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Innovative Treatment Methods for Brights Anemia Developed in Chronic Renal Failure

Makhmonov Lutfullo Saydullayevich[1], Amerova Dilafruz Abdikhalimovna[2], Kholmatov Baxtiyor Urazovich[3], Rakhmatov Khusniddin Tulkinovich[4]

[1]*PhD, Head of the Department of Hematology Samarkand State Medical University*
[2]*Teacher of the department of Hematology Samarkand State Medical University*
[3]*Head of the department urology Samarkand regional multi-network medical center*
[4]*Ordinator of the department of urology Samarkand regional multi-network medical center*

Abstract: ***The use of innovative treatment methods for Bright's anemia in chronic renal failure has shown promising results in improving patient outcomes. These innovative treatment methods have the potential to revolutionize the management of Bright's anemia in patients with chronic renal failure.***
Keywords: anaemia, kidney, iron therapy, disease, patients, Nosographies, medicine

I. INTRODUCTION

Studies done in America show the prevalence of anaemia in patients with CRF is more than 85%, with 50% of patients in ESRF having a haemoglobin concentration of less than 10-11 g/dL. The National Kidney Foundation in 2000 published new clinical practice guidelines for diabetes and chronic kidney disease. These guidelines included a new target haemoglobin concentration of 11-12 g/dL for patients with CRF, to be achieved through treatment with EPO and iron. The change in the target haemoglobin concentration reflects an understanding of the harmful effects of anaemia on the cardiovascular system, left ventricular hypertrophy, congestive heart failure, and mortality. Although EPO and iron therapy has been effective in increasing haemoglobin concentrations and improving patient well-being, there have been concerns about the possible increased risk of cardiovascular events and death. Thus, it is clear that there is a great need for alternative treatments for anaemia in patients with CRF. EPO and iron therapy only fulfills part of the demands in the new target haemoglobin concentration.

Anaemia is a condition that has been known to be associated with impaired physical activity and well-being. For patients suffering from chronic renal failure (CRF), anaemia has been considered to be a major medical problem. Although this topic applies to all ranges of CRF, this paper will specifically focus on patients in end stage renal failure (ESRF). In these patients, a deficiency in the production of erythropoietin (EPO) by the kidney is the primary cause of anaemia. EPO is a glycoprotein growth factor that is a specific regulator of red blood cell production. In these patients, it is the loss of EPO as opposed to the renal loss of iron or blood, which distinguishes the pathogenesis of anaemia in those with sufficient kidney function from those with CRF.

II. METHODS

Chronic renal failure (CRF) is a form of kidney disease which represents a category of kidney disease in which there is permanent damage to the kidney tissue. There are two forms: 1) chronic renal insufficiency 2) end stage renal disease, and the cause of the disease can range from immunologic diseases, to genetic disorders, to toxicity from drugs. CRF affects over 800,000 individuals in the United States, and this number may be an underestimate. The most common cause of CRF is diabetes mellitus type II and hypertension. Other primary causes of CRF also coincide with the primary causes of Bright's anemia. As the CRF progresses, a person can still be functional and lead a normal life with the help of medications and other treatments, however, as it progresses towards the end stage of renal disease, life becomes debilitated and complicated. At this point, patients with CRF or chronic renal disease are in need of renal replacement therapy. As of today, the most common and effective form of renal replacement therapy is a kidney transplant. But due to the shortage of organs, many of these patients will spend a significant portion of their life being treated with hemodialysis or peritoneal dialysis. These treatments often require lifelong therapy and monitoring, and it is very easy for these patients to succumb to additional disease processes during this time. One of the most common and unfortunate complications associated with dialysis treatment of CRF is the development of anemia. Anemia of chronic disease is a result of a cytokine-mediated reduction in erythropoietin production from the damaged kidney.

