DOWNLOAD PDF NUTRITIONAL MANAGEMENT OF NEPHROTIC SYNDROME

Chapter 1 : Nutrition and Nephrotic Syndrome

The syndrome is characterized by hypoalbuminemia, edema formation, hyperlipidemia, and, less well appreciated, hypovitaminosis D, resulting in hypocalcemia and rarely in iron-deficiency edema. Nutritional Management of Nephrotic Syndrome - Journal of Renal Nutrition.

Print Bananas are a good source of vitamin B6, a nutrient with suspected links to nephrotic syndrome. Nephrotic syndrome NS occurs in approximately 3. Primary etiologies differ between children and adults. In children, the primary etiologies are minimal change disease or focal sclerosing glomerulonephritis, whereas in adults the common etiologies are membranous glomerulonephritis, minimal change disease, or focal sclerosing glomerulonephritis. The most common secondary etiology of NS is diabetes mellitus. The pathophysiology behind NS is thought to be related to either reduced oncotic pressure possibly due to marked proteinuria or increased sodium retention. Pediatric and adult patients with NS often have weight gain due to edema, extremely low visceral protein concentrations due to significant losses in their urine, and hyperlipidemia. Serum albumin concentrations less than 2. Macronutrient recommendations Currently, there are no published clinical guidelines outlining management of NS. Nutritionally, NS patients present a significant challenge. NS patients must ensure adequate intake of B vitamins, as well as of vitamins C and D, iron, and zinc. The pocket guide does not provide specific recommendations for pediatric patients with NS. It should be noted that no large trials have provided evidence to confirm or refute the pocket guide recommendations. Does NS cause B6 deficiency? Recently, a few articles have been published on vitamin deficiencies in NS patients. Isolated case reports and small studies might not be worthy of attention, but in aggregate they bear consideration. Researchers in Japan recently reported on a case involving a year-old girl who presented with edema and a swollen abdomen. Some of her significant biochemistries were a serum albumin of 1. The patient was eventually diagnosed with NS and went into respiratory failure. A second case reported in involved a 6 year old boy who had been diagnosed with NS. During a relapse the boy went through a series of treatments involving methylprednisolone, tacrolimus, and cyclosporine to control his high proteinuria. Following initiation of the tacrolimus, the patient began having convulsions and an MRI was performed. These independent cases of vitamin deficiency in children can be further highlighted as an area of concern by two studies, one in children and one in adults, both with NS. In , researchers published the results of a cross-sectional study comparing the vitamin and homocysteine status of 84 adult patients with NS and 84 age- and sex-matched normal subjects Thromb Res. The study showed that after statistically controlling for confounders, the NS patients had significantly lower concentrations of vitamins B6 9. Finally, an Egyptian study published in examined antioxidant status in children with NS. The study compared healthy children, children in remission, and children who had relapsed. The researchers did not define relapse or provide descriptive data for the groups. The results showed that both groups of children with NS had lower concentrations of albumin, glutathione peroxidase a selenium dependent enzyme , vitamin C, and vitamin A. Additionally, the children in the relapsed group had significantly lower concentrations of serum albumin, glutathione peroxidase, and vitamins A, C, and E compared with both the healthy controls and the children in remission Pediatr Res. Taken together, these data provide a small amount of evidence indicating that patients with NS may be at increased risk of lower vitamin concentrations. Further research needs to be done, but patients with high proteinuria or very low dietary intakes should be carefully monitored to prevent severe deficiencies.

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Chapter 2 : Nephrotic Syndrome in Adults | NIDDK

Nephrotic Syndrome Diet. When diagnosed with nephrotic syndrome, patients cannot help concerning about nutritional management. As we all know, diet is the most basic part in keeping whole body health.

