Editorial

Chronische Erkrankungen in der Adoleszenz stellen häufig eine besondere Belastung für die Betroffenen dar. Ursache hierfür ist die Entwicklung vom unselbständigen Kind zum eigenverantwortlichen Erwachsenen. In dieser Phase spielen hormonelle Veränderungen im Rahmen der Pubertät und psychische Änderung bis zur Entwicklung einer eigenständigen Persönlichkeit eine besondere Rolle. In der Konsequenz dieses Geschehens kommt es bei den chronisch kranken Adoleszenten zu einer fortwährenden konfliktbesetzten Auseinandersetzung mit ihrer Erkrankung bis hin zur Verleugnung und Vernachlässigung der Behandlung. Im Extremfall können solche Konflikte in eine individuelle Katastrophe münden, beispielsweise in den Verlust eines Nierentransplantats durch Non-Compliance, evtl. sogar in einen versuchten oder vollendeten Suizid. Daher ist die Beschäftigung mit den medizinischen und psychologischen Problemen dieser Altersphase ganz besonders wichtig. Eine zusätzliche Brisanz ergibt sich im Wechsel der Betreuungssituation, die vom Kindernephrologen auf den internistischen Nephrologen übergeht.

Ziel dieser Publikation ist es, die speziellen Probleme der Adoleszenz bei chronisch niereninsuffizienten und nierentransplantierten Patienten zu beschreiben, zu analysieren und Strategien zu ihrer Vermeidung vorzuschlagen. In Teil I ist das Augenmerk auf die, insbesondere im Hinblick auf Pubertät, gestörtes Körperhöhenwachstum und Körperzusammensetzung sich ändernde somatische Situation gerichtet. Anschließend wird auf die besondere psychische Verfassung chronisch niereninsuffizienter und transplantierter Kinder und Adoleszenten eingegangen und psychologische Interventionen vorgestellt.

Der 2. Teil des Bandes befaßt sich mit der Frage, wie unerwünschte Arzneimittelwirkungen durch neuere immunsuppressive Strategien bei nierentransplantieren Adoleszenten vermieden bzw. abgemildert werden können und damit die Akzeptanz für die Medikation und das Organ erhöht werden kann. Im Einzelnen wird auf Erfahrungen zur Reduktion und Vermeidung von Steroiden, auf Konzepte zur Reduktion von Calcineurininhibitoren und auf die Bedeutung von Mykophenolatmofetil in der Immunsuppression von Kindern und Jugendlichen eingegangen.

Der 3. und letzte Teil des Bandes beschäftigt sich aus pädiatrischer und internistischer Sicht mit möglichen Stategien, die den Übergang vom pädiatrisch zum internistisch betreuten Adoleszenten verbessern können. Hierbei wird neben der Analyse der bestehenden Probleme des Übergangs auf neue Konzepte im Sinne eines Coaching der Jugendlichen und Adoleszenten eingegangen.

Die in diesem Band dargestellten Übersichten sind Ergebnis eines Symposions, das mit freundlicher Unterstützung der Firma Hoffman-La Roche im Sommer 2003 in Nürnberg stattfand. Der vorliegende Band soll Anstöße zum besseren Verständnis der speziellen Situation von chronisch niereninsuffizienten und nierentransplantieren Jugendlichen und Adoleszenten geben und gleichzeitig dazu beitragen, Kommunikationswege in der gemeinsamen Betreuung dieser Patientengruppe zwischen pädiatrischen und internistischen Nephrologen zu verbessern.

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Endocrine Dysregulation in Adolescents with Chronic Renal Failure

Endocrine disorders in adolescents with renal failure commonly increase the difficulties of transition to adulthood. Puberty is frequently delayed and close with an abbreviated growth spurt contributing to late and insufficient growth. In addition several parameters impair the gain of a normal final height such as malnutrition, reduced function of growth hormone and IGF-I receptor and renal osteopathy. Malnutrition and inadequate body shape may be aggravated by the dysregulation of endocrine regulators of appetite and energy consumption such as leptin and ghrelin.
The proper treatment of growth impairment includes the exclusion of causes not associated with renal failure and the consideration of all underlying conditions. Treatment with recombinant human growth hormone has recently been shown to effectively increase final height. In contrast, no successful treatment options have been suggested for the correction of pubertal disorders due to renal failure. However, disorders with primary gonadal insufficiency have to be carefully ruled out. As yet the discovery of endocrine regulators of energy consumption and food intake has not led to therapeutic or diagnostic consequences.
Therefore, adequate psychological care is of utmost importance to help the adolescent with chronic and end-stage renal failure to overcome the additional problems arising from the endocrine disorders in this age.

