February 19, 2015

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Available at: https://works.bepress.com/kangchuentat/30/
Research article

NEPHROLOGY: ENCOURAGING SELF-STUDY AMONG PATIENTS OF NON-MEDICAL SCIENTIFIC PROFESSIONALS

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Abstract

Medical journals are seldom touched by the scientific professionals that worked in non-related field of study. However, using nephrological journals to solve the health issues independently may be rare without relying on medical doctors and professionals that charge very high cost of medical consultation. For those patients potentially have been affected by renal diseases particularly related to diabetic nephrology, then it is recommended to assist oneself by taking additional initiatives to understand more about the health problem by summarising the content of independent nephrological research as below, not only able assist the patient affected but also helping the nephrology professionals to cure the renal diseases faster with proper care of diets.

Keywords: nephrology, renal, diabetes, angiotensin, nutrient, neuropathy

FORMATION OF RENAL STONE - 3 THEORETICAL POSSIBILITIES

In the nephrological study, one of the aspects that interested the scientists and the public will be the mechanism of kidney stone formation that could affect people randomly among us. Established theories predicted the chemicals composed in the colloid materials, crystals and inhibitor substances from the fluid consumed in daily diets maybe the major contributors to the constituents of the kidney stones, but more scientific evidences are required to justify the theories. [1] In the exploration of inhibitor substances or “crystal poison”, growth rate of stone maybe reduced by the presence of pyrophosphate. [2] Other potential inhibitory substances could be diphosphonate EHDP. [3] Crystal formation of kidney stones due to existing colloid material and crystal nature of the solution consumed are more widely accepted theories, where the material of colloid could be pisolitic calcium oxalate calculi. [4]
DIABETES MELLITUS AND RENAL DISEASE

Although there seemed no direct correlations between diabetes mellitus or high blood glucose level and the fatal renal disease, certain conditions of the kidneys could affect the seriousness of kidney problems. Such relationships are also synonymous to diabetic nephropathy, Kimmelstiel-Wilson lesion, diabetic renal disease or diabetic glomerulosclerosis. [5] Three different mechanisms have been proposed for the linkage between the kidney and diabetes mellitus - (1) arteriolosclerosis and arteriosclerosis; (2) acute and chronic pyelonephritis; (3) diabetic glomerulosclerosis per se. Protein deposits on the endothelial aspects have confirmed the correlations. [6] Initial changes are also found in the capillary basement membrane and mesangium that accumulate matrix and membrane-like material, leading to true nodule formation in lobular centre in nodular diabetic glomerulosclerosis. [7]

IMMUNOLOGICAL RESPONSES IN RENAL SYSTEM

Subsets of T-cells will normally function as suppressor cell to modulate the progression and rate of immune response in renal system although the mechanism of regulation in the quantity and quality of antibody response is not very well understood. There are various factors that determine the glomerular localization of immune complexes, inclusive of the role played by glomerular C3 receptors. [8] The degree of injury caused by the immune complexes of antigen with immunoglobulins to interact with various effector mechanisms, could also be determined. The effector mechanism could be vasoactive amines, the coagulation system, complement and kinin systems, and with cells having receptors for Fc portion of immunoglobulins and to activated C3. [9] Larger sources of immune complexes with IgG and complement, maybe cleared by reticuloendothelial system (RES) rapidly, remaining smaller complexes and those without complement may persist in the circulation longer. [10]

NEPHRONOPHTHISIS AND RENAL MEDULLARY CYSTIC DISEASE – CLINICAL MANIFESTATION

Prior to the detection and diagnosis of nephronophthisis-cystic renal medulla complex, there exist health problems among the patients. [11] In the typical 110 cases of analysis, polyuria, enuresis and polydipsia dominated 80%. This include urinary concentrating defects or diminishing urinary concentration ability, abnormal urinalysis without protein, blood and formed elements in the urine of certain patients, renal salt or sodium wasting where kidney fails to handle sodium or other salts normally, aminoaciduria with the urinary excretion of typical amino acids like proline etc. [12] Frequency of 60% cases are detected with anemia and hypertension hat may lead to weakness and pallor. [13] Another 40% of the reported cases are mainly confined to children and adolescents with disordered bone growth or metabolism and parathyroid gland pathology-related hyperplasia. [14] Vomiting, signs of azotemia, bleeding and convulsions constituted 10% of the abovementioned reported cases.