This article has been cited by other articles in PMC. In , the Indian Pediatric Nephrology Group formulated guidelines for management of patients with steroid-sensitive nephrotic syndrome. In view of emerging scientific evidence, it was felt necessary to review the existing recommendations. Following a preliminary meeting in March , a draft statement was prepared and circulated among pediatric nephrologists in the country to arrive at a consensus on the evaluation and management of these patients. To revise and formulate recommendations for management of steroid-sensitive nephrotic syndrome. The need for adequate corticosteroid therapy at the initial episode is emphasized. Guidelines regarding the initial evaluation, indications for renal biopsy and referral to a pediatric nephrologist are updated. It is proposed that patients with frequently relapsing nephrotic syndrome should, at the first instance, be treated with longterm, alternate-day prednisolone. The indications for use of alternative immunosuppressive agents, including levamisole, cyclophosphamide, mycophenolate mofetil, and cyclosporin are outlined. The principles of dietary therapy, management of edema, and prevention and management of complications related to nephrotic syndrome are described. These guidelines, formulated on the basis of current best practice, are aimed to familiarize physicians regarding principles of management of children with steroid-sensitive nephrotic syndrome. Nephrotic syndrome, practice guidelines, recommendations Introduction Nephrotic syndrome is an important chronic disease in children. Most patients have multiple relapses, placing them at risk for steroid toxicity, systemic infections, and other complications. A small proportion of patients who are not steroid sensitive steroid resistant are also at risk for similar complications and renal insufficiency. Most pediatricians would encounter patients with nephrotic syndrome in their practice. They should be familiar with management of these patients and be aware of situations in which referral to a pediatric nephrologist is required. Long-term management of these patients should thereafter be a joint effort between the pediatrician and the pediatric nephrologist. Objectives Guidelines on the management of children with nephrotic syndrome were first formulated by the Indian Pediatric Nephrology Group in Therefore, following a preliminary meeting in New Delhi 7 March , a draft statement was prepared, circulated, and reviewed by pediatric nephrologists across the country Annexure I. The present document reflects the current opinion on management of patients with steroid-sensitive nephrotic syndrome. Recommendations Important revisions in this document are listed in Table 1. Table 1 Important revisions in this document Investigations necessary at initial and subsequent evaluation are described. While updating the literature, the Group endorses the existing guidelines on therapy for the initial episode of nephrotic syndrome. The role of other medications, including mycophenolate mofetil, cyclosporine, and tacrolimus in patients with frequent relapses and steroid dependence is discussed and therapeutic choices clarified. Details on dose and duration of therapy with corticosteroids, when coadministered with other agents are included. Guidelines on immunization, isoniazid prophylaxis, and hypertension updated in conformity with recommendations of the Indian Academy of Pediatrics. Management of complications updated. Precise quantitative assessment of proteinuria, including h urine protein measurement is seldom necessary. Definitions for clarifying the course of nephrotic syndrome are shown in Table 2. Frequent relapses Two or more relapses in initial 6 months or more than three relapses in any 12 months. Steroid dependence Two consecutive relapses when on alternate day steroids or within 14 days of its discontinuation. Open in a separate window Initial Evaluation A detailed evaluation is necessary before starting treatment with corticosteroids. The height, weight, and blood pressure should be recorded. Regular weight record helps to monitor the decrease or increase of edema. Physical examination is done to detect infections and underlying systemic disorder, e. Infections should be treated before starting therapy with corticosteroids. Investigations recommended at the initial episode include urinalysis, complete blood count,

blood levels of albumin, cholesterol, urea, and creatinine. Estimation of blood levels of antistreptolysin O and C3 is required in patients with gross or persistent microscopic hematuria. Appropriate tests are performed, if necessary, for associated conditions e. Urine culture is not necessary unless the patient has clinical features suggestive of a urinary tract infection. Treatment of the Initial Episode Adequate treatment of the initial episode, both in terms of dose and duration of corticosteroids, is important. Evidence from multiple studies suggests that appropriate therapy at the first episode of nephrotic syndrome is an important determinant of the long-term course of the disease. The medication is administered after meals to reduce its gastrointestinal side effects. The use of methylprednisolone, dexamethasone, betamethasone, triamcinolone, or hydrocortisone is not recommended. There is also limited evidence on the efficacy or benefits of therapy with deflazocort for nephrotic syndrome. Treatment regimen Various treatment regimens have been used for the treatment of the initial episode of nephrotic syndrome. The International Study for Kidney Diseases in Children had originally recommended a regimen comprising of 4-weeks each of daily and alternate day steroid therapy, 4 which was used for almost three decades. Controlled studies later suggested that prolongation of initial steroid therapy for 12 weeks or longer is associated with significantly reduced risk for subsequent relapses. However, prolonged treatment with steroids is associated with a higher frequency of adverse events. It further suggests that the benefits of sustained remission and reduction in relapse rates are superior if alternate-day treatment is not stopped abruptly at 12 weeks, but tapered over the next months. It is emphasized that none of the studies included in this analysis was placebo-controlled, most lacked allocation concealment and were not powered to evaluate side effects of prolonged treatment. Other regimens are being examined that reduce the risk of relapse without increased side effects. The benefits and safety of prolonged initial steroid therapy, beyond weeks, require further studies. Treatment of Relapse The patient should be examined for infections, which should be treated before initiating steroid therapy. Appropriate therapy of an infection might rarely result in spontaneous remission, thereby avoiding the need for treatment with corticosteroids. Subsequently, prednisolone is given in a single morning dose of 1. Prolongation of therapy is not necessary for patients with infrequent relapses see below. In case the patient is not in remission despite 2 weeks treatment with daily prednisolone, the treatment is extended for 2 more weeks. Infrequent relapsers Patients who have three or less relapses a year and respond promptly to prednisolone are managed using the aforementioned regimen for each relapse. Such children are at a low risk for developing steroid toxicity. Frequent relapsers and steroid dependence Patients with frequent relapses or steroid dependence should be managed in consultation with a pediatric nephrologist. It is usually not necessary to perform a renal biopsy in these cases. Following treatment of a relapse, prednisolone is gradually tapered to maintain the patient in remission on alternate day dose of 0. A close monitoring of growth and blood pressure, and evaluation for features of steroid toxicity is essential. If the prednisolone threshold, to maintain remission, is higher than 0. Occasionally, it might be possible to discontinue treatment with corticosteroids. The chief side effect of levamisole is leukopenia; flu-like symptoms, liver toxicity, convulsions, and skin rash are rare. The total leukocyte count should be monitored every weeks. Therapy with cyclophosphamide should be instituted preferably following remission of proteinuria. An increased oral fluid intake and frequent voiding prevents the complication of hemorrhagic cystitis; other side effects are alopecia, nausea, and vomiting. The risk of gonadal toxicity is limited with a single 12 weeks course of cyclophosphamide. In view of its toxicity, the use of chlorambucil, unless under close supervision, is not recommended. Prednisolone is coadministered at a dose of 1. Occasionally, treatment with corticosteroids may be discontinued. Estimation of serum creatinine is required every months and a lipid profile annually. A repeat kidney biopsy, to examine for histological evidence of nephrotoxicity, should be done if therapy with calcineurin inhibitors is extended beyond 2 years. Side effects include hyperglycemia, diarrhea, and rarely neurotoxicity headache, seizures. The use of tacrolimus is preferred especially in adolescents, because of lack of cosmetic side effects. Choice of agent The advantages of using these drugs should be balanced against their potential toxicity. There are few studies comparing one agent with another, but evidence for efficacy is strongest for cyclophosphamide and CsA. Levamisole has a modest steroid sparing effect and is a satisfactory