Key words:
puberty, malnutrition, growth, appetite regulation

Endokrine Störungen bei Jugendlichen mit chronischer Niereninsuffizienz

Endokrine Störungen bei Patienten mit chronischem Nierenversagen erschweren häufig den Übergang in die Betreuung als erwachsener Patient. Die Pubertät ist häufig bei diesen Patienten verzögert. Sie schließt meist mit einem verkürzten Wachstumsschub und einem insgesamt unzureichenden Wachstum. Zusätzlich tragen verschiedene weitere Parameter dazu bei, dass keine normale Endgröße bei den Patienten erreicht wird. Hierzu gehören die Malnutrition, eine reduzierte Funktion von Wachstumshormon und IGF-I-Rezeptor und die renale Osteopathie. Malnutrition und pathologische Körperzusammensetzung werden verstärkt durch eine Dysregulation der endokrinen Regulatoren von Appetit und Energieverbrauch wie z. B. Leptin und Ghrelin.
Die adäquate Behandlung der Wachstumsstörung beinhaltet den Ausschluss von nicht renalen Ursachen. Bei der Behandlung muss dem multifaktoriellen zugrunde liegenden Geschehen Rechnung

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getragen werden. Die Therapie mit rekombinantem humanen Wachstumshormon ist effizient im Hinblick auf eine Verbesserung der Endgröße. Leider existieren derzeit keine adäquaten therapeutischen Möglichkeiten für die Korrektur der Pubertätsstörung bei chronischem Nierenversagen. In jedem Fall müssen primäre gonadale Störungen sorgfältig ausgeschlossen werden. Inwiefern die Entdeckung der endokrinen Regulation von Energieverbrauch und Nahrungszufuhr langfristig zu neuen therapeutischen Möglichkeiten führt ist noch unklar.
Daher ist die psychologische Betreuung von Patienten mit chronischer und terminaler Niereninsuffizienz von entscheidender Bedeutung, um aus der endokrinen Dysregulation resultierende zusätzliche Probleme der Erkrankung zu meistern.

Schlüsselwörter:
Pubertät, Appetitregulation, Malnutrition, Wachstum

Introduction

Puberty and adolescence are highly active endocrine phases in human life. At that stage final height is determined and the development towards a sexually mature person is established. In addition, body composition is frequently influenced and delineates the body shape in adult life. It is well established that an abnormal onset or extent of these developmental stages not only leads to somatic consequences in future life but disturbs the individuals acceptance in his or her peer group (Soliday et al., 2000). This, in turn, may have a negative impact on the patients' coping strategies especially when confronted with a severe physical disease (Furr, 1998).
Patients with chronic or end-stage renal failure frequently present with a delayed and abbreviated pubertal growth spurt (Schaefer et al., 1990). The onset of puberty is on average late by two years (Wühl and Schaefer, 1999). Finally, body shape is frequently altered due to nausea, protein catabolism and potentially frequent surgery.
It is the objective of this review to summarize the current knowledge of alterations in endocrine regulation in young patients with chronic renal failure leading to disturbances in the physical development of these individuals. Secondly, potential therapeutic options will be discussed where available.

Mechanisms of Endocrine Pathophysiology in Chronic Renal Failure

In patients with chronic renal failure different mechanisms lead to an alteration of endocrine responses:
First, *secretion of hormones and the function of their receptors* may be altered by *uremic toxicity*. Uremia can promote an increased release of hormones such as parathyroid hormone (Kuizon and Salusky, 2004). On the other hand the synthesis of hormones may be impaired as shown for growth hormone (Challa et al., 1993). Similarly, the conversion of hormones into their active form may be disturbed as this could be demonstrated for the peripheral conversion of L-thyroxin to triiodthyroidine (Lim et al., 1997). A different site of action is the hormone receptor and its signal transduction. For insulin and growth hormone receptors interaction of receptor function with uremia has been shown (Smith et al., 1982; Schaefer et al., 2001).
Secondly, *decreased renal production of hormones* or essential regulators of hormones may occur in patients with chronic renal failure due to a *reduction in functional renal mass*. This effect is well known for the decrease in erythropoietin and calcitriol concentrations (Fogo and Kon, 2004).
Finally, *renal clearance of several hormones, hormone binding proteins, or hormone fragment is reduced*. This, for instance leads to an increase in the concentration of Insulin-like-growth-factor binding proteins (IGFBPs) thereby reducing the availability of active IGF-I (Tönshoff et al., 1995). Serum concentrations of leptin and ghrelin are increased due to a decrease in renal clearance (Yashimoto et al., 2002; Nüsken et al., in press). On the other hand inactive hormone fragments may interfere with the assays used for the determination of protein concentration and mimic elevated hormone concentrations.

