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Review Article

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Renal Failure: A Review

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Abstract Advanced Renal Failure or ESRD is one of the most serious and growing issues in the community necessitating the delivery of appropriate services to patients suffering from the disease. Carrying out medical interventions in the early stages of chronic renal failure can delay disease progression and reduce mortality. Results of the Literature review were exported to Endnote. Prior to the formal screening process, a calibration exercise was undertaken to pilot and refine the screening. Formal screening process of titles and abstracts were conducted by two researchers according to the eligibility criteria, and consensus method was used for solving controversies among the two researchers. Chronic Kidney Disease (CKD) is a progressive disease of kidney failure in a period of months to years. Renal failure refers to temporary or permanent kidney damage that results in the loss of normal kidney function. Reduced function of some nephrons causes hypertrophy of other remaining nephrons. The plasma flow in one nephron and the pressure inside the glomeruli increases due to the vasodilatation of the aphrodisiacs. Patients with CKD usually do not show symptoms until GFR decreases down to 15 ml / min. Uremia is a syndrome that affects each organ. Uremic syndrome is probably the result of a series of factors, including retention of molecules, major hormonal deficiencies and metabolic factors, more than the effect of a single uremic poison. Among these toxic compounds, urea can cause fatigue, nausea, vomiting and headache. The product of the decomposition of this material (cyanate) can lead to carbamylation of lipoproteins and peptides and adverse effects which, in turn, disturb several organs.

Keywords Renal failure, CKD, Review

Introduction

Advanced Renal Failure or ESRD is one of the most serious and growing issues in the community necessitating the delivery of appropriate services to patients suffering from the disease. Carrying out medical interventions in the early stages of chronic renal failure can delay disease progression and reduce mortality. The type of treatment chosen is also effective in improving the quality of life and it is suggested that treatments should be considered separately in each patient in order to maximize the benefits of existing interventions and treatments [1].

Renal failure is the disability of the kidneys in the disposal of waste materials, causing the waste to discharge into the body and generating symptoms of variable intensity. Chronic kidney disease (CKD) is defined as progressive and irreversible impairment in renal function. The CKD spectrum varies from proteinuria to serum creatinine, which reflects a decrease in glomerular filtration rate (GFR) and ultimately a complete renal elimination, the final stage of renal disease (ESRD) [2]. According to the Kidney Disease Alliance's Quality Initiative, the CKD (K / DOQI) is

categorized according to GFR, regardless of the cause of the stage, from steps 1 to 5. In steps 1 and 2, where GFR is more than 60 mL per minute for 1.73 m², evidence of kidney damage, such as proteinuria, hematuria, or other disorders in the blood, urine or imaging tests must be available to meet the diagnostic criteria for CKD. Additionally, evidence of kidney damage should persist for at least 3 months in order to differentiate CKD from acute kidney damage (AKI) [3].

Search Strategy

Searches were conducted by two independent researchers in international (PubMed, Web of science, Scopus and Google scholar) and national (SID, Magiran) databases for related studies from the inception of the databases to September 2017 (without time limitation) in English and Persian languages. To ensure literature saturation, the reference lists of included studies or relevant reviews identifed through the search were scanned. The specifc search strategies were created by a Health Sciences Librarian with expertise in systematic review search using the MESH terms and free terms according to the PRESS standard. After the MEDLINE strategy was finalized, it was adapted to search in other databases. Accordingly, PROSPERO was searched for ongoing or recently related completed systematic reviews. The key words used in the search strategy were "Renal failure and Renal" which were combined with Boolean operators including AND, OR, and NOT.

Study Selection

Results of the Literature review were exported to Endnote. Prior to the formal screening process, a calibration exercise was undertaken to pilot and refine the screening. Formal screening process of titles and abstracts were conducted by two researchers according to the eligibility criteria, and consensus method was used for solving controversies among the two researchers. The full text was obtained for all titles that met the inclusion criteria. Additional information was retrieved from the study authors in order to resolve queries regarding the eligibility criteria. The reasons for the exclusion criteria were recorded. Neither of the review authors was blinded to the journal titles, the study authors or institutions.