initial choice for patients with frequent relapses or steroid dependence. Treatment with cyclophosphamide is preferred in patients showing: Treatment with CsA or tacrolimus is recommended for patients who continue to show steroid dependence or frequent relapses despite treatment with the above medications. The chief concern with their use is nephrotoxicity, but with careful assessment of renal function, minimizing the maintenance dose and utilizing renal biopsies in those receiving prolonged therapy, this risk can be minimized. Recent case series 14 and a controlled trial 15 support the use of MMF as a steroid sparing agent. The lack of renal, hemodynamic and metabolic toxicity with this agent makes it an attractive alternative to calcineurin inhibitors. In some patients receiving therapy with levamisole, MMF and calcineurin inhibitors, treatment with prednisolone might be tapered and discontinued after months. Some patients who respond to therapy with levamisole, MMF, and calcineurin inhibitors may relapse once these medications are discontinued. Relapses during or following therapy with these agents are treated with prednisolone as described above. Failure of alternative medication If a patient has two or more relapses over 6 months while on treatment with any of the above agents, its replacement with an alternative medication should be considered. A protocol summarizing the management of patients with steroid-sensitive nephrotic syndrome is shown in Fig.

Chapter 3 : Management of Nephrotic Syndrome in Children

Nephrotic syndrome is caused by urinary loss of proteins of intermediate size. Albumin protein is lost in the greatest quantity, but important protein-bound nutrientsâ€"such as iron bound to transferrin, vitamin D bound to vitamin Dâ€"binding protein, copper bound to ceruloplasmin, and zinc bound mostly to albuminâ€"are lost as well.