Growth in Adolescent Patients with Chronic Renal Failure

The factors leading to growth failure in very young and in older children with chronic renal failure are quite distinct. However since approximately 40% of total postnatal body height is achieved until the age of 4 years the factors contributing to growth failure in the first years of life contribute considerably to short stature later on.

Pathophysiology

A number of different factors contribute to growth failure in children and adolescents with chronic renal failure (Figure 1). In infancy the major factor leading to failure to thrive and consecutive short stature is the lack of calories (Jones et al., 1982; Kari et al., 2000). This is usually driven by a loss of appetite due to uremia (Ikizler et al., 1995). Therefore adequate calorie replacement has to be insured e.g. by using gastrostomy feeding (Ramage et al., 1999). In contrast, a lack of protein does not appear to contribute to the genesis of growth failure in patients with renal insufficiency.
A different factor contributing to growth failure in particular in the very young patients is the tubular loss of sodium in renal disorders such as bilateral dysplasia, uropathy or reflux nephropathy. The lack of sodium inflicts a decrease in protein synthesis (Chua et al., 1986).
Likewise, the renal loss of bicarbonate and the insufficient excretion of organic acids leading to metabolic acidosis con-

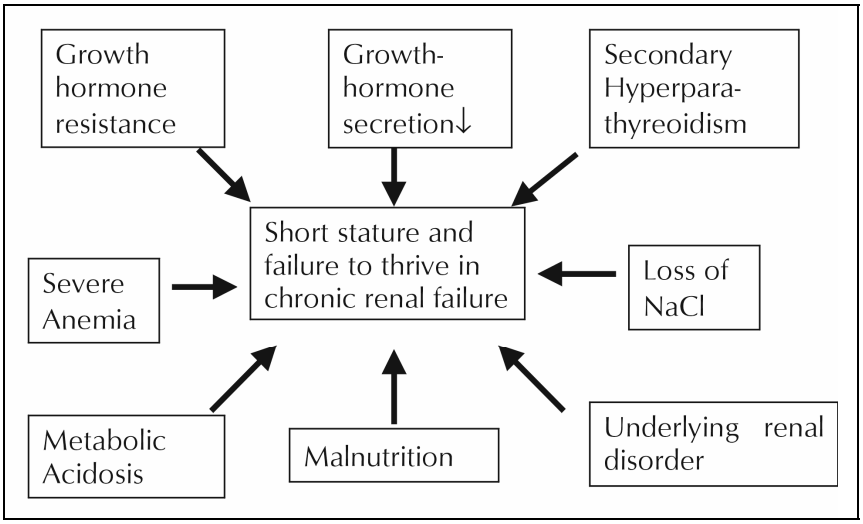

Fig. 1: Factors contributing to failure to thrive and short stature in children and adolescence with chronic renal failure (modified according to Dötsch et al., 2002).

tribute to the poor protein synthesis (May et al., 1986) and alterations of the somatotrop axis (Kuemmerle et al., 1997). In addition, the somatotrop axis is impaired on all levels: Growth hormone secretion is reduced (Challa et al., 1993a) and growth hormone receptor function is impaired leading to a decreased synthesis of IGF-I. Finally, IGF-I receptor function is impaired as a consequence of prolonged uremia (Tsao et al., 2002).

Further factors adding to the failure to grow may be renal osteopathy and severe anemia. Finally, the role of the underlying disorder must be put into consideration. Complex tubulopathies such as Fanconi syndrome leading to losses of electrolytes, protein losses and the side effects of steroid treatment in recurrent nephrotic syndrome may enhance growth failure. Complex syndromes may be associated with renal failure and osteodysplasia increasing the likelihood of short stature (Dötsch et al., 2002).

