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Commentary

Efficacy and Safety of Glucocorticoids in Treating IgA Kidney Disease

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DESCRIPTION

IgA or IgA-based immunoglobulin is diffusely deposited in the glomerular mesangial region in IgA Nephropathy (IgAN), a glomerulonephritis. Berger and Hinglais proposed it as a primary glomerular illness in 1968. At now, it accounts for more than 40% of primary glomerular disorders, making it the most prevalent. The primary factor causing End-Stage Renal Disease is it (ESRD). Before the disease manifests, there are numerous inducements, such as upper respiratory tract infection. The disease has a wide range of clinical symptoms, varying disease durations, and unclear pathophysiology. There are currently not enough efficient therapy options available. Young people are increasingly more likely to develop IgA nephropathy, with a male to female ratio of roughly 2:1. One of the more frequent causes of ESRD (End-Stage Renal Failure) in young people is due to it. The annual incidence rate is rising globally. About 20.00%-30.00% of patients experience renal failure every 10 years, while more than 40.00% do so every 40 years.

Clinical signs, histological alterations, and prognosis of IgA nephropathy differ significantly from one another. The danger of nephropathy progressing may be made worse if the illness cannot be handled in a timely manner. There isn't a specific treatment available right now. Reducing proteinuria, managing blood pressure, delaying the onset of ESRD, and slowing the progression of renal disease are the main therapy objectives. Massive proteinuria, high blood pressure, persistent hematuria, visibly impaired renal function, hyperuricemia, and pathological alterations to the kidneys are currently thought to be clinical signs of a bad prognosis. Since they contain anti-inflammatory, antiallergy, and immunosuppressive properties, glucocorticoids are frequently used to treat immune-mediated inflammatory conditions, such as kidney disease. In clinics, glucocorticoid therapy is frequently used.

Currently, the majority of clinical activity is guided by the clinical practice recommendations of the global organization for improving the prognosis of kidney illnesses (KDIGO). Many debates have, however, arisen as a result of the diversity of treatment plans and prognostic indicators, such as whether glucocorticoid treatment is utilized for IgA renal disease with moderate proteinuria or whether ACEI medications combined with glucocorticoids can treat IgA nephropathy. The main factor affecting the prognosis of IgA nephropathy is proteinuria. To assess the prognosis, clinical trials are typically carried out in a single centre with limited samples. The credibility of the result will be low because of the substantial constraints and the numerous affecting factors.

As a result, we gathered and organized the pertinent research data that had been published both domestically and internationally, overlaid the sample size, used the proteinuria efficacy index to assess the risk of kidney disease progression from various gradients, and investigated the effectiveness and safety of glucocorticoids in the treatment of IgA nephropathy. Due to the many personal variations of the patients, the hormone treatment course varies in the process of IgA nephropathy. There are currently few clinical research on the hormone treatment protocol, and there is still no agreed-upon standard to recommend the ideal hormone treatment protocol. The average usage of hormone in IgA nephropathy patients for 36 months can successfully reduce urine protein excretion, however it is not advantageous to protect renal function. According to a recent study, using hormones for 24 months and monitoring the results for 60 months can significantly lower urine protein excretion, but the other consequences are less clear. The outcomes shown that it may lessen proteinuria and safeguard renal function in IgA nephropathy patients.

The study discovered that hormone therapy for six months can successfully lower proteinuria and safeguard renal function. Although there aren't many clinical studies on the subject, the ones that do all indicate that hormone therapy can reduce proteinuria. However, there is considerable disagreement over the impact of long-term hormone use on kidney health. Therefore, it is advised that clinicians exercise caution when using hormones given the numerous negative effects of hormone therapy. For confirmation, additional large sample tests are required. There is currently no agreed-upon classification system for proteinuria in IgA nephropathy. According to the majority of research, moderate proteinuria is defined as having a 24-hour

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urine protein quantity of more than 3.00 g/d as opposed to less than 1.00 g/d, which is mild proteinuria. The principle of the degree of proteinuria in accordance with the aforementioned standards was also specified with guidelines for the diagnosis and treatment of IgA nephropathy from the international organization for improving the prognosis of kidney disorders (KDIGO). The use of hormones to treat various levels of proteinuria is still debatable, though. Hormone in combination with antiplatelet medication and antihypertensive medication for the treatment of moderate proteinuria caused by IgA nephropathy.

CONCLUSION

The progression of mild proteinuria IgA nephropathy can be effectively stopped by hormone. It can lessen proteinuria levels and stop the decline of renal function. the negative implications of using hormones Hormone therapy is still advised in the majority of instances due to insufficient sample size, a brief follow-up time, and a lack of sufficient side effect data support; nonetheless, large sample testing and follow-up reports are still required. A considerable impact on the management of IgA nephropathy with severe proteinuria (>3.00 g/d). However, it does not negate the fact that hormone application is ineffective for IgA nephropathy patients with proteinuria less than 1.00 g/d. For proteinuria 1.00 g/d, the effect of hormone was worse than that in major proteinuria IgA patients many IgA individuals with proteinuria (2.00-4.50 g/d) hormone treatment. The findings demonstrated that in patients with essentially normal renal function, hormone could successfully reduce proteinuria, stabilize renal function, and stop progression.

IgA nephropathy can be effectively treated with glucocorticoids generally. IgA nephropathy can be effectively treated with hormones at various stages. It is advised that the duration of hormone therapy be appropriately reduced because there is no discernible difference between the efficacy of various treatment courses. More effectively than ACEI medications alone, hormone in combination with ACEI can reduce proteinuria.