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Review Article

Fibrinogen to Albumin Ratio and its Possible Role among Systemic Lupus Erythematosus Patients: A Review Article

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ABSTRACT

Background: There is a vast variety of clinical manifestations and illness courses that can be associated with systemic lupus erythematosus (SLE), a chronic autoimmune disorder that affects multiple systems, including the kidneys, liver, and neurological system. There is a significant gender gap when it comes to SLE, with females being diagnosed nine times more frequently than males. Patients who have SLE display essential clinical characteristics such as hypercoagulability, an exaggerated inflammatory and autoimmune responses. Within the framework of inflammation and chronic inflammation-related disorders, there is an intriguing field of study involving fibrinogen (FIB), albumin (ALB), and their ratio, fibrinogen-to-albumin ratio (FAR). We intended to outline an overview about Fibrinogen to Albumin Ratio and its possible use for assessment of disease activity of Systemic Lupus Erythematosus.

Conclusions: In SLE, there is often endothelial dysfunction, which can contribute to inflammation and hypercoagulability. Elevated FAR might be associated with endothelial damage, as fibrinogen is involved in clot formation, and albumin has potential anti-inflammatory properties. FAR could serve as a marker for endothelial injury and its contribution to SLE pathogenesis.

Keywords: Fibrinogen to Albumin Ratio; Roles; Systemic Lupus Erythematosus

INTRODUCTION

An autoimmune illness that affects multiple systems is known as systemic lupus erythematosus (SLE). Clinical signs might range from modest mucocutaneous indications to severe involvement of many organs and the central nervous system, reflecting the fact that the disorder has multiple phenotypes. The etiology of SLE is complex and involves multiple immunopathogenic mechanisms. Later, a number of harmful autoantibodies were discovered. Technology and our knowledge of the pathophysiology and risk factors for SLE have both advanced in recent years, but the exact aetiology is still a mystery. While other criteria for SLE classification have been proposed, the practicality of these criteria in clinical practice remains debatable. Organ system involvement dictates SLE management. Patients with SLE continue to face a substantial risk of morbidity and mortality, even though there are several effective medications for the treatment of the condition [1].

North America has the greatest reported incidence and prevalence of SLE globally, while Africa has the lowest incidence, and Australia has the lowest prevalence. This disparity is strongly influenced by location. The clinical outcome and therapy of the condition are greatly influenced by age, gender, and ethnicity. Although SLE is more common in women, men experience a more rapid and severe progression of the disease, leading to a less favorable prognosis. Environmental factors and genetic variations are thought to be responsible for this discrepancy [2].

Incidence rates for African Americans are 31.4 per 100,000 per year, whereas those for Caucasians are 6.73 per 100,000. The rate of occurrence is 517 per 100,000 among Black Americans, compared to 134 per 100,000 among White Americans and Europeans in the United States [3].

Among the most gender-differentiated autoimmune illnesses, SLE primarily affects women between the ages of 15 and 44, when they are in the prime of their reproductive years (9:1). Severe Lupus Erythematosus (SLE) is a prevalent illness among women of childbearing age and has been associated with several medical and psychological complications that impact fertility and family planning [4].

The intricate interplay between the genome and the exposome (environmental impact) results in an epigenetic alteration that modifies the expression of genes that have a role in the etiology of SLE. For those who are genetically predisposed, environmental factors like ultraviolet B (UVB) radiation, infections, and pollutants can cause a breakdown in immunological tolerance and the abnormal activation of autoimmunity [5]. When self-antigens are exposed to immune cells, a feedback loop between innate and adaptive immunity is set in motion, maybe because of an increased apoptotic cell load. The clinical picture of systemic lupus erythematosus (SLE) happens when complement activation, cytokine release, autoreactive T cells and B cells, and the subsequent creation of autoantibodies and immune complexes cause extensive tissue damage [6].

The clinical symptoms of SLE are diverse and the profile of autoantibodies is extensive. Due to the wide range of possible symptoms and results from tests, a correct diagnosis is quite difficult to achieve. A combination of clinical symptoms, serological results, imaging, and histology is required for a definitive diagnosis of SLE, placing a premium on medical expertise [7].

Biomarkers

To diagnose systemic lupus erythematosus, evaluate disease activity, categorize sequelae, and evaluate therapeutic intervention efficacy, biomarkers are important. Unfortunately, because to the complicated pathophysiology and clinical variability of SLE, it is tough to find a single biomarker that adequately reflects the disease's status. Also, no one biomarker has demonstrated perfect sensitivity and specificity for SLE; thus, it may be more effective to use a panel of biomarkers that reflect several disease presentations to evaluate SLE [8].