Diagnosis

The diagnosis of growth failure in older children and adolescents with chronic renal failure needs to address the uremic aspects as well as extrarenal and endocrine factors that may be concealed.

Patient history taking should include parental height on puberty to calculate the patient's target height. Clinical examination includes signs of disproportion (standing height and sitting height), dysmorphisms, and pubertal stages.

An X-ray of the wrist usually reveals a retardation of skeletal age. T3 and T4 concentrations are determined. They may be low due to an increased peripheral metabolism in chronic renal failure (Faber et al., 1983). TSH serum concentrations, however, will help to decide whether the patient is hypothyroid or not. IGFBP-3 is commonly increased in relation to the decreasing glomerular filtration rate (Tönshoff et al., 1995). In uremic patients the synthesis of IGF-I is decreased due to partial growth hormone insensitivity. Nonetheless, the increased concentration of binding proteins leads to normal total IGF-I concentrations. Reduced serum concentrations of IGF-I may, therefore, indicate growth hormone deficiency, malnutrition, or hepatic insufficiency in patients with chronic renal failure.

Treatment

After the exclusion of extrarenal causes of growth failure treatment needs to address the factors contributing to short stature in patients with chronic renal failure. Calories, bicarbonate, sodium chloride, calcitriol, and erythropoietin need to be replaced. Renal transplantation considerably improves growth velocity in children below the age of 4 years as compared to dialysis (Turenne et al., 1997). In contrast, older children and adolescents frequently do no show catch up growth after renal transplantation (Turenne et al., 1997; Tejani et al., 1993). Poor prognostic factors after transplantation are reduced renal function and prolonged treatment with high doses of glucocorticoids (Ettenger, 2001). If, after adequate conservative treatment has been installed, growth velocity remains below the 25th percentile, treatment with recombinant human growth hormone (hGH) has to be considered. Treatment of children and adolescents with chronic renal failure using hGH lead to a height gain of 1.4 standard deviation scores. In contrast, untreated patient lose up to 0.6 standard deviation scores of height (Haffner et al., 2000). The dose of hGH that is administered is twice the dose used in patients with growth hormone deficiency.

Despite the relatively low rate of serious side effects a number of adverse reactions has to be monitored: Among these are benign cerebral hypertension (Dötsch et al., 1998), carpal tunnel syndrome (Saenger, 2000), and pathological glucose tolerance. At any event, a multifactorial approach to growth failure is necessary in children and adolescents with growth failure.

Puberty in Patients with Chronic Renal Failure

In adolescents with chronic renal failure the onset of puberty and the onset of the pubertal growth spurt are frequently delayed (Wühl and Schaefer, 1999; Handelsman et al., 1998). In addition, the length of the pubertal growth spurt is frequently shortened by up to 50% contributing to the loss in final height (Schaefer et al., 1990).

Young adults with chronic renal failure frequently suffer from disturbances of the menstrual cycle as well as from infertility. In boys the number of germ cells is reduced (Burke et al., 1989). The percentage of surviving fetuses in women on dialysis is between 50 and 80% and many of the infants suffer from intrauterine growth retardation (Hou, 2003).

Pathophysiology

The exact mechanisms leading to the onset of puberty are still unknown. The decisive signal, however, leading to the characteristic changes of puberty is the

