clinicians. It is pertinent to underscore that the simultaneous presence of anemia, tuberculosis, and *Candida albicans* infection within individuals engenders thought-provoking inquiries regarding the repercussions of this convergence on immune functionality. A mounting body of evidence corroborates the deleterious influence of anemia on the clinical trajectory of the ailment [1]. Tuberculosis and *Candida albicans* imposition two distinct yet potent challenges upon the immune system, effectively underscoring the fragile equilibrium the body must assiduously preserve to counteract pathogenic invasion [2,3]. Within the context of individuals afflicted by concurrent anemia, tuberculosis, and *Candida albicans* infection, the overt manifestation of an immunosuppressive state introduces an added stratum of intricacy into the comprehension of immune response kinetics. With due

consideration for the potential repercussions upon the course of disease and therapeutic outcomes, the present case report effectively elucidates the distinctive clinical milieu encountered by individuals concurrently grappling with anemia, Tuberculosis, and candida albicans infection.

Case Presentation

The patient was diagnosed with secondary pulmonary tuberculosis and mediastinal lymph node tuberculosis a year prior. This was followed by a 12-month course of anti-tuberculosis therapy in conjunction with interventions targeting microcirculation enhancement and counteractive measures against systemic sclerosis. During the examination conducted one month ago, the patient exhibited a lower hemoglobin level compared to previous assessments, leading to the suspension of all anti-tuberculosis medications. Recently, the patient experienced fatigue, albeit without concurrent symptoms of nausea, emesis, abdominal discomfort, diarrhea, or fever. While a sporadic mild cough and expectoration of small quantities of white sputum were noted, no indications of chest tightness or dyspnea were present. The patient also bore a medical history of bilateral digital edema and induration persisting for over two years, with superficial ulcers observed on certain finger tips. Upon consultation within our hospital's rheumatology department, the patient received a diagnosis of systemic sclerosis with concomitant Raynaud's syndrome, subsequently undergoing hormonal and immunosuppressive therapy. A recent high-resolution chest computed tomography (CT) scan revealed persistent infectious lesions within both lung fields, with marginal alterations in extent from prior assessments. Simultaneously, localized bronchial dilation and pleural thickening were observed bilaterally. Similar to previous images, the left lung exhibited an air sac phenomenon and aortic calcification.

Laboratory findings indicated positive mycobacterium cultures and drug susceptibility tests in alveolar lavage fluid for the mycobacterium tuberculosis complex group, while routine stool examinations revealed microscopic evidence of candida. The patient exhibited an elevated neutrophil percentage, heightened levels of highly sensitive C-reactive protein, and an increased reticulocyte count. Indices including urea, aspartate aminotransferase, γ -glutamyltransferase, creatinine, blood glucose, and prothrombin time demonstrated increments, whereas hemoglobin, total protein, and albumin showed a decline. In summary, the patient's comprehensive diagnosis encompasses secondary pulmonary tuberculosis (upper middle lower/upper middle lower tubi (-) (+) primary treatment), mediastinal lymph node tuberculosis, systemic sclerosis, Raynaud's syndrome, chronic arterial occlusion (occlusive arteritis), and moderate anemia.

The patient received a diagnosis of systemic sclerosis and Raynaud's syndrome, prompting the initiation of hydroxychloroquine tablets at a dose of 0.2 mg administered orally twice daily to modulate immune function. In parallel, Kenar 40 micrograms, administered orally thrice daily, was employed to augment peripheral microcirculation. Furthermore, prednisone tablets at a dosage of 10 mg were orally administered once daily for their potent anti-inflammatory attributes. Complementing this regimen, methotrexate tablets, Each containing 10 mg, were taken orally on a weekly basis to exert inhibitory effects on the progression of systemic sclerosis. Concurrently, the patient was prescribed 2 capsules of shuilinjia, orally administered thrice daily, to safeguard hepatic function. Omeprazole enteric capsules, containing 20 mg, were orally ingested once daily on an empty stomach to mitigate gastric acid secretion. Subsequent chest

CT examinations unveiled the presence of multiple patchy, linear, and nodular high-density opacities marked by indistinct margins in both lung fields, with a predilection for the lower left lobe. The patient presented with symptoms of cough, productive sputum, and dyspnea. Notably, chest CT scans indicated an escalated degree of pneumonic exudation within the lower left quadrant. Fiberoptic bronchoscopy corroborated bronchial inflammatory alterations, precipitating the adjunctive administration of moxifloxacin for antimicrobial intervention. Alveolar lavage cultures revealed the presence of *Candida albicans* infection, prompting the incorporation of fluconazole at a dose of 0.4 g administered intravenously once daily to combat fungal proliferation. Concomitantly, the detection of mycobacterium tuberculosis DNA instigated the initiation of a dual regimen involving isoniazid at 0.3 g, administered once daily, along with levofloxacin at a dose of 0.5 g, orally administered once daily for targeted anti-tuberculosis therapy. Noteworthy is the temporary suspension of glucocorticoids and immunosuppressants during this therapeutic course.