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III. RESEARCH AND DISCUSSION

The prevalence of anemia in patients with renal disease was first reported by Dr. Richard Bright in 1827. Reports of anaemia in patients suffering from chronic kidney disease have occurred throughout the medical literature from that time to the present. In fact, in view of the high prevalence of anemia associated with chronic renal failure, I have chosen to confine this discussion to the current literature published in the last 10 years. Anemia is a major reason for the high morbidity and mortality of chronic kidney disease (CKD) patients. The most recent estimates of the prevalence of anemia in patients with CKD come from the NHANES III study. The prevalence of anemia increases as GFR decreases starting at about 8.4% in patients with stage 1 CKD and rising to 53.4% in stage 5 CKD. This translates to the fact that more than 95% of patients with GFR less than 30 are anemic. As the population continues to increase so will the prevalence of anemia associated with CKD; thus creating a long-term need for effective treatments.

Pathologies

The monumental work De Sedibus, et causis morborum por anatomen indagatis libri quinque, or On the Seat and Cause of Diseases Shown by Anatomy, was published in 1761 by the Italian physician Giovanni Battista Morgagni (1682–1771). In the late 18th and early 19th centuries, doctors ardently embraced clinical–pathological correlation as an objective approach to studying illnesses, especially in post-revolutionary Paris but also in other European capitals. Correlating patterns of findings in the sick individual with specific structural abnormalities located deep inside the autopsied body was the goal. Nosographies based only on patient symptoms would be replaced by classifications determined from morbid anatomy.

A key player in this effort, Richard Bright, expanded the complex by including albuminuria, an early laboratory symptom. Based on 24 instances, his first publication from 1827, the Reports of Medical instances, proposed three types of abnormal kidney structure that go hand in hand with albuminuric dropsy. The first was a golden mottling type of softness. "The whole cortical part is converted into a granulated texture" was the second form. The third "is where the kidney appears to rise in numerous projections not much larger than a large pin's head, and is quite rough and scabrous to the touch externally." Each and every portion of the organ is contracting.

Since the 21st-century nephrologist hardly ever sees or handles a newly sick kidney, these three descriptors have little significance for him. Though he appeared to favor three categories, Bright acknowledged that the three forms might merely represent phases of a single process. Thus, from the moment the ailment was initially reported, nobody—not even Bright—held that Bright's illness was a singular, distinct thing.

The word "nephritis" was used for pathological categorization by 1840, while Bright's disease remained the wide category of sickness, even if not all later doctors and pathologists agreed that Bright's disease was inflammatory in character (as did Bright). The history of the succeeding and rival categories is simply too intricate to get into beyond the broadest generalizations. The use of a microscope began to replace tactile examination and casual observation of the renal surface. In 1858, the renowned pathologist and thinker Rudoph Virchow (1821–1902) proposed three conditions: "amyloid degeneration," "parenchymatous nephritis," and "interstitial nephritis." The British physician George Johnson suggested in 1873 to distinguish between three chronic forms of kidney disease: "large white kidney," "red granular kidney," and "lardaceous kidney" (also known as amyloid kidney). The popular publication The Principles and Practice of Medicine by William Osler (1849–1919) supported "acute Bright's disease," "chronic parenchymatous nephritis," and "chronic interstitial nephritis." Amyloid was assigned to a separate pathological group. Die Brightsche Nierenkrankheit, the highly influential 1914 monograph by Franz Volhard (1872–1950) and Theodor Fahr (1877–1945), offered a new, but still trinitarian, classification of diseases into three categories: arteriosclerotic diseases, or "nephroscleroses," inflammatory diseases, or "nephritides," and degenerative diseases, or "nephroses." The final framework was modified by Stanford University's Thomas Addis in the 1920s, and it became rather popular. It was called "hemorrhagic Bright's disease," "degenerative Bright's disease," and “arteriosclerotic Bright's disease”. The fact that an arteriosclerotic category first emerged in the early 20th century is certainly noteworthy as it represents our current way of developing chronic illness (see "Causes" below).