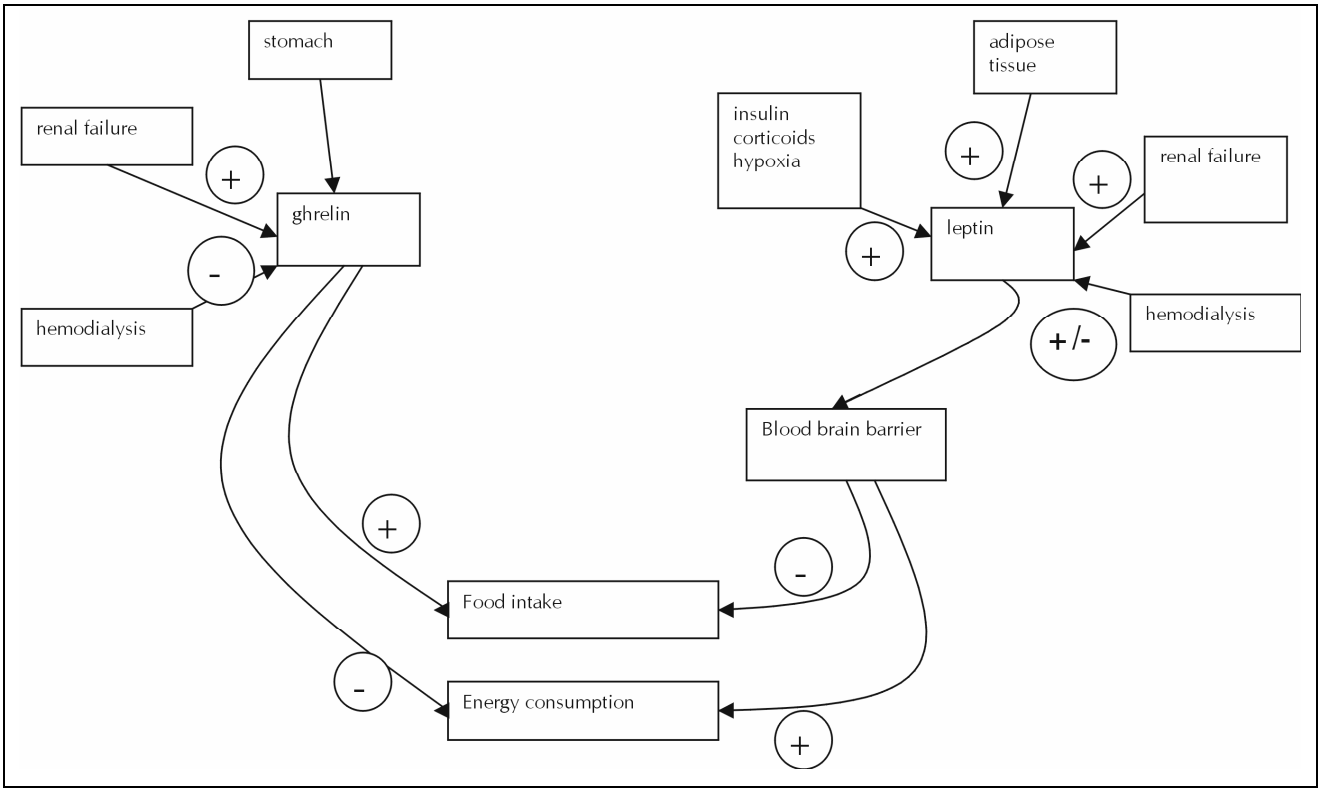

Fig. 2: Interaction of different endocrine factors potentially contributing to loss of appetite and malnutrition in patients with chronic renal failure and on dialysis.

pulsatile secretion of gonadotropin-releasing hormone (GnRH) (Mellon et al., 1990). This signal leads to a pulsatile release of the gonadotropins LH and FSH. The serum concentrations of LH and FSH in patients with chronic renal failure are normal or elevated. However, the secretion profile of LH is pathological displaying a reduced amplitude of the pulses. Consequently, plasma concentrations of LH do not change considerably in patients with end-stage renal failure (Schaefer et al., 1994). An additional factor contributing to the retardation of puberty and to infertility in patients with chronic renal failure is the increase of prolactin concentrations (Gomez et al., 1990). Hyperprolactinemia impairs the function of the GnRH-pulse generator which is reminiscent of patients with prolactinomas (Dötsch et al., 1999). After transplantation a normal pattern of LH-pulses is reestablished (Talbot et al., 1990).

Diagnosis

The diagnosis of retarded puberty is usually made if no visible clinical signs of puberty are present by the age of 14 years in girls and 15 years in boys (Largo et al., 1983a,b). Not always the delay of puberty is caused by the abnormal release pattern of pituitary gonadotropins in patients with chronic renal failure. Apart from the family history concerning the onset of puberty, clinical signs of anorexia nervosa and Turner syndrome have to be looked for in girls whereas genital abnormalities and signs of Kallmann and Klinefelter syndrome should be searched in boys. An ultrasound of uterus and ovaries is essential as well as the determination of the skeletal age by X-ray of the left wrist. Blood tests include estradiol, testosterone, prolactin, LH, and FSH. The interpretation of the basal gonadotropins is considerably facilitated be performing an GnRH-Test which helps to differentiate between hypogonadotropic and hypergonadotropic hypogonadism. Hypergonadotropic hypogonadism is found in patients with primary gonadal failure such as Frasier syndrome, a rare disorder caused by a Wilms Tumor 1-Gene mutation leading to focal sclerosis with consecutive steroid resistant nephrotic syndrome, chronic renal failure and ambiguous genitalia (Benz et al., 2004).