Description Nephrotic syndrome has a course of remissions and exacerbations that usually lasts for months. Nephrotic syndrome, or nephrosis , is defined by the presence of nephrotic-range proteinuria, edema, hyperlipidemia, and hypoalbuminemia. While nephrotic-range proteinuria in adults is characterized by protein excretion of 3. Pathophysiology In a healthy individual, less than 0. The glomerular capillaries are lined by a fenestrated endothelium that sits on the glomerular basement membrane, which in turn is covered by glomerular epithelium, or podocytes, which envelops the capillaries with cellular extensions called foot processes. In between the foot processes are the filtration slits; these three structuresâ€"the fenestrated endothelium, glomerular basement membrane, and glomerular epitheliumâ€"are the glomerular filtration barrier. Filtration of plasma water and solutes is extracellular and occurs through the endothelial fenestrae and filtration slits. The importance of the podocytes and the filtration slits is shown by genetic diseases: The glomerular structural changes that may cause proteinuria are damage to the endothelial surface, the glomerular basement membrane, or the podocytes; one or more of these mechanisms may be seen in any one type of nephrotic syndrome. Albuminuria alone may occur or, with greater injury, leakage of all plasma proteins ie, proteinuria may take place. There are two current hypotheses for the formation of edema in nephrotic syndrome: Statistics and Incidences Nephrotic syndrome is present in as many as 7 children per , population younger than 9 years of age. The average age of onset is 2. In the United States, the reported annual incidence rate of nephrotic syndrome is cases per , children younger than 16 years. In children younger than 8 years at onset, the ratio of males to females varies from 2: In children, nephrotic syndrome may occur at a rate of 20 cases per million children. Because diabetes is major cause of nephrotic syndrome, American Indians, Hispanics, and African Americans have a higher incidence of nephrotic syndrome than do white persons. There is a male predominance in the occurrence of nephrotic syndrome, as for chronic kidney disease in general. Causes There are two causes of nephrotic syndrome: Secondary causes include systemic diseases such as diabetes mellitus , lupus erythematosus, and amyloidosis. Clinical Manifestations The first sign of nephrotic syndrome in children is usually swelling of the face; this is followed by swelling of the entire body. Edema is the salient feature of nephrotic syndrome and initially develops around the eyes and legs; with time, the edema becomes generalized and may be associated with an increase in weight, the development of ascites, or pleural effusions. Malnutrition may become severe, however, the generalized edema masks the loss of body tissue, causing the child to present a chubby appearance and to double his or her weight. Irritability and loss of appetite. Anorexia, irritability, and loss of appetite develop. These children are generally susceptible to infection , and repeated acute respiratory conditions are the usual pattern. Albumin leaks out due to structural damage, leading to proteinuria. A history of a respiratory tract infection immediately preceding the onset of nephrotic syndrome is frequent. Assessment and Diagnostic Findings In order to establish the presence of nephrotic syndrome, laboratory tests should confirm 1 nephrotic-range proteinuria, 2 hypoalbuminemia, and 3 hyperlipidemia. Therefore, initial laboratory testing should include the following: Serum albumin levels in nephrotic syndrome are generally less than 2. Medical Management The management of nephrotic syndrome is a long process with remissions and recurrence of symptoms common. The general consensus now is daily induction steroid treatment for 6 weeks, followed by alternate-day maintenance therapy for another 6 weeks. Home monitoring of urine protein and fluid status is an important aspect of management; all patients and parents should be trained to monitor first morning urine proteins at home with urine dipstick; urine testing at home is also useful in monitoring response or non-response to steroid treatment. A sodium-restricted diet should be maintained while a patient is edematous and until proteinuria remits; during severe edema, careful

and modest fluid restriction may be appropriate, but the patient must be monitored closely for excessive intravascular volume depletion. A normal activity plan is recommended. Pharmacologic Management Prednisone is the first-line therapy for children with nephrotic syndrome. Other immunosuppressive medications may be useful in those whose symptoms fail to respond to standard corticosteroid therapy or in those who have frequent relapses. All glucocorticoids are effective; however, prednisone or prednisolone is used most commonly; their specific mode of action in nephrotic syndrome is unknown. Diuretics promote excretion of water and electrolytes by the kidneys; these agents are used to treat heart failure or hepatic, renal, or pulmonary disease when sodium and water retention has resulted in edema or ascites. This agent is used to supplement diuresis in patients with edema; it increases oncotic pressure and thereby promotes a fluid shift from interstitial tissues. Nursing Management The nursing management of a child with nephrotic syndrome include the following: Nursing Assessment Assess for the following: Observe for edema when performing physical examination of the child with nephrotic syndrome. Weigh the child and record the abdominal measurements to serve as a baseline. Obtain vital signs, including blood pressure. Note any swelling about the eyes or the ankles and other dependent parts. Inspect the skin for pallor, irritation, or breakdown; examine the scrotal area of the male child for swelling, redness, and irritation. Nursing Diagnoses Based on the assessment data, the major nursing diagnoses are: Excess fluid volume related to fluid accumulation in tissues and third spaces. Risk for imbalanced nutrition: Risk for impaired skin integrity related to edema. Fatigue related to edema and disease process. Risk for infection related to immunosuppression. Deficient knowledge of the caregiver related to disease process, treatment, and home care. Compromised family coping related to care of a child with chronic illness. The major nursing care planning goals for the child with nephrotic syndrome are:

Chapter 4 : Revised guidelines for management of steroid-sensitive nephrotic syndrome

Al-Bander H & Kaysen G A. Ineffectiveness of dietary protein augmentation in the management of the nephrotic syndrome. Pediatric Nephrology () 5; Feehally J, Baker F & Walls J. Dietary Protein Manipulation in Experimental Nephrotic Syndrome.