PROTEINURIA AS INDICATOR OF RENAL DISEASE

It was undeniable that progression of kidney failure is inexorable once a degree of renal damage has occurred, leading to the hypothesis that maladaptive response happens in the remaining nephrons that may cause eventual destruction by common pathogenic mechanism. [15] There are also many clinical observations depicting a strong correlation between the rate of chronic renal failure (CRF) and a quantity of proteinuria, with severity of proteinuria linked to faster rate of progression of CRF and poor renal outcome. Those presenting with nephritic syndrome had a worse prognosis than those presenting with less proteinuria. [16] The rate of progression of CRF could be predicted by the severity of proteinuria, signifying the relationships between proteinuria and the development of renal scarring. Those with decreasing proteinuria symptom possessed higher cumulative renal survival rate than those patients with higher or constant rate of proteinuria. [17]

DIABETIC NEPHROPATHY [18]

One of the major cause of illness and death of diabetes is nephropathy. However, associated cardiovascular disease especially among non-insulin-dependent diabetes mellitus (type II diabetes, NIDDM) patients may cause...
excess mortality of diabetes, besides the end-stage renal disease (ESRD) that lead to proteinuric insulin-dependent diabetes mellitus (type I diabetes, IDDM) and NIDDM as well. [19] In a typical studies done between 1933 and 1952 in a cohort of 1030 IDDM patients, 40% higher relative mortality has been encountered in patients with proteinuria, whereas patients without proteinuria had a significantly lower relative mortality. [20]

MICROALBUMINURIA AND DIABETIC NEPHROPATHY

Microalbuminuria is defined as urinary albumin excretion between 300 mg per 24 hours (or 200 \( \mu \)g / min) and 30 mg per 24 hours (or 20 \( \mu \)g / min) regardless of urine collection method after consensus was obtained on early diabetic nephropathy at a conference. [21] Similar clinical definition of diabetic nephropathy could be applied in insulin-dependent diabetes mellitus (type I, IDDM) and also non-insulin dependent diabetes mellitus (type II, NIDDM), when persistent albuminuria has been the hallmark among diabetic nephropathy patients in concurrent with additional valid criteria such as diabetic retinopathy without laboratory evidence of urinary tract or kidney other than diabetic glomerulosclerosis. [22] In the development of diabetic nephropathy, there exists formation of new glomerular macromolecular pathway and loss of glomerular charge selectivity on the determination of rejection for immunoglobulin G : immunoglobulin GA, that may partially lead to microalbuminuria. [23] Typical clinical experimentation apply filtration fraction to reflect the glomerular pressure because the glomerular hydraulic pressure cannot be measured in humans.

During the 18th century proteinuria was detected as one of the symptom in diabetic patients. Only until year 1836 then it was postulated that albuminuria could be an indication of serious nephron or renal diabetic related disease. [24] This observation has further been justified in current findings where elevated urinary albumin excretion was diagnosed in both IDDM and NIDDM patients. The amount of albumin filtered and the amount reabsorbed by the tubule cells could determine urine albumin excretion. Alteration of size and charge-selective properties of glomerular capillary membrane may change glomerular pressure and flow that affect the diffusive and convecting driving forces for transglomerular passage of protein. [25]

DIFFERENT DIABETIC ASSOCIATED URINARY TRACT COMPLICATIONS WITH TREATMENT

Bladder dysfunction like neurogenic bladders are very common among diabetes patient with typical statistics of 25% cases among non-insulin-dependent diabetes mellitus (type II diabetes, NIDDM) and approximately 26 - 87% among patients of insulin-dependent diabetes mellitus (type I diabetes, IDDM). [26] Typical therapy maybe : (a) possible incision of the internal sphincter; (b) 10 - 50 mg of bethanechol, three times a day in cholinergic therapy; (c) long term intermittent or indwelling catheterization; (d) ensure actual bladder emptiness with repetitive scheduled voiding every 3 to 4 hours. The probability of urinary tract infections among diabetic patients are comparatively higher than non-diabetics, that could be caused by fungus with counts greater than \( 10^4 \) / mL on catheterized specimen in typical primary infection. [27]

POTENTIAL APPLICATION OF HERBAL MEDICINES IN THE TREATMENT OF DIABETIC NEPHROPATHY

As in chronic kidney diseases, diabetic nephropathy was believed to be treated using traditional herbal medicines effectively by applying indigenous systems of healthcare in poorer sections of the society in the developing world, and such alternative treatment has become more popular in the developed countries. [28] Although traditional native medicines are relatively cheap and easily prepared compared to modern medical care that requires adherence to good manufacturing practice (GMP), the nephrotoxic potential of herbal remedies in diabetic nephropathy inclusive is being increasingly recognized. [29] There are various ways where herbal toxicity that may develop in the following situations, further worsening the health of diabetic patients of nephron problems, namely - (a) incorrect identification leading to substitution of an innocuous herb with unknown toxicity; (b) consumption of unknown toxic in herbs etc. [30]
FUNCTIONS OF ANGIOTENSIN II BLOCKERS IN THE PREVENTION OF DIABETIC NEPHROPATHY

There are various chemicals that act as agents to inhibit the rennin-angiotensin system could be used to reduce the risks of diabetic nephropathy and other types of renal diseases especially the type 2 diabetes. [31] In typical Irbesartan and Diabetic Nephropathy Trial (IDNT), each selected patient had received either amlodipine (10 mg daily), irbesartan (300 mg daily) or placebo, with blood pressure to be controlled equal or less than 135 / 85 mm Hg using antihypertensives except angiotensin converting enzyme (ACE)-inhibitors or angiotensin II receptor blockers, and calcium channel blockers. [32] Irbesartan was found to be effective in reducing the risk of a doubling of the serum creatinine concentration by 33%, reducing the risks of end-stage renal disease by 23% with proteinuria reduction by 33%, with lower level of similar risks reduction in amlodipine group and placebo.

