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A review of acute or chronic renal failure, common kidney diseases, and herbal plants used for management

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Abstract
Disputes in kidney disease (KD) incidence and progression are commonly believed to be based upon group differences in the prevalence of risk factors for KDs, such as diabetes, hypertension, and obesity. But the prevalence of these comorbidity disorders does not fully explain the increased rate of developments in high-risk populations from chronic kidney disease (CKD). Renal diseases arise as renal function losses eventually resulting in total kidney failure. There is no complete understanding of the processes underlying the causes and development of KDs. The kidneys are a common target organ for the toxicity of different environmental compounds and agents from contact. We must recognize both chemical and pathological pathways and factors that could alter the susceptibility of injury to understand the risk to human health from exposures like these. At present, the test for early disease, predicting disease progression or monitoring therapeutic response is not sufficiently sensitive or specified. Despite several advances in treatment and understanding acute renal failure (ARF) pathogenesis, there persist conflict, ambiguity, and lack of certainty on many aspects in that field. The incidence, etiology, and clinical characteristics of RF are important in promoting preventative strategies and the use of sufficient resources for managing the disease. The article contains brief information about kidney failure, different KDs, their risk factor, prevention and also contains numerous herbs that are effective in the treatment of KDs.

Keywords: renal failure, kidney diseases, environmental agents, prevention, herbal plants

Introduction

The major causes of complications and mortality in patients with end-stage renal disease (ESRD) remain infections and cardiovascular disease (CVD), and it is uncertain how to avoid them [1]. Globally, chronic renal failure (CRF) and ESRD patients have risen significantly. The vast number of patients have hypertension and play a crucial role in the gradual loss of renal function and the extraordinarily high rates of coronary events, the main cause of morbidity and death in patients [2]. The majority of KDs have complicated hereditary or environmental pathogenesis. The decisions on diagnosis and care are usually focused on renal histology, a small number of serological markers, and clinical symptoms of the disease [3]. The effects on the forming kidney of environmentally-based compounds, medications, and physical agents are caused by renal growth and maturation [4]. The rise in the risk of CVD and mortality of patients with KDs has been steadily recognized as a public health issue, and the high cost of care is also being borne out [5].

Four decades earlier, many children with serious ARF or CRF died due to technological limits in renal replacement therapy (RRT) or because dialysis or transplants in many countries were limited [6]. ARF is a common complication of high mortality-related critical illness with different independent effects on the risk of death [7].

In-house patient complications include ARF. When described as a 50% change in baseline serum creatinine, the occurrence in patients who undergo cardiac surgery is between 5 percent and 31 percent.

A significantly lesser proportion of admitted patients (usually between less than 1 and 2 percent) experience extreme ARF to require dialysis [8]. Despite advances in RRT, the mortality rate of ARF patients remains high [9]. In 2010 more than 2 million maintenance dialysis patients were registered in the world, and in 20 years, a rise of 400 percent is expected. This development, which is mainly occurring in developed nations, is motivated by a rise in diabetes worldwide and is too high to be compensated by higher renal transplantation rates [10]. Previous CKD phases are characterized by combining kidney damage and reduced kidney function. A strict framework for estimating CKD prevalence has been given in the National Health and Nutrition Examination Surveys (NHANES) [11]. One such population with a slightly higher risk of CVD in people with CRF. While dialysis is successful in preventing the fatal immediate results of ESRF, deaths between dialysis patients remain high [12]. The number of CRF and ESRF patients is increasingly growing, globally [13]. The documented complication of organ transplantation is chronic renal failure [14]. In the USA there was a significant increase in the number of patients with KDs treated with dialysis and transplantation, from 209000 in 1991 to 472000 in 2004 [11].

The 2015 Global Burden of Disease study reports that CKD directly caused more than 1 million deaths in 2011, a rise of 31.7% relative to 2005 [15].