The liver is responsible for synthesizing fibrinogen, a 340 kDa hexameric plasma glycoprotein. The process of making fibrinogen is encoded by three separate genes on chromosome 4. There is a concentration of about 200-400 mg/dL in the plasma. Among the coagulation factors, its concentration is the highest. The main structural element of a blood clot is it. Three or four days is the plasma half-life. To keep the blood clots together, a concentration of 100 mg/dL is necessary [9].

Three important enzymes—thrombin, plasmin, and factor XIIIa—use fibrinogen as a substrate. It is essential for hemostasis because of its many functional connections. In addition to facilitating platelet aggregation, fibrinogen is the soluble progenitor of insoluble fibrin. Because the fibrin clot activates the fibrinolytic system, the clinical symptoms are determined by the balance between the two processes [10].

For fibrin to be formed, the A alpha and B beta chains of fibrinopeptide A (FPA) and FPB, respectively, are released when thrombin (factor IIa) attaches to fibrinogen. A fibrin clot is formed when the resulting molecule, a fibrin monomer, spontaneously undergoes polymerization. Once fibrin has polymerized, factor XIIIa initiates cross-linking, which fortifies the clot and protects it from mechanical or enzymatic rupture [11].

Monitoring

Clotting tests: When fibrinogen levels are below 100 mg/dL, the tests of activated partial thromboplastin time (aPTT), thrombin time (TT), and prothrombin time (PT) prolongation become detectable. Even though TT is a screening test, it doesn't really tell you anything because there are a lot of common things that can make it last longer. Another effective screening test is reptilase time (RT), which is unaffected by heparin. Because dysfunctional fibrinogen inhibits mixing studies, results from these tests may indicate improvement in afibrinogenemia and hypofibrinogenemia but will be inconclusive in cases of dysfibrinogenemia [12]. A quantitative test that checks the amount of fibrinogen in a blood sample is the fibrinogen antigen test [13].

Fibrinogen activity test: After a specific amount of thrombin is added to plasma, the time it takes for a fibrin clot to form is measured. This test looks for fibrinogen only since it uses thrombin, which isn't present in other coagulation factors. The quantity of active fibrinogen in a sample determines the time needed for clot formation. Decreased fibrinogen levels or malfunctioning fibrinogen can cause the time to increase [14].

Thromboelastography (TEG): This test evaluates the physical characteristics of clot formation using a viscoelastic hemostatic method. This point-of-care test necessitates many calibrations daily and is

quick to administer, making it ideal for comparison and contrast. It is useful for assessing fibrinolysis, platelet function, and coagulation since it assesses the strength and speed of clot formation. The various parameters studied include: [15].

R time (reaction time): It is the amount of time that elapses between the beginning of the test and the first generation of fibrin. The presence of clotting factors is crucial.

K (seconds): It represents the time required to attain a given clot strength (amplitude of 20 mm) and is dependent on fibrinogen.

Alpha angle (degrees): It evaluates the clotting process by measuring the pace of fibrin accumulation and cross-linking. Fibrinogen levels are another factor.

Maximum amplitude (mm): Platelets account for 80% and fibrin for 20% of the total, hence this number represents the ultimate clot strength. It is useful for determining if mechanical disruption or coagulopathy is the cause of the bleeding.

LY30 (%): Half an hour after the amplitude reaches its peak, it is the proportion by which the amplitude decreases. The article discusses fibrinolysis. It is crucial to administer antifibrinolytics within three hours following trauma to reduce mortality, according to the CRASH-2 randomized controlled trial data. Therefore, antifibrinolytic treatment and the proper usage of fibrinogen and cryoprecipitate are guided by early hyper-fibrinolysis diagnosis [16].

Albumin

When it comes to proteins in circulation, albumin is king in plasma. It makes up between three and five grams per deciliter of plasma in healthy humans, which is half of the total protein content. Hepatocytes in the liver produce albumin, which the body quickly eliminates from the body at a rate of 10–15 gm/day. The liver quickly excretes the majority of albumin, therefore there isn't much of it preserved. Serum albumin plays an important role in human health by transporting both endogenous and exogenous ligands, including medications, and by significantly regulating plasma oncotic pressure. One indicator of a patient's nutritional condition that may be assessed using routine serum laboratory tests is serum albumin, which is used in clinical medicine [18].