Treatment

The treatment of delayed puberty due to insufficient gonadotropin pulsing in patients with chronic renal failure is not well established. A short-term replacement of testosterone or conjugated estrogens leads to an initiation of puberty but does not result in a gain of final height (Jones et al., 1980). However, hormone replacement should be considered if patients are distressed by the lack of pubertal signs and to avoid a potential loss of peak bone mass.
It is not known yet whether a prolongation of the shortened pubertal growth spurt would result in an increase in final height. One study using Leuprolin in 4 patients with renal transplantation and growth hormone therapy did not show an increase in final height (Alonso et al., 2000).

Appetite Regulation in Adolescents with Chronic Renal Failure

General Aspects

The loss of appetite and lean body mass is common in patients with chronic renal failure (Guarnieri et al., 2003). Several mechanisms are involved including poor food intake, chronic disease, protein catabolism and, more recently, cytokine activation and oxidative stress (Pupim and Ikizler, 2004). In addition the role of endocrine mechanisms involved in the regulation of food intake has been the focus of more recent research activities. In the following the current knowledge of the appetite and energy expenditure regulating hormones leptin and ghrelin will be discussed.

Leptin

Leptin, a 167 amino acid peptide hormone with a molecular weight of 16,7 kDa, is predominantly produced by adipocytes (Halaas et al., 1995). Its effects are mediated through the specific transmembrane receptor OB-Rb (Stephens et al., 1995). The soluble leptin receptor (sOB-R) is an extracellular domain of OB-Rb released to the circulation (Tartaglia et al., 1995). SOB-R is the main leptin binding protein (Lammert et al., 2001).

In adults with chronic renal failure (CRF) (Iida et al., 1996, Stenvinkel et al., 1997) and end stage renal disease (ESRD), serum leptin concentrations are significantly elevated. Leptin is not eliminated by hemodialysis (Merabet et al., 1997, Sharma et al., 1997). The elevation is due to free leptin, leptin bound to sOB-R or the sOB-R concentration in serum is not elevated in adult patients with CRF or ESRD compared to healthy controls (Pecoits-Filho et al., 2002). There is an inverse relationship of leptin- and sOB-R concentrations both in patients with CRF and healthy subjects. In healthy subjects, leptin concentrations decrease during fasting and energy-restriction, independently of body fat changes, ensuring that feeding is triggered before body energy stores have become depleted (Havel, 2001). In CRF and ESRD, hyperleptinemia permanently signals satiety to the hypothalamus. In contrast to hemodialysis, leptin is eliminated by peritoneal dialysis (Landt et al., 1999). There is evidence that adult ESRD patients treated by hemodialysis show higher leptin concentrations than adults treated by CAPD (Zbroch et al., 1999).

In children with CRF and ESRD it has been reported that leptin serum concentrations are significantly elevated. There is a significant, negative correlation between glomerular filtration rate (GFR) and leptin. Leptin serum concentrations are highest in patients with end stage renal disease (Daschner et al., 1998). RIA was used to measure leptin and Western-Blot analysis was used to show that the increase of immunoreactive leptin levels was not due to accumulation of leptin degradation products. In children, we could show that the serum concentration of soluble leptin receptor is not influenced by renal failure. The measurement of total Leptin is in accordance to the FLI, suggesting detection of Leptin by RIA to be adequate in patients with chronic renal failure and end stage renal disease (Nüsken et al., 2003).

In summary, high FLI values in renal failure indicate that Leptin may contribute to loss of appetite and anorexia in these patients However, due to physiological leptin resistance in humans with increasing leptin concentrations the exact contribution to malnutrition in these patients is difficult to estimate (Dötsch et al., 2003).

Ghrelin

Ghrelin, a 28 amino acid peptide hormone with a molecular weight of about 3.2 kDa, is predominantly produced by the stomach (Kojima et al. 1999). It specifically binds to the growth hormone secretagogue receptor (GHS-R) that is localized in hypothalamus and brainstem. Through this receptor it promotes the release of growth hormone by the pituitary gland (Date et al. 2000).