A meta-analysis of 33 research reports that 10.4% in males and 11.8% in women, with most of the patients from developed nations, was CKD in 2010 [5]. Figure1 shows the mortality and prevalence of KDs in different regions of the globe in 2017.

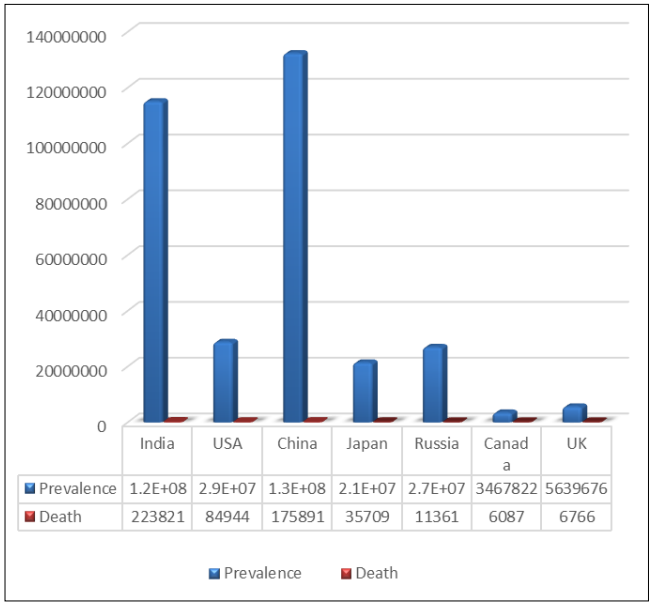

Fig 1: Prevalence and Death from chronic kidney diseases in different countries in 2017 [16].

Difference between Acute and Chronic Renal Failure

Acute Renal Failure

During the air attacks over London, seven decades ago, the current tradition of ARF begun. Epidemiology has since been nearly forgotten, despite thousands of publications covering many different facets of the issue. Different retrospectives and prospective ARF epidemiology trials were also conducted [17].

About 19% of patients with mild sepsis suffer from ARF, 23% with extreme sepsis, and 51% with a septic shock of healthy blood cultures. Smooth and extreme sepsis and septic shock are also linked with a steady rise in acute respiratory distress syndrome [18].

The basic concept of ARF is a slow, persistent reduction in the glomerular filtration rate (GFR), leading to an accumulation of nitrogen waste products and uremic contaminants and in general to azotemia and a drop in urine production [19].

The diagnosis of ARF should be carefully made based on improvements in urinary production, urea nitrogen in the blood, or creatinine content alone [20].

In these conditions, the development rate of metabolic waste is higher than the rates of renal excretion, with serum levels of renal indicators such as urea and creatinine rising. ARF is typically classified into three categories: prerenal, postrenal, and intrinsic [21].

The pathophysiology of ARF due to ischemia also plays a significant role in inflammation. A disorder that occurs after a rapid, temporary decrease in overall or regional blood flow to the renal ischemic ARF. The inflammatory reaction contributes to endothelial activation and damage, increased endothelial cell-leucocyte adhesion, leukocyte interference, and a balance on microvascular blood supply during kidney ischemic injury [22].

Diuretics are linked with an elevated risk of mortality and non-recovery of renal function in critically ill patients with ARF [23].

Figure 2 shows the different types of ARF.

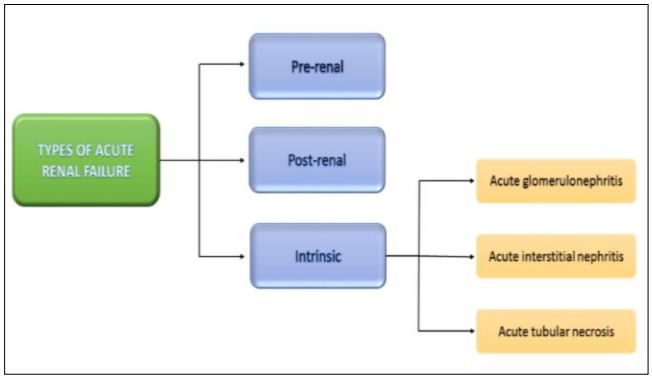

Fig 2: Types of Acute Renal Failure.