Patients requiring fluid resuscitation, such as those experiencing hypovolemic shock due to trauma or undergoing large-volume paracentesis, are often given albumin, another colloid fluid. Clinicians can gain insight into patients' liver function or their ability to biosynthesize proteins and components crucial to total body homeostasis by analyzing serum albumin as a laboratory measurement [19].

Function

In addition to transporting ligands, human albumin is the most important modulator of plasma oncotic pressure. Serum albumin can transport both endogenous and exogenous ligands, including bilirubin, ions, fatty acids, and medications. Albumin transports a wide variety of medications, including but not limited to methadone, propranolol, thiopental, furosemide, warfarin, methotrexate, alfentanil, and many more. Hypoalbuminemia, a complication of advanced liver disease, reduces the number of accessible binding sites for medications derived from outside sources [20]. This causes more exogenous medications to remain unbound, which can make the body more sensitive to pharmaceuticals. Serum albumin values below 2.5 g/dL cause this sensitivity to show up in patients [21].

About 30–40% of albumin stays in the bloodstream when it enters the circulation, whereas the rest goes into the interstitial space. The lymphatic system recirculates most proteins that exit the bloodstream. Albumin has a half-life of sixteen hours in the bloodstream. It is believed that albumin's negative charge accounts for the remaining osmotic effect, but its enormous molecular weight is responsible for the bulk of it. The second mechanism enables albumin to entice water and other positively charged molecules into the intravascular space [22].

Serum Albumin as a Laboratory Test

One way to assess a patient's liver function is to take serum albumin, which is a measure of the biosynthetic capacity of the liver. A prothrombin time and/or international normalized ratio are commonly used in conjunction with albumin to provide a more comprehensive evaluation of liver biosynthesis. However, although liver function is normal, serum albumin levels might be normal in chronic liver disease. A review of gastric bypass patients found a weak correlation between liver function tests and pathology. Another possible cause of hypoalbuminemia is a drop in albumin production or a drop in concentration when compared to free fluid. Hepatic failure with ascites, renal failure, and congestive heart failure are all conditions that can lead to the second type of hypoalbuminemia. So, while evaluating and diagnosing the patient, the full clinical background must be taken into account [23].

Low serum albumin levels, often known as hypoalbuminemia, are observed in some malnourished people. Within 24 to 48 hours of beginning fasting, albumin levels drop by one-third, indicating that the benefits of fasting can be felt quickly. Fortunately, this effect is short-lived; the liver's albumin synthesis capabilities are restored within 15 to 30 minutes after replenishment. It is a typical clinical measure for optimizing nutrition and getting ready for surgery because malnutrition has been linked to complications in the postoperative period. The nutritional status of a patient can be assessed using a battery of laboratory tests, albumin included. Additional laboratory tests include retinol-binding protein, transferrin, and pre-albumin. A physical examination of the patient is necessary in addition to these laboratory measurements, as none of them are sufficient on their own. Patients with anasarca and malabsorption can be diagnosed and monitored with hypoalbuminemia. Low albumin levels can occur in a variety of medical situations, including inflammatory illnesses. One possible explanation is that the liver's albumin mRNA production has been downregulated, which has lowered synthesis, increased albumin catabolism, and vascular permeability [24].

Ascites, a condition characterized by fluid accumulation in the peritoneum, can be further evaluated using albumin. Many medical conditions, such as cancer, liver failure, and congestive heart failure, can cause ascites in a patient. To determine the serum ascites-albumin gradient (SAAG), a doctor can drain ascites fluid by a diagnostic paracentesis and then compare the albumin levels in the fluid to the levels in the patient's blood [25,26].

Fibrinogen to Albumin Ratio

Researchers are interested in studying the fibrinogen-to-albumin ratio (FAR) and its association to inflammation and illnesses associated with chronic inflammation [27].

Fibrinogen (FIB) as an Indicator of Inflammation

Although fibrinogen is most associated with its role in coagulation, it is now also known to indicate inflammation. Several inflammatory disorders are commonly linked to elevated fibrinogen levels. The production of additional fibrinogen by the liver can be stimulated by inflammatory mediators, such as cytokines, which are released when inflammation occurs. Inflammation throughout the body might cause FIB levels to increase [27].