Biopsy is unnecessary in most children with nephrotic syndrome. A urinalysis with microscopic examination is the single most useful diagnostic test. The oral corticosteroids prednisone and prednisolone form the mainstay of treatment. Counsel parents and patients to expect several relapses during the childhood years. Nephrotic syndrome is one of the more common renal disorders encountered in children in the primary care setting. Most patients have a favorable response to treatment, which can lead to the misconception that it is a minor, benign illness. In reality, the care of children with nephrotic syndrome requires diligence, patience, and long-term follow-up. This review will focus exclusively on children with high-grade proteinuria but no hematuria. Management of adults with nephrotic syndrome differs in many respects. Clinical Features Children of any age can develop nephrotic syndrome, but typically patients are between the ages of 18 months and 8 years. Boys appear to be affected more frequently than girls. Nephrotic syndrome describes a constellation of findings, not a specific disease process. The classic triad of proteinuria, hypoproteinemia, and edema defines the syndrome. Other features, such as hypercholesterolemia, hypertension, or hyponatremia, are often present but are not essential for diagnosis. The vast majority of children with clinical findings of nephrotic syndrome have a histologic diagnosis of minimal-change disease, so named because these children have normal or near-normal biopsy findings on light microscopy. Electron microscopy shows characteristic changes in the glomerular podocyte foot processes and slit diaphragm. The cause of this disease and the exact relationship between the histopathologic and clinical findings remain unclear. Decades of experience have shown that in children with typical presentations, one can usually presume this diagnosis and begin treatment accordingly. Although a reasonable and evidence-based practice, this can lead to a misconception that nephrotic syndrome and minimal-change disease are synonymous. Nephrotic syndrome is a clinical finding associated with many renal diseases, just as a cardiac murmur is a clinical finding associated with many different types of heart disease. A basic physical examination and urinalysis can help differentiate patients into 1 of 5 clinical groupings Table 1. Etiology Minimal-change disease accounts for the majority of uncomplicated nephrotic syndrome cases in children. A small percentage of patients has focal segmental glomerulosclerosis FSGS , and a much smaller percentage has a variety of other diseases. Recent data have indicated an increased incidence of FSGS, although the reasons are unknown. Edema can shift, depending on the position of the body. A supine patient may have marked ascites but no lower-extremity edema; an ambulatory patient may show more prominent leg edema. When evaluating a patient suspected of nephrotic syndrome, always examine for genital edema, an especially uncomfortable condition. The presence of rash, purpura, petechiae, or arthritis suggests a multisystem disease with renal involvement rather than isolated nephrotic syndrome. Renal biopsy Although nephrologists routinely perform renal biopsies in adults with new-onset nephrotic syndrome, the overwhelming prevalence of minimal-change disease and the prognostic value of a therapeutic trial of corticosteroids make biopsy unnecessary in most children with nephrotic syndrome. Patients with evidence of nephritis or other atypical or "red flag" clinical features Table 2 or those who do not respond to therapy may, however, require renal biopsy. In practice, response to corticosteroid therapy is usually more significant than the histologic diagnosis in terms of management and outcome. Patients who improve with corticosteroid therapy have a more favorable prognosis, whereas those who do not remain at a much greater risk for progressive renal disease, regardless of histologic findings. Renal biopsy is also subject to sampling error; since only a few glomeruli are collected, a biopsy specimen may not include any diseased glomeruli in a patient with FSGS, creating a false impression of minimal-change disease. Laboratory testing A urinalysis with microscopic examination is the single most useful diagnostic test. In a