Chronic Renal Failure

Patients of CRF and ESRD, globally have risen significantly. The overwhelming majority (up to 80%) of these patients have hypertension that is a key factor in the gradual degradation of kidney function and in the extraordinarily high rates of CVD that are the principal cause of the patient's morbidity and mortality [24]. A persistently irregular GFR characterizes progressive renal failure. There are major differences in the rate of development. There are some prominent morphological traits, such as fibrosis, depletion of native cells, monocyte, and macrophagous infiltration [25]. The gradual and permanent loss of kidney function in CKD. It arises from the decline in renal activity. The diagnosis of GFR is based on a reduction in plasma creatinine concentrations which results in a progressive increase [26]. CRF in coronary and brain circulation associated with the vascular disorder has been well reported in patients with ESRF [27]. CRF is associated with a disease of excess, with an incomplete absorption of nutrients and reduced anabolism of tissues, and/or increased catabolism. While progress in the treatment of side complications such as anemia and metabolic acidosis, chronic anorexia continues to be a clinical concern, especially in children with ESRF and babies [28]. Oxidative stress can indirectly encourage hypertension/atherosclerosis or specifically induce glomerular damage and kidney ischemia in the development of KDs [29]. The obesity in people who are not suffering from asthma, diabetes, or existing renal problems can also be detrimental to the kidneys [30]. Furthermore, low serum levels in patients with severe renal disease are associated with a higher risk of mortality [31]. Non-renal organ transplantation is frequently affected by multifactorial CRD. Pre-transplant renal dysfunction, biopsy hemodynamic kidney insult, other medical products' nephrotoxic effects, dyslipidemia, hypertension, and diabetes mellitus will all aid CRF in non-renal organs. Renal dysfunction after non-renal organ transplantation complicates patient treatment and leads to elevated morbidity and mortality [32].

Risk Factors

Traditionally, CKDs and acute KDs are considered as separate entities with various etiologies [33]. CKD is a risk factor for ACF because of the prevalence of CKD in patients with ACF episodes and epidemiologic data supports this. However, this finding is difficult to read due to the heavy prevalence of co-morbidities, such as age, diabetes, vascular periphery, CVD, and liver disorders, and the difficulty of identifying AKI in the setting of CKDs [34].

AKIs and CKDs are increasingly regarded as closely related and likely to promote one another. The underlying CKD is a strong risk factor for ACF because of its major interaction with AKI, which has both a reduced GFR and elevated proteinuria [35, 36]. Half of all ESRD cases are due to diabetic nephropathy, most of which are driven by Type II diabetes and high blood pressure [37]. Patients diagnosed as having other underlying psychiatric problems, ESRD patients have risk factors that prevent them from experiencing severe or enhanced depressive disorders. Depression in ESRD patients may be secondary to lack of an important role in their job or families, change in physical activity, a decline in cognitive capacity, or a decrease in sex [38]. AKI is a significant and debilitating disease, which has been on the rise in predisposing conditions in recent years. AKI has several risks and is an independent risk factor impacting the rate of mortality. The prognosis of this condition ranges according to the clinical state, comorbidities, disease incidence, and geographical conditions of the patient [39]. AKI is related to increased mortality and long-term risk of developing CKDs. The chronic lack of renal function that often happens over time characterizes CKD. About 8-16% of the world's population are advanced CKD, which has added low quality of life problems, financial pressures, and demand for renal transplant care [40]. AKI is also triggered separately or mixed, as in septic disease (inflammation, and hypoxia), by causes (e.g., radioactive, chemical, immune), and is activated at a certain time. Also, an individual AKI case is increasingly obvious to the reality that a radical CKD may be more predisposed to growth [41]. Powerful complications such as abrupt ascites, open encephalopathy, gastritis, non-obstructive jaundice, and/or bacterial infections contribute to acute outbreaks of AD. AD may advance further to acute-on-chronic liver failure, associated with high short-term mortality [42]. AKD is a rapid (over time) reversible GFR decrease, which occurs either in previously stable renal function or in preexisting CKD [43]. AKD may be present in patients with a pre-existing GFR. AKD is most frequently combined with Rhabdomyolysis due to heat injury. Rhabdomyolysis is a biological and clinical condition, induced by skeletal cell disturbance and release into the interstitial space and plasma of creatine phosphokinase, lactate dehydrogenase, and myoglobin [44]. A greater age, higher preoperative serum creatinine, and diabetes mellitus may independently increase ARF risk, which includes reduced cardiac expulsions and longer durations of cardiopulmonary bypass [45]. Figure 3 represents some major risk factors involved in ARF or CRF.