Albumin (ALB) as an Anti-Inflammatory and Antioxidant Molecule

In contrast, albumin acts as an antioxidant and has anti-inflammatory characteristics. By attaching to and eliminating dangerous chemicals, such as free radicals, it can aid in reducing the impact of inflammation. Hypoalbuminemia, or abnormally low albumin levels, is a common symptom of chronic inflammatory diseases and may indicate how severe the inflammatory response is [28].

Fibrinogen-to-Albumin Ratio (FAR) as an Inflammatory Marker

The fibrinogen-to-albumin ratio (FAR), which combines fibrinogen and albumin levels, is suggested as a new indicator for evaluating the inflammatory condition. FAR might be a measure of how well the body's pro- and anti-inflammatory systems are working together. An elevated FAR may signal a greater level of inflammation, which in turn may increase the risk or severity of diseases connected to chronic inflammation [29].

The value of FAR is in the more complete picture of inflammation it may give of the body. Although fibrinogen is most recognized as a coagulation marker, it plays an important role in inflammation as well. The liver boosts its production of fibrinogen in response to inflammatory signals, which is why elevated levels of fibrinogen are commonly linked to inflammatory disorders. Albumin, in contrast, is an antioxidant and has anti-inflammatory effects. By attaching to and eliminating dangerous chemicals like free radicals, it helps mitigate inflammation's consequences. Oncology is one of the most prominent fields investigating FAR. Researchers have discovered that FAR can help cancer patients predict their prognosis. More advanced cancer stages and worse survival rates have been linked to elevated FAR levels. A possible tool for evaluating disease progression and directing treatment options, the ratio can indicate the inflammatory condition of cancer patients [30].

There are additional chronic inflammatory illnesses where FAR has demonstrated potential, not just cancer. For instance, diabetic retinopathy and other consequences may be better predicted using FAR in the diabetes population. Because it can show how much inflammation there is in diabetes patients, it can help doctors target their care to those who are most likely to experience difficulties. One other advantage of FAR as a clinical diagnostic is how easily it can be measured via standard blood testing. Healthcare workers looking for new tools for patient assessment may find it appealing due to its simplicity and potential clinical value [31].

The clinical value of FAR is currently being validated in numerous illness scenarios, while the research around it is promising. To find its specificity and sensitivity in various clinical situations and to develop precise cutoff values, more comprehensive research is required. The future of FAR as a biomarker for inflammatory assessment and illness outcome prediction appears bright, according to current studies. An emerging biomarker that has been studied for its possible relevance in a range of medical problems is the fibrinogen-to-albumin ratio (FAR). Its possible relevance in SLE warrants investigation, even though it has not been researched in SLE to the same extent as other medical disorders. But there haven't been many suggestions for possible mechanisms and relationships [27].

Inflammation and Disease Activity

It is well-established that FAR reflects an individual's inflammatory state, and SLE is defined by chronic inflammation. It is possible that a greater level of systemic inflammation is indicated by elevated FAR levels in SLE patients. One possible explanation is that this occurs when the body responds to inflammation by producing acute-phase proteins, such as fibrinogen [32].

Endothelial Dysfunction:

Endothelial dysfunction is a common feature of systemic lupus erythematosus (SLE), further exacerbating inflammation and hypercoagulability. Endothelial damage may be linked to elevated FAR because albumin may have anti-inflammatory effects, and fibrinogen is implicated in clot formation. One possible role of FAR in SLE pathogenesis is as a marker for endothelial damage [33].

Immune Complex Deposition

Symptoms of systemic lupus erythematosus include the development and accumulation of immune complexes in different tissues. Inflammation and tissue damage can be triggered by these combinations. Because immune complex deposition can cause inflammation and changes in albumin and fibrinogen levels, their existence and activity may impact FAR [34].

Renal Involvement

Lupus nephritis (LN) results from systemic lupus erythematosus. Renal damage can occur because of immune complex development and deposit in the renal tissue. Since FAR includes indicators linked to inflammation (fibrinogen) and renal function (albumin), it can indicate the extent to which the kidneys are involved [35].

CONCLUSIONS

Several studies have investigated the fibrinogen to albumin ratio and what it could mean for SLE patients. Endothelial dysfunction is a common feature of systemic lupus erythematosus (SLE), further exacerbating inflammation and hypercoagulability. Endothelial damage may be linked to elevated FAR because albumin may have anti-inflammatory effects, and fibrinogen is implicated in clot formation. One possible role of FAR in SLE pathogenesis is as a marker for endothelial damage.

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