The patient remained afebrile, occasionally experienced coughing and sputum production, while no manifestations of chest tightness or dyspnea were reported. Moreover, renal function was successfully restored, facilitating the patient's discharge. A continued regimen of oral fluconazole capsules for antifungal therapy spanning two weeks was advised, concurrent with the ongoing anti-tuberculosis regimen.

Discussion

The etiology of anemia is intricately intertwined with oxidative stress, inflammation, and immune responses, precipitating the development of vasculopathy and other intricate complications [4]. Mycobacterium tuberculosis's pathogenicity involves alveolar tissue destruction, provoking pulmonary inflammation, and inducing immune reactions within lymphocytes and plasma cells, consequently modulating the immune milieu. This inflammatory context disrupts iron homeostasis, fostering anemia through aberrant iron distribution and utilization. Reduced iron absorption and concurrent sequestration in macrophages diminish the available iron pool, subsequently compromising erythropoiesis. Notably, mycobacterium tuberculosis has the potential to breach the blood-brain barrier, colonize the central nervous system, and propagate locally, culminating in conditions like meningitis and brain abscess. When mycobacterium tuberculosis gains access to the bloodstream, it incites septicemia and, in severe instances, bacteremia, contributing to anemia. Tuberculosis prompts symptoms like reduced appetite and weight loss, curbing nutrient intake and compromising the availability of anemia-related nutrients like iron, Vitamin B12, and folic acid, thereby exacerbating anemia's onset. Furthermore, anti-tuberculosis interventions can detrimentally affect hematopoiesis, fostering anemia. Conversely, anemia hastens susceptibility to mycobacterium tuberculosis infection.

In the context of *Candida albicans* infection, a pivotal mechanism comes to light: Aire + MHCII + innate lymphoid cells (ILC3s), which possess the ability to detect, internalize, and present *Candida albicans* antigens. This intricate process assumes a central role in orchestrating the generation of candida-specific T-helper [17] (TH17) cell clones, thus shaping the immune response [5]. *Candida albicans* infection, in its wake, has the propensity to incite an inflammatory cascade, galvanizing immune activation. This inflammatory milieu, in turn, can perturb iron homeostasis within the body, consequently impinging upon the erythropoietic process and culminating in anemia.

In the management of *Candida albicans* infection, the utilization of immunosuppressive agents to modulate inflammation and immune responses might become imperative. Nevertheless, these pharmacological interventions can engender deleterious repercussions on immune system dynamics, thereby influencing its efficacy. Immunosuppression, while aimed at quelling excessive immune activity, concurrently dampens the body's defense mechanisms against diverse pathogens, thereby rendering individuals more susceptible to infections. Within an immunosuppressed context, the vulnerability to infections-comprising anemia, tuberculosis, and *Candida albicans*-can be substantially heightened.

The ramifications of immunosuppression can extend to the realm of hematopoiesis, particularly the erythropoietic cascade. Immunosuppressants, by virtue of their mode of action, may intrude upon the normal processes governing red blood cell production, precipitating anemia. Moreover, certain immunosuppressive agents possess the potential to impede the generation of hematopoietic cells, thereby exacerbating the existing anemic state. *Candida albicans*, a ubiquitous fungal entity, stands as an exemplar. In the milieu of immunosuppression, the body's vigilant defenses against *Candida albicans* are attenuated, effectively affording the opportunistic spread of candida infection.

Importantly, it merits attention that the nexus between immunosuppression and the triad of anemia, tuberculosis, and *Candida albicans* infections isn't unidirectional; rather, it assumes a reciprocal and amplifying dynamic. This intricate interplay perpetuates a reinforcing cycle, wherein immunosuppression fosters the emergence of these conditions, and in turn, their presence further fuels the immunosuppressive milieu. Such a self-perpetuating cycle begets a formidable challenge for patients in their quest for effective disease management and treatment.

Conclusions

In summation, the concurrent presence of individual anemia, tuberculosis, and *Candida albicans* infection is intricately linked to a pronounced state of immunosuppression. This revelation underscores the intricate interplay woven among these coexisting pathologies, delineating their shared impact on the immune system's equilibrium.

Declarations

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