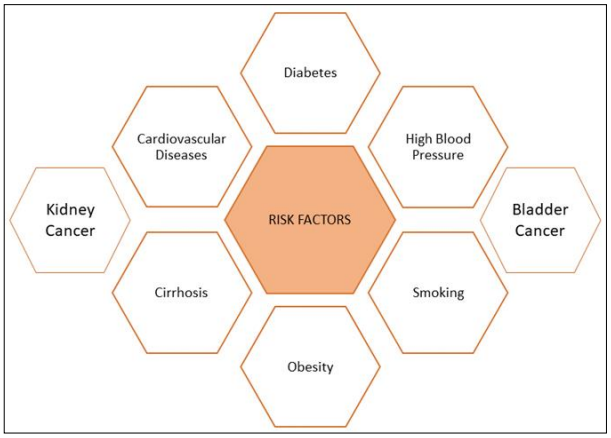

Fig 3: Major risk factors involved in the ARF or CRF.

Common Kidney Diseases

Glomerulonephritis

Glomerulonephritis continues to be one of the world's leading causes of ESRD. It is the cause of ESRD in 23, 32, 42, and 48% of the UK, USA, Australia, and China's renal transplant population respectively [46]. The prevalence has improved with follow-up time and has been identified in 6.0 to 19.4 percent of renal program recipients [47]. The word "glomerulonephritis," while not all synonymous with inflammation, is mostly used simply to cover all forms of glomerular diseases. The histological diagnosis cannot be reliably predicted, but some histological forms of the glomerular disease also have a clinical image, as well. The renal biopsy also is seen in Glomerulonephritis [48]. Any prevalent glomerular diseases do not cause progressive renal dysfunction but cause severe morbidity and a significant health cost. The pathogenesis of glomerular inflammation is affected by both humoral and cell-mediated immune systems. Two fundamental mechanisms have been identified for antibody-mediated glomerular damage. Antibodies will first bind to either a structural glomerulus portion or a substance not intrinsic to the glomerulus due to its physical and chemical characteristics. Second, antigen circulation complexes form, the reticuloendothelial system is free to escape and are stored in the blood vessel [49]. Several risk factors for Glomerulonephritis include younger age at transplantation, male sex, shorter duration of primary renal disease history, low/zero HLA mismatch, steroid avoidance/early steroid withdrawal, shorter total ischemic time, non-use of lymphocyte-depleting induction agents, and, shorter duration of pretransplant dialysis [50].

Kidney stone

Renal stone condition is widespread in a working-age demographic and has a considerable health care burden. The latest literature suggests that influences in lifestyle and diet play a significant role in stone disease risk [51]. Genetic, nutritional, and environmental factors can influence the occurrence and prevalence of kidney stones. About 80 percent, typically calcium oxalate and less so often calcium phosphate, of the stones are composed of calcium salts in most industrialized nations. The other 20 percent of stones are uric acid, struvite or apatite carbonate, cystine, and rare stones [52]. An elevated risk of renal stone is associated with a family history of kidney stones, insulin-resistant states, a history of hypertension, primary hyperparathyroidism, a history of gout, chronic metabolic acidosis, and surgical menopause. The development of kidney stones in postmenopausal women is related to a history of hypertension and a poor diet of magnesium and calcium [53].