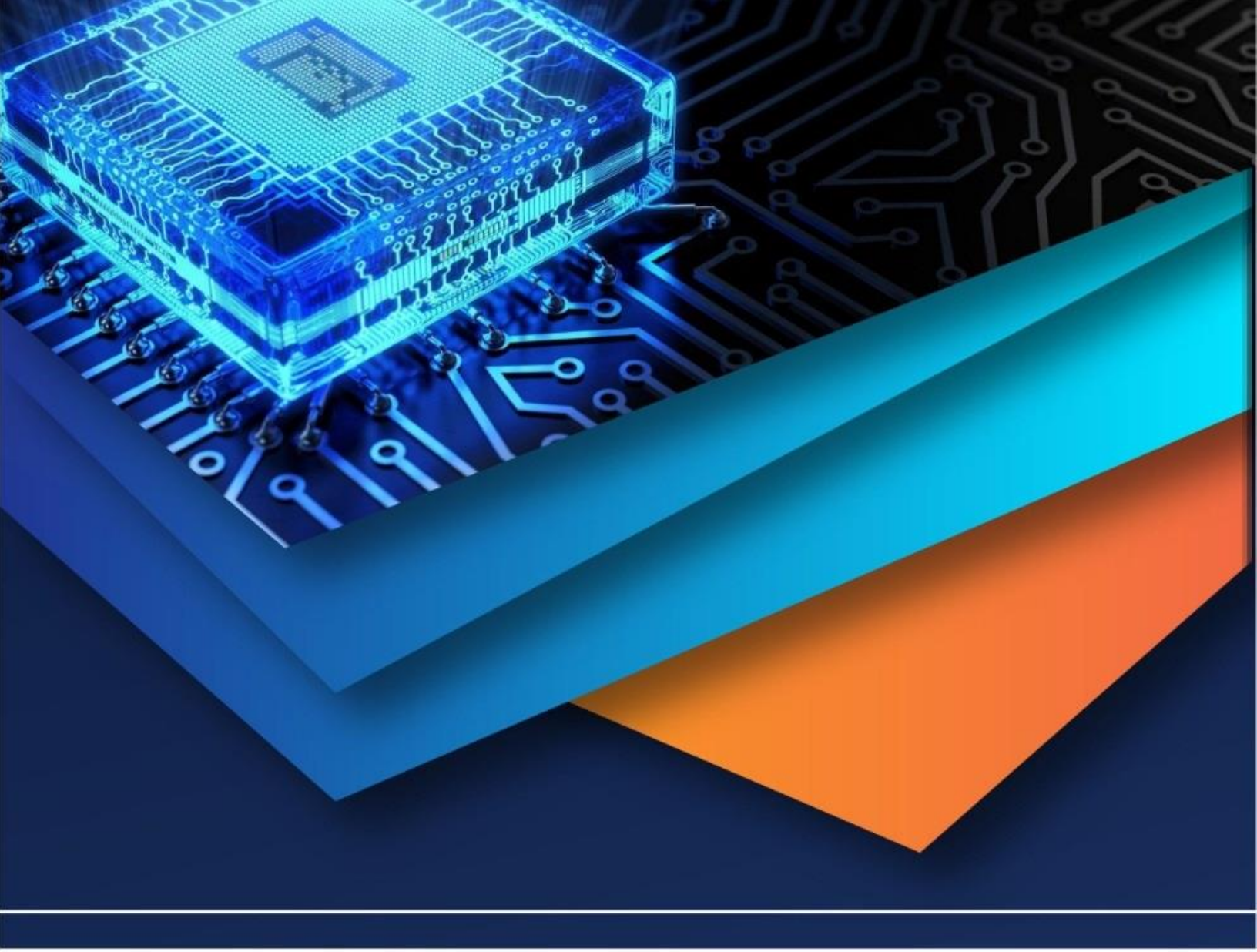
IV. CONCLUSION

These days, it helps to conceive in terms of vascular, tubulointerstitial, or glomerular diseases; nevertheless, when necessary, use biopsy or the identification of marker molecules in blood or urine to obtain a precise, causative diagnosis. The term "chronic" in relation to renal illness has historically been used to describe the diseased state rather than a specific clinical course. For the most part, chronic meant fibrosis or sclerosis. A diagnosis of chronic kidney disease now stresses a homogenous clinical picture and suggests a lack of interest in underlying anatomy. The term "CKD" rejects pathology.

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