patient with nephrotic syndrome, it would show an elevated protein level without other abnormalities. In a patient with obvious nephrotic syndrome, quantifying urinary protein with a urine protein: Serum albumin concentration will be low? Hypoalbuminemia invariably reduces total serum calcium because of a loss of the albumin-bound fraction. However, the ionized portion of the total calcium may remain normal or near-normal; the ionized calcium atoms are more clinically significant than the albumin-bound portion, because only the ionized calcium is available to cells. A simple formula for calculating the corrected total calcium value is: Obtain an ionized calcium level if true hypocalcemia is a concern. Small elevations in blood urea nitrogen BUN and creatinine often reflect the intravascular volume depletion typical of nephrotic syndrome and may not be clinically important. Cholesterol levels are typically elevated because of a loss of the serum lipoproteins responsible for lipid metabolism. However, serum lipid values are of little use in the initial management, since lipidlowering therapy is seldom indicated in this setting, and the abnormalities will resolve with remission of the nephrotic syndrome. Serologic tests, such as antinuclear antibodies, complement C3 and C4, antistreptolysin O, anti-DNase B, or Streptozyme, should be reserved for patients with findings suggestive of nephritis, which is beyond the scope of this article. In these settings, a brief phone consultation with a nephrologist may be helpful for selecting the initial laboratory evaluation. These tests are not indicated in a patient with uncomplicated nephrotic syndrome without evidence of nephritis. Imaging studies Imaging studies provide little useful information in uncomplicated nephrotic syndrome and are unnecessary in most cases. Renal ultrasound will be normal, because the pathologic changes of nephrotic syndrome occur at a microscopic level. Small pleural effusions are common but seldom affect respiratory function. Imaging studies should be ordered only if clinical findings suggest a significant concern, such as respiratory impairment or congestive heart failure. Treatment The following treatment guidelines apply only to patients with clinical features suggestive of minimal-change disease. Patients believed to have other renal diseases should undergo additional diagnostic workup, usually including renal biopsy, and may require alternative management. The treatment algorithm Figure summarizes the approach to the child with minimal-change disease. The majority of children can be managed as outpatients, but some do require hospitalization Table 3. Steroids The oral corticosteroids prednisone eg, Deltasone, Meticorten, Orasone and prednisolone Table 4 form the mainstay of treatment for minimal-change disease. Lengthening the initial course of treatment appears to increase the interval between relapses. Potential adverse effects of prednisone therapy, in addition to obesity, include acne, hirsutism, striae, emotional lability, hyperactivity, and loss of bone mineral content. Patient and family education and follow-up can help minimize adverse effects, most of which improve or resolve with the discontinuance of prednisone. Nonsteroidal agents Nonsteroidal agents also shown to be effective in minimal-change disease include oral cyclophosphamide Cytoxan , cyclosporine Gengraf, Neoral, Sandimmune , and levamisole Ergamisol. They should be prescribed only under the guidance of a nephrologist who is experienced in their use. Dietary restriction Dietary education is critical. Sodium restriction significantly reduces water retention and edema. Corticosteroids stimulate a voracious appetite, and education on low-fat foods and healthy snacks can help reduce the potential for fatty weight gain during treatment. Dietary protein restriction has no role in the management of nephrotic syndrome and will not reduce proteinuria. Patients taking diuretics require careful follow-up as well as laboratory monitoring. IV albumin infusions followed by furosemide yield only transient improvement in edema. The albumin leaves through the urine almost as quickly as it infuses. In addition, IV albumin can exit the pulmonary capillaries and create or exacerbate pulmonary edema. Therefore, albumin and furosemide infusions should be reserved for rare special situations, in consultation with a physician experienced in the management of this condition. Inducing remission of the primary disease remains the most important long-term strategy for managing edema. Educate the parents Always counsel parents and patients to seek medical attention if the child develops abdominal pain or fever, or appears ill. Complications related to nephrotic syndrome can be severe and even life-threatening, so the physician must evaluate the ill-appearing nephrotic child very carefully for the presence of serious conditions. They should check the urine once a day recording the results on a calendar , using the first-morning specimen,

until the child is in remission. Write a prescription for urine albumin dipsticks and emphasize the importance of using the correct type of dipsticks. Ask the family to bring the dipsticks to their next appointment to verify that they are suitable. If any uncertainty remains as to the accuracy of home testing, ask the family to bring a urine sample to test in the office or laboratory. Complications Nephrotic syndrome, even from minimal-change disease, is not a benign condition. Loss of plasma proteins creates a complex derangement of the coagulation regulatory process. As a result, nephrotic patients are at very high risk for thromboembolic events, including pulmonary embolism and sagittal sinus thrombosis. The role of prophylactic anticoagulant therapy is poorly defined. Current evidence does not support routine use, but in the absence of clear guidelines, some nephrologists use low-molecularweight heparin in selected, very high-risk patients. Similarly, loss of immunologic proteins places nephrotic patients at risk for infectious complications, particularly spontaneous bacterial peritonitis, most frequently caused by Streptococcus pneumoniae. Nephrotic patients with abdominal pain require very careful evaluation for peritonitis. Patients with significant exposure to the varicella virus who have no history of varicella vaccination or disease may require varicella-zoster immunoglobulin. Relapses often occur during an acute infectious illness, such as an upper respiratory infection or otitis media. Most relapses represent the normal course of the disease and not a failure on the part of the parents or the physician. Patients with prolonged courses or multiple relapses may receive unusually large cumulative amounts of prednisone. This can cause linear growth failure, despite marked weight gain or obesity. Persistent nephrosis in poorly controlled patients also results in loss of essential serum growth factors. Therefore, documentation of height, weight, and blood pressure at each visit is extremely important, because this information cannot be recovered later if it is not recorded. Continue counseling on healthy dietary choices and nutritional follow-up at each visit. Refer patients with growth or weight concerns to a nephrologist who can determine whether nonsteroidal alternative therapies can be used. The role of exogenous growth hormone in patients with nephrotic syndrome without renal failure remains undefined and is not a standard therapy. Nonlive vaccines may be given to patients with active nephrotic syndrome and to those who are receiving high-dose immunosuppression. The main theoretical concerns about giving nonlive vaccines to such children are that optimal seroconversion may not occur or that the antigenic stimulus might trigger a relapse of nephrotic syndrome. Practices vary among centers, and very little evidence exists supporting or refuting these concerns. In the real world, this can lead to disturbing immunization delays. The pediatric community in general emphasizes vaccination whenever possible, dismissing minor or unproven excuses for deferring immunizations.