Polycystic kidney diseases

Polycystic kidney disease (PKDs), a widespread hereditary cause of child and adult CRF is a diverse class of disorders characterized by fluid-filled cyst aggregation, development, and growth in kidney and other organs sometimes induced ESRF. As autosomal dominant or autosomal recessive traits, the most frequently inherited PKD can be transmitted [54, 55]. The definition of PKDs has been limited by tradition to two conditions: autosomal dominant PKDs and autosomal recessive PKD. Collecting tubules are generalized in recessive polycystic renal disease, while cysts form in the nephron in prevalent polycystic disease, in localized parts [56]. In PKD, however, abnormalities were

also identified in gene expression, cell polarity, fluid secretion, apoptosis, or extracellular matrix, but the cyst forming process remains unidentified [57]. Recent research has contributed to insights into the processes behind the cause and prognosis of these diseases [58].

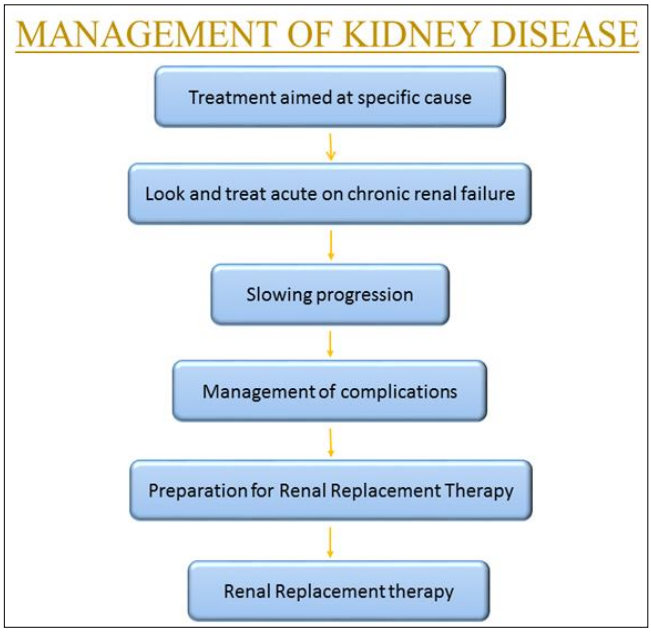

Fig 4: Management of Kidney Diseases.

Herbal Treatment

The use of phytochemicals for the treatment of many diseases has been enormously improved from the past few decades, because of the broader assumption of fewer adverse effects [59]. These days, herbal treatments are becoming more common due to the reduced adverse effects and easy availability of herbal plants [60]. Ethno-medicinal herbs for the management of diseases and their symptoms have historically been used. Presently, ongoing pre-clinical and clinical trials have shown that many have potential benefits in laboratory animals on certain mechanisms associated with impaired renal function [61]. In 35 percent of all patients with ARF in Africa, the use of traditional herbal medicines is involved [62]. Table 1 lists some of the medicinal plants that are effective in the treatment of KDs.

Table 1: List of potential plants that are used to treat kidney diseases [63].

Plant Name	Common Name	Part of Plant	Family
Aerva lanata	Mountain knotgrass	Aerial parts	Amaranthaceae
Alhagi maurorum	Camelthorn	Leaf, Fruit, and Flower	Fabaceae
Carica papaya	Papaya	Seeds	Caricaceae
Ceratonia siliqua	Carob	Pods and leaves	Fabaceae
Cucurbita pepo	Pumpkin	Fruits	Cucurbitaceae
Dichrostachys cinera	Chinese lantern	Roots	Mimosaceae
Kigelia africana	Sausage	Matured fruits	Bignoniaceae
Pedaliusssm murex	Bada gokhru	Fruits	Pedaliaceae
Silybum marianum	Milk thistle	Seeds	Asteraceae
Veronia cinerea	Sahadevi	Aerial parts	Compositae

There are reports that a certain number of herbal Chinese medications, including Astragalus plusAngelica, Ligusticum, Triptolide and Rhubarb, play a significant role in slowing or stopping progression of renal disease [64].