GROWTH HORMONE EFFECTS IN DIABETIC-INDUCED RENAL DISEASES

When the number of disaccharide units increased in the blood circulation systems, caused by the assembly of carbohydrate units, this will lead to the thickening and increasing permeability of diabetic basement membrane due to interference in the packing of the collagen helices into fibrils. Such problems may also be due to excess growth hormone in addition to insulin deficiency and glucose excess. [33] When growth hormone is elevated, such insulin antagonist mobilizes free fatty acids and inhibit glucose utilization at the phosphofructokinase step, as growth hormone levels are high with wide fluctuation in uncontrolled diabetics. The growth hormone may also caused hypertrophied kidneys in diabetic human or animal. Together with the effects of insulin deficiency, ultimate basement membrane thickening may occur in the absence of leukocyte and phagocyte. [34]

DIETARY PROTEIN INTAKE AND EFFECTS ON THE RENAL FAILURE PROGRESSION

Animal body’s protein intake may be used to predict the dietary effects in the renal diseases among humans, where the restrictions on protein consumption are beneficial in the renal function preservation. [35] The experimental results suggest in chronological order that semi-defatted fish meat, defatted pork diet, followed by equivalent group of control casein diet and fully defatted fish meat diet, able to ameliorate renal insufficiency progression in Imai rats, with beneficial effects from oils derived from fish rather than protein. In other experiments applying vegetable protein, lower renal plasma flow and a lower glomerular filtration rate were observed among kidney patients. [36] Chronic intake of high amounts of fish protein was related to a lower risk of microalbuminuria in type I diabetic patients. [37]

NUTRIENT ADMINISTRATION FOR DIABETIC-INDUCED ACUTE RENAL FAILURE (ARF)

Oral feedings should be encouraged for tolerable patients with initial 40 g / day of high quality protein is given to meet the daily protein requirement of about 0.6 g / kg body weight per day, that may be increased to 0.8 g / kg per day for blood urea nitrogen (BUN) level of 100 mg / L or lower. [38] Small amount of enteral nutrient maybe helpful to maintain kidney’s function, prevent sepsis development from intestinal bacteria, limit bacterial translocation from gut etc.

CHRONIC KIDNEY DISEASES AND DIET

Care need to be taken in food consumption for patients affected by Chronic Kidney Diseases (CKD), especially for those in the morbid states. [39] Recommendation in the restriction of salt intake is less than 6 g / day, with estimated salt intake (g / day) = urinary sodium (mEq / day) ÷ 17. Restriction of protein intake is suggested around 0.6 - 0.8g/(kg•day), good particularly for stage 3 -5 CKD, with Maroni’s formula applied. Estimated protein intake (g / day) = [ urea nitrogen in urine (g / day) + 0.031 g / kg x body weight (kg)] x 6.25. Total calcium concentration corrected for albumin is proposed to be maintained at 8.4 - 10 mg, with corrected Ca concentration is calculated by special formula. Corrected Ca concentration (mg / dL) = measured Ca concentration (mg / dL) + [ 4 – serum albumin concentration (g / dL) ], when the serum albumin concentration

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is less than 4 g / dL. Obesity is also recommended with BMI being less than 25 kg/m\(^2\), with standard body weight (kg) = [height (m)]\(^2\) x 22. [40]

**CHRONIC RENAL DISEASE AND INORGANIC COMPOUNDS**

Inorganic compounds, mainly derived from phosphorus, aluminum, magnesium etc, may be used to define uremic toxins that have been implicated strongly in the pathogenesis with uremic state alterations, although more attention is being placed on organic compounds. [41] Serum phosphorus will be able to maintain in the normal range of 3.5 - 4.5 mg per deciliter until the glomerular filtration rate falls below 25% of normal even with renal tubular reabsorption of phosphorus occur. [42] Aluminum, as the 5\(^{th}\) most common element in earth crust, found around 4 mg aluminum / liter of municipal water, with systemic aluminum elimination approximately 15 µg / day. [43] Other element like magnesium may be cleared fractionally with increment as the renal function falls progressively.

**CONCLUSION**

Proper independent research among patients affected by renal problems may be possible with self-study, by following routine habit of reading especially among non-medical professionals. This will assist the patients to cure themselves faster and more effectively rather than over-relying on costly medical advice with potential treatment errors not usually aware by persons outside the area of nephrological medical professions.

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