DOWNLOAD PDF NUTRITIONAL MANAGEMENT OF NEPHROTIC SYNDROME

Chapter 5 : Nutritional and Non-nutritional Management of the Nephrotic Syndrome â€" UC Davis

Diet and Nutrition A healthy diet for Nephrotic Syndrome patients consists of low salt, low fat and low cholesterol, with emphasis on fruits and vegetables. NOTE: The amount of protein and fluid a patient with Nephrotic Syndrome should have depends on the patient's current condition, age and weight.

It is characterised by: The syndrome is not a diagnosis of a particular renal disease but it can occur in a number of conditions including glomerulonephritis, diabetes mellitus, systemic lupus erythematosus SLE and amyloidosis. Patients who are not oedematous or hypoalbuminaemic are said to have nephrotic range proteinuria. The mechanism of the proteinuria is complex. It occurs due to structural damage of the glomerular basement membrane, which leads to an increase in the size and number of its pores, allowing passage of more and larger molecules including plasma proteins into the urine. This may lead to reduced plasma albumin levels hypoalbuminaemia and a consequent decreased colloid osmotic pressure. A reduced colloid osmotic pressure in turn causes fluid to be lost from the intravascular space into the surrounding tissues thus lowering blood volume, cardiac output and blood pressure. A reduced plasma oncotic pressure activates plasma renin and aldosterone, leading to salt and water retention oedema. An increase in the hepatic secretion of lipoproteins and coagulation factors also occurs in the nephrotic syndrome. This leads to the clinical abnormalities such as hyperlipidaemia and hypercoagulability. The increased loss of protein can result, directly or indirectly, in excessive losses of several ions and trace elements from the system. Micronutrients such as vitamin D, iron and zinc, which are normally bound to proteins in plasma, are lost in the urine, making it possible for depletion to occur when proteinuria is excessive. Medical management One of the main objectives in treating the nephrotic syndrome is to reduce the oedema using diuretics. These can cause potassium to be excreted or retained in large quantities so patients blood levels should be monitored and dietary potassium intake encouraged or restricted depending on the results. If the oedema is resistant to diuretics, salt-poor albumin is sometimes administered. Dietary management The reasons for altering a nephrotic syndrome patients diet are to help: So how much protein? There has always been a lot of debate over the amount of protein recommended for nephrotic patients. High protein diets were used in an attempt to maintain serum albumin concentration. However, a number of studies have shown that increasing the protein intake merely increases the losses in the urine, and that maintaining a good energy intake is more important for maintaining a positive nitrogen balance. Studies also suggest that a high protein diet: Some studies suggest that the composition of protein in the diet may be as important as its absolute nitrogen content. Energy Energy intake needs to be adequate for correct utilisation of protein. This is essential to maintain the patients nitrogen balance. Nutritional supplements may be recommended to patients with a poor appetite to improve their energy intake. Sodium Oedema formation is one of the most distressing symptoms of the nephrotic syndrome for most patients. Patients can gain as much as 30kg and while they may have the appearance of being well nourished, they may not actually be. Oedema in itself can also lead to a reduced appetite and anorexia. This can further reduce a patients nutritional intake leaving them at risk of malnutrition. In the past, strict sodium restriction played a central role in the management of the oedema associated with nephrotic syndrome. It is also worth noting that a no added salt diet can also help to reduce thirst in those patients who are also advised to limit their fluid intake. Lipids Elevations in serum lipid concentrations are a feature of the nephrotic syndrome. Increased serum concentrations of total cholesterol, low density lipoprotein LDL cholesterol and very low density lipoprotein VLDL cholesterol are the most frequent abnormalities reported. These abnormalities have been shown to reverse once the patients achieve remission. And although the degree of atherosclerotic risk as a consequence of the hyperlipidaemia is uncertain, it seems sensible to advise patients to modify their fat intake by using polyunsaturated and monounsaturated fats and by reducing their intake of saturated fats. Micronutrients The importance of these losses to the clinical manifestations of the nephrotic syndrome is not well established. For this reason the routine administration of a supplement is not recommended. However, it

is felt that if the patient adhere to a moderate protein, no added salt, and low saturated fat diet the extent of the proteinuria and oedema formation should be reduced and the loss of these important micronutrients minimised.

Chapter 6 : Nutrition - Symptom control in nephrotic syndrome

Nutrition and Nephrotic Syndrome Nutritional requirements for a child with nephrotic syndrome. Children with nephrotic syndrome may have trouble regulating their body's water balance.