Intracerebroventricular (i.c.v.) and intraperitoneal (i.p.) injections of ghrelin in rodents elevate food intake and induce obesity (Tschöp et al. 2000, Wren et al., 2000). Ghrelin stimulates NPY- and AGRP-neurons in the hypothalamus of rats and prevents the reduction of food intake mediated by leptin (Nakazato et al., 2001, Shintani et al., 2001). Fasting and weight loss result in augmented ghrelin plasma concentrations (Toshinai et al., 2001, Hansen et al., 2002). In healthy children, ghrelin concentrations are inversely correlated with body mass index (BMI) and age (Haqq et al., 2003).

In patients with end-stage renal disease (ESRD), a 3-fold rise in plasma ghrelin levels has been reported. Bilateral nephrectomy in mice causes a marked increase of plasma ghrelin levels. The kidney therefore is an important site for clearance and/or degradation of ghrelin. Ghrelin concentrations decline significantly during hemodialysis (Yoshimoto et al., 2002). Markedly elevated plasma ghrelin levels are found in advanced renal failure and correlate with fat mass, plasma insulin and serum leptin levels. Changes in plasma ghrelin during 12 months of peritoneal dialysis treatment are associated with changes in body composition (Rodriguez Ayala et al., 2004).

In children and adolescents with chronic renal failure ghrelin concentrations are elevated, as well (Nüsken et al., in press). This is consistent with data in adults (Yoshimoto et al., 2002). Hemodialysis results in a decline of serum ghrelin levels and a lower serum concentration thereafter. This fact as well as our finding of a mean ghrelin clearance of up to 45,9 % of the plasma flow and a total cleared plasma amount of 2.46 to 2.93 l/h suggest an effective, potentially long-lasting clearance of ghrelin by hemodialysis. The high clearance of ghrelin either suggests a powerful counterregulation of circulating ghrelin concentration or high tissue concentration. This hormonal dysregulation may provide an additional mechanism to explain malnutrition in hemodialysis patients.

In summary, ghrelin concentrations are influenced by the degree of CRF and the mode of dialysis. In hemodialysis patients, relatively low concentrations of ghrelin may contribute to malnutrition.

Despite the new potential insights of leptin and ghrelin regulation in chronic renal failure it is not entirely obvious to what extent this knowledge will influence potential therapeutic options in the field of loss of appetite and malnutrition.

Conclusion

Taking care of adolescent patients with chronic renal failure not only needs to account for the somatic problems of young adults and the psychological changes of the maturing person. Aggravating factors such as poor growth, delayed puberty and abnormal appetite regulation have to be considered and if possible treated. Only this integrative approach allows for the transition of a mature and responsible young person into the care of the adult nephrologist.

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I. v. d. Osten-Sacken

Psychosoziale Belastungen von Kindern/ Jugendlichen durch Nierenersatztherapie und ihre Verarbeitung

Chronische Erkrankung mit notwendigen lebenserhaltenden Behandlungsmaßnahmen wie Nierenersatztherapie bedeutet Lebensverlängerung und eine erhebliche Verbesserung der Lebensqualität, führt aber durch tiefgreifende Veränderungen in der individuellen Entwicklung des Kindes und des familiären Systems mit existenziell bedrohlichen Erfahrungen zu psychischen und psychosozialen Belastungen, die bei nicht angemessener Verarbeitung erhebliche psychische Störungen mitverursachen können.
Im ersten Teil dieser Arbeit werde ich unter Einbeziehung von Fallbeispielen auf die psychosozialen und psychischen Auswirkungen bei Nierenersatztherapie eingehen, im zweiten Teil den Bewältigungsverlauf bei chronischer Erkrankung als Regelkreis darstellen und im dritten Teil das integrative Konzept psychosozialer Betreuung erläutern.