Preventions

A broad variety of current renal disease stresses the need to carry out therapeutic and population-based measures for the prevention of the CKD, the predictions for an elevated occurrence of CKD, and the limits of our existing renal insufficiency therapies Strict blood sugar regulation in diabetes, strict blood pressure control (BP) control, use of ACEIs (Angiotensin Converting enzyme Blockers) and Angiotensin II receptor blockers (ARBs) and protein restriction are the important treatments [65]. The present chronic disease burden represents prior exposures to these risk factors, and current exposure will largely decide the future burden. In fact, in the next decades, we are likely to increase the prevalence of these chronic diseases worldwide [66]. Diuretics, particularly loop diuretics, are typically used in clinical practice to avoid AKI by increasing urine production. For seriously ill patients, furosemide is the most effective loop diuretic and several clinical trials are aimed at testing its efficacy in AKI. In certain experiments, the neutral or deleterious effects of furosemide were found. Several studies have, however, indicated that Furosemide can reduce the need for RRT and lessen the severity of AKI [67]. Keeping sufficient hydration (reversing dehydration), maintaining an adequate mean arterial pressure, and minimizing sensitivity to nephrotoxins are key non-pharmacological techniques for the prevention of ARF [68]. N-acetylcysteine (NAC) has recently been demonstrated to provide defense against contrasted renal dysfunction, a substance that buffers a spectrum of free oxygen-derived radicals and enhances renal vasodilation via endothelium-dependent mechanisms [69]. Several clinical studies have recently reported mild but substantial effectiveness in preventing radiocontrast nephropathy in antioxidants including N-acetylic cysteine (NAC) and ascorbic acid. While it is desirable to delay the rate of weakening renal failure using angiotensin-converting enzyme inhibitors (ACEI) and angiotensin receptor blockers [70].

Conclusion

Science and technology in the 21st century have evolved and improved far beyond our imagination in the fields of living sciences and medical treatment. Kidney failure happens as kidneys lose their working capacity. It is necessary to know whether kidney dysfunction unexpectedly (acute) or over the long term (chronic) evolved to treat kidney failure effectively. Many diseases, illnesses, and drugs will lead to acute and chronic diseases of the kidneys. In different parts of the world, AKD and CKD are big health problems. In both advanced and developing countries, the number of ESRD patients is raising, increasing considerably the need for chronic dialysis and reinforcement. The power of your body to manage blood filtering, extracting water extra from your blood, and helping to regulate blood pressure will be affected by KDs. It may also influence the development of red blood cells and bone health vitamin D metabolism. An ultrasound from the kidneys will also help to decide whether acute or chronic kidney issues. The therapy will depend on the cause of kidney failure. The aim is to recover the function of the

kidney. Fluids and waste must be prevented from building up during the recovery of your kidneys. A kidney doctor named a "nephrologist" evaluates in the majority of cases. Periodic physical exercise and a sensible diet can help avoid KDs are the safest way to prevent them. Prioritize a sound lifestyle. Eat a sensible, nutritious diet. There are certain steps listed in Figure 4 for the management of KDs.

The only realistic alternative for promoting the effectiveness statements for all plants in future studies aimed at the detection of bioactive constituents. All induced ARF models are effectively regulated by medicinal herbs. The most effective chemicals for ARF management are herbals with the hydroxyl group including flavonoids and phenols. Health professionals and patients alike should be cautious but vigilant in the lack of such standardization. For optimum patient treatment and protection, scientific research to explore possibly potential benefits of herbal plants is extremely important.

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Conflicts of Interests

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