The loss of different proteins from the body can lead to a variety of complications in people with nephrotic syndrome. Blood clots can form when proteins that normally prevent them are lost through the urine. Blood clots can block the flow of blood and oxygen through a blood vessel. Loss of immunoglobulinsâ€”immune system proteins that help fight disease and infectionâ€”leads to an increased risk of infections. These infections include pneumonia, a lung infection; cellulitis, a skin infection; peritonitis, an abdominal infection; and meningitis, a brain and spine infection. Medications given to treat nephrotic syndrome can also increase the risk of these infections. Urine samples are taken to diagnose people suspected of having nephrotic syndrome. Nephrotic syndrome is diagnosed when large amounts of protein are found in the urine. The blood protein albumin makes up much of the protein that is lost, though many other important proteins are also lost in nephrotic syndrome. The presence of albumin in the urine can be detected with a dipstick test performed on a urine sample. For the test, a nurse or technician places a strip of chemically treated paper, called a dipstick, into the urine. Patches on the dipstick change color when protein is present in urine. A more precise measurement is usually needed to confirm the diagnosis. Either a single urine sample or a hour collection of urine can be sent to a lab for analysis. With the single urine sample, the lab measures both albumin and creatinine , a waste product of normal muscle breakdown. The comparison of the measurements is called a urine albumin-to-creatinine ratio. A urine sample containing more than 30 milligrams of albumin for each gram of creatinine may signal a problem. With a hour collection of urine, the lab measures only the amount of albumin present. The single urine sample is easier to collect than the hour sample and is usually sufficient to confirm diagnosis, though the hour collection may be used in some cases. Once nephrotic syndrome is diagnosed, blood tests are usually needed to check for systemic diseases that may be causing the nephrotic syndrome and to find out how well the kidneys are working overall. Though blood tests can point toward systemic diseases, a kidney biopsy is usually needed to diagnose the specific underlying disease causing the nephrotic syndrome and to determine the best treatment. A kidney biopsy is a procedure that involves taking a piece of kidney tissue for examination with a microscope. Kidney biopsies are performed by a health care provider in a hospital with light sedation and local anesthetic. How is nephrotic syndrome treated? Treating nephrotic syndrome includes addressing the underlying cause as well as taking steps to reduce high blood pressure, edema, high cholesterol, and the risks of infection. Treatment usually includes medications and changes in diet. Medications that lower blood pressure can also significantly slow the progression of kidney disease causing nephrotic syndrome. Two types of blood pressure lowering medications, angiotensin-converting enzyme ACE inhibitors and angiotensin receptor blockers ARBs , have proven effective in slowing the progression of kidney disease by reducing the pressure inside the glomeruli and thereby reducing albuminuria. Many people require two or more medications to control their blood pressure. In addition to an ACE inhibitor or an ARB, a diureticâ€”a medication that aids the kidneys in removing fluid from the bloodâ€”can also be useful in helping to reduce blood pressure as well as edema. Beta blockers, calcium channel blockers, and other blood pressure medications may also be needed. Statin medications may be given to lower cholesterol. People with nephrotic syndrome should receive the pneumococcal vaccine, which helps protect against a bacterium that commonly causes infection, and yearly flu shots. Blood thinning medications are usually only given to people with nephrotic syndrome who develop a blood clot; these medications are not used as a preventive measure. Nephrotic syndrome may go away once the underlying cause has been treated. More information about treating the underlying causes of nephrotic syndrome is provided in the NIDDK health topic, Glomerular Diseases. Eating, Diet, and Nutrition Eating, diet, and nutrition have not been shown to play a role in causing or preventing nephrotic syndrome in adults. For people

who have developed nephrotic syndrome, limiting intake of dietary sodium, often from salt, and fluid may be recommended to help reduce edema. A diet low in saturated fat and cholesterol may also be recommended to help control hyperlipidemia. What are clinical trials, and are they right for you? Clinical trials are part of clinical research and at the heart of all medical advances. Clinical trials look at new ways to prevent, detect, or treat disease. Researchers also use clinical trials to look at other aspects of care, such as improving the quality of life for people with chronic illnesses. Find out if clinical trials are right for you. What clinical trials are open? Clinical trials that are currently open and are recruiting can be viewed at www. References [1] Nephrotic Syndrome. Accessed February 15, The NIDDK translates and disseminates research findings through its clearinghouses and education programs to increase knowledge and understanding about health and disease among patients, health professionals, and the public.

Chapter 7 : Nephrotic Syndrome Nursing Care Planning & Management: Study Guide

Nephrotic syndrome is a common disease in children. Minimalchange disease remains the most frequent cause, but a careful evaluation to exclude other renal conditions is important, particularly to distinguish between isolated nephrotic syndrome and nephrotic syndrome with nephritis. Corticosteroids.

Chapter 8 : Nutritional Management of Nephrotic Syndrome â€" UC Davis

Nephrotic syndrome is a collection of symptoms that indicate kidney damage. Nephrotic syndrome includes the following: Albuminuriaâ€"large amounts of protein in the urine hyperlipidemiaâ€"higher than normal fat and cholesterol levels in the blood edema, or swelling, usually in the legs, feet, or.

Chapter 9 : Nephrotic syndrome(NS) Diet | what to eat and what to not eat in nephrotic syndrome

Nutritional requirements for a child with nephrotic syndrome Children with nephrotic syndrome may have trouble regulating their body's water balance. This can cause fluid retention (also known as edema).