Schlüsselwörter:
Kinder, Nierenersatztherapie, psychosoziale Belastungen, Bewältigungsstrategien

Psychosocial Burden of Renal Replacement Therapy in Children and Young People – How to Cope with Chronic Disease

Chronic Disease and the ensuing life-sustaining therapies such as renal replacement therapy imply prolongation of life time and significant improvement of quality of life. However, due to dramatic changes in the individual development of the child and within the family systems and due to extremely threatening experiences, this process involves psychical and psycho-social stress which might cause major psychical disorders in case of unappropriate coping.
In the first part of this paper the psycho-social and psychical impact of renal replacement therapy will be discussed on the basis of case reports, in the second part the process of coping in patients with chronic disease will be demonstrated and finally, the third part aims at explaining the integrative concept of psycho-social care.

Key words:
children, renal replacement therapy, psycho-social stress, coping strategies

Klinik für Kinder und Jugendliche der Universität Erlangen

I. Teil

Das Bild, das Kinder und Jugendliche von sich haben, wird ja maßgeblich von ihrem vitalen Körpererleben und ihrer Leistungsfähigkeit bestimmt.
Appetitlosigkeit, Übelkeit, Erbrechen, Schmerzen, Kontrollverlust über körperliche Funktionen, Diätflüssigkeitsrestriktion, Infektionen, medikamentöse Nebenwirkungen, eine verzögerte körperliche und sexuelle Entwicklung, Mehrfachtransplantationen führen zur Beeintächtigung der körperlichen Unversehrtheit mit Veränderung des Selbstbildes (1,2).

Hierzu einige Beispiele:

Fallgeschichte:

Anna, 6 Jahre, wurde nach einem hämolytisch-urämischen Syndrom peritonealdialysepflichtig. Ständige Peritonitiden komplizierten den Verlauf.
Anna war ein unkompliziertes, fröhliches, altersgerecht entwickeltes Mädchen, das mit Begeisterung malte und über ein gesundes Körperbild verfügte (Abb. 1, 2).
Sie hatte Vertrauen zu ihrem Arzt, erlebte ihn als wachsam wie einen Hund und fürsorglich wie eine Schwester (Abb. 3).
Vor einer schweren körperlichen Krise malte sie folgende Bilder, die ihre Mutter entsetzten (Abb. 4, 5).
Diese verzerrten Gesichter sind meines Erachtens Ausdruck einer schweren, existenziellen körperlichen und psychischen Bedrohung. Anna erlebte sich vergiftet entweder über das Gehirn oder über den Bauch. (Bei der OP wurde eine Peritonitis bestätigt.)
Nach Gesundung fragte sie die Mutter: „ Weißt du, was hinter der Welt ist“, als Hinweis, dass sie sich dem Tode nahe gefühlt hat. Nach Genesung war ihre Vitalität und damit ihr positives Selbstbild schnell wiedergewonnen. Angesprochen, auf Wunsch der Mutter, auf die verzerrt gemalten Gesichter, also auf ihre Lebensbedrohung, wehrte sie ein Gespräch darüber vehement ab. Sie wollte sich mit ihrer Vergangenheit nicht mehr konfrontieren, wollte nur noch im Hier und Jetzt leben.
Ich gehe später im Rahmen des Bewältigungsverlaufes auf diesen Krankheitsverlauf Annas nochmals ein.

Abbildung 1

Abbildung 2

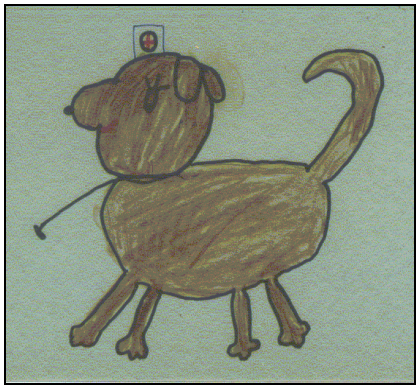

Abbildung 3

Abbildung 4

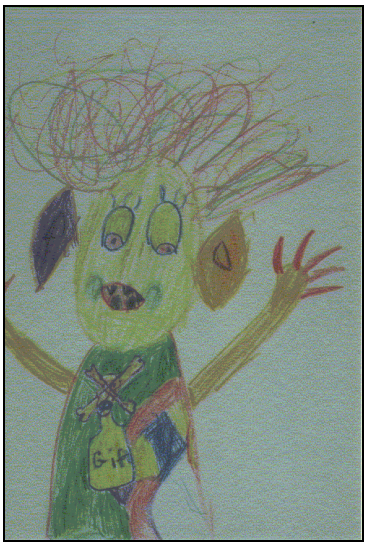

Abbildung 5