Quality Assessment

To assess the methodological quality and risk of bias of each included observational study, the Hoy et al. tool was used. This 10-items tool was used to evaluate the quality of studies in two dimensions including external validity (items 1–4 assessed the target population, sampling frame, sampling method and non-response bias minimal) and internal validity (items 5–9 assessed the data collection method, case definition, study instrument, mode of data collection while item 10 assessed bias related to data analysis). The risk of bias was independently evaluated by two researchers. Disagreements were resolved through consensus method.

Definition and Epidemiology

Chronic Kidney Disease (CKD) is a progressive disease of kidney failure in a period of months to years. Renal failure refers to temporary or permanent kidney damage that results in the loss of normal kidney function [4]. The National Cancer Institute of the United States of America has defined chronic renal disease as a kidney injury or glomerular filtration rate of less than 60 ml/min in a body surface area of 1.73 m² for three months or more [5].

Pathophysiology

Reduced function of some nephrons causes hypertrophy of other remaining nephrons. The plasma flow in one nephron and the pressure inside the glomeruli increases due to the vasodilatation of the aphrodisiacs [6]. Hyper-filtration of residual glomeruli preserves GFR in the early stages; however, it later leads to glomerulosclerosis due to intra-thecal glomerular hypertension and glomerular hyper-trubrium, resulting in a decrease in GFR and an increase in proteinuria, which ultimately leads to ESRD. The rate of kidney damage is determined by biopsy of the kidneys [7].

The remaining nephron should be modulated by increasing the filtration rate and secretion level, acid-base balance, water and soluble materials. Patients with CKD, especially in stages 3 to 5, are prone to edema and excessive hypertension, hypercalemia, hyponatremia and azotemia. During progressive kidney disease, sodium balance is maintained by increasing the secretion of sodium by nephrons [8]. Acid excretion is maintained until the end of the CKD, when the GFR reaches less than 15 ml/min. Initially, an increased synthesis of ammonium tubules provides sufficient buffer for hydrogen in the distal nephron. Subsequently, a significant reduction in distal bicarbonate regeneration results in hypercaleric metabolic acidosis. A further decrease in the mass of the nephron causes the retention of organic ions, such as sulfates, which causes metabolic acidosis with an anionic cleft [9].

For decades, it has been thought that when GFR falls below a sensitive level, the CKD tends to advance towards the ESRD, regardless of the initial problem. This suggests that the loss of a significant number of nephrons promotes the greater loss of nephrons as a desirable cycle [10].

Detailed studies have identified a number of interconnected mechanisms that contribute to the progression of CKD, including glomerular hemodynamic responses to neuronal loss, peritonitis, and pro-inflammatory responses, tubular hypertrophy which are associated with increased energy consumption, and metabolic anoxia is associated with the production of oxygen-responsive metabolites [11]. Oxygen inhibitor metabolites have been proposed as a mechanism for tubular injury and interstitial kidney tissue in animal models. Additionally, hyperlipidemia was thought to be involved in progressive renal disease through proliferation and mesangial sclerosis. The activation of renin-angiotensin-aldosterone (RAAS) and increased β-depleting factor (IGF-β) also play a critical role in progressing towards kidney fibrosis. Interventions that lower intraglomerular pressure, such as protein constraints and the use of angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs), slow down the progression of renal disease and confirm the importance of glomerular and RASS hemodynamics in progressive renal disease [12].

Clinical Symptoms

Patients with CKD usually do not show symptoms until GFR decreases down to 15 ml / min. Uremia is a syndrome that affects each organ. Uremic syndrome is probably the result of a series of factors, including retention of molecules, major hormonal deficiencies and metabolic factors, more than the effect of a single uremic poison. Among these toxic compounds, urea can cause fatigue, nausea, vomiting and headache. The product of the decomposition of this material (cyanate) can lead to carbamylation of lipoproteins and peptides and adverse effects which, in turn, disturbseveral organs (13).

Guanidines, exogenous metabolites or endogenous products, increase in renal failure. These side effects can inhibit the activity of 2α-hydroxylase in the kidney, leading to the production of calcitriolacne and secondary hyperparathyroidism. High levels of parathyroid hormone are effective in various uremic symptoms, especially cardiomyopathies and metastatic clusterisation. B2-microglobulin accumulation in patients with ESRD is associated with neuropathy, carpal tunnel syndrome and amyloid infiltration [14].

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