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Malnutrition and Catch-Up Growth during Childhood and Puberty

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This chapter reviews important recent papers related to catch-up growth. Catch-up growth is generally defined as body growth that (1) is more rapid than normal for age and (2) follows a period of growth inhibition. As can be seen by the studies reviewed in this chapter, catch-up growth can occur after either pre- or postnatal growth inhibition, can show a variety of temporal patterns, and can be either complete (yielding a normal adult body size) or incomplete. These studies explore the implications of catchup growth not only for adult body size but also for pubertal timing, bone strength, and the risk of metabolic syndrome.

Patterns of catch-up growth

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J Pediatr 2013; 162: 415–420

Summary

This paper reviews the possible mechanisms, the precipitating conditions, and especially the temporal patterns of catch-up growth in children. There are two principal hypotheses to explain catch-up growth – central nervous system sensing of expected body size and intrinsic capacity of the growth plate. Two temporal patterns of catch-up growth that were introduced by Tanner were discussed. The type A pattern is a transient increase in growth velocity after cessation of the growth restriction, followed by normal growth velocity when the original growth curve is achieved. The type B pattern shows a slightly greater growth velocity than expected for chronological age but a normal growth velocity for bone age. This review article shows evidence that type A catch-up growth is seen in some children with

hypothyroidism and with growth hormone deficiency, especially on higher doses of growth hormone. Type B catch-up growth has been demonstrated in a prospective study of celiac disease. The authors also propose an intermediate pattern of catch-up growth, which they term, 'type AB' which shows an increased growth velocity initially, followed by a stabilization of height SDS for years, and a delayed pubertal growth spurt, which brings the individual closer to target height. The authors indicate that this pattern is seen in some children with celiac disease, hypothyroidism, GH deficiency, and children born prematurely.

Comments

Catch-up in bone acquisition in young adult men with late normal puberty

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J Bone Miner Res 2012; 27: 2198–2207

Background

Sex steroids at the time of puberty have a positive effect on bone density. In females, late menarche has been associated with lower BMD persisting into adulthood. The long-term effects of pubertal timing on the skeleton of young men has not been well established. Malnutrition and Catch-Up Growth during Childhood and Puberty 91

Methods

Subjects were from a population-based cohort from the Gothenburg Osteoporosis and Obesity Study. Areal bone mineral density (aBMD), bone mineral content (BMC), volumetric bone mineral density (vBMD) and cortical bone size were measured in 501 men between 19 and 24 years using dual-energy x-ray absorptiometry (DXA) and peripheral quantitative CT scan (pQCT) at baseline and at follow-up. Detailed growth and weight charts were used to calculate age at peak height velocity (PHV).

Results

Subjects were divided into three groups, those with early, middle and late puberty, according to age at PHV. There were no significant differences in age, calcium intake, or smoking between the groups. Age at PHV was a strong positive predictor of gain in bone mass. Thus, the group with late puberty gained markedly more in aBMD and BMC at the total body, radius, and lumbar spine, and lost less at the femoral neck than the group with early puberty. Age at PHV was also an independent positive predictor of the increase in cortical thickness and periosteal circumference of the radius, measured by pQCT. At age 24 years, no significant differences in aBMD or BMC of the lumbar spine, femoral neck, or total body were observed. pQCT measurements of the radius at follow-up also demonstrated no significant differences in bone size although cortical and trabecular vBMD were lower in

men with late versus early puberty. Age at PHV was not associated with distal forearm fracture prevalence.

Conclusion

Late puberty in males was associated with a substantial catch-up in bone density and in bone thickness by young adulthood, leaving little or no deficits in the parameters studied.

Comments

The effects of pubertal timing on adult bone density are difficult to ascertain in human studies. In women, late puberty is associated with lower bone density persisting into adulthood. However, this association may not represent a cause-and-effect relationship between pubertal timing and adult BMD because the difference in BMD actually precedes the onset of puberty, suggesting that the later puberty and the persistent low BMD may both reflect genetic/nutritional/body mass effects [2]. In males, it is not clear whether pubertal timing is associated with persistent differences in BMD, or, if there is an association, whether there is a direct causal relationship. In the study reviewed here, delayed puberty was associated with catchup in aBMD, BMC and vBMD. One could argue that these increases in bone density should not be considered catch-up *growth*. Trabecular bone formation is a process that is not restricted to childhood but rather persists into adulthood, setting it apart from growth of many other structures. However, increasing bone width, which involves periosteal bone formation, is part of childhood growth, occurring rapidly during early years and slowing as adulthood approaches. One interesting finding from this study is that catch-up in the periosteal circumference of the radius also occurred. Thus, this study demonstrates that, in children with late puberty, catch-up growth occurs not only in linear bone growth (which determines height) but also in cross-sectional bone growth (which affects bone strength). Whether catch-up in bone width requires subsequent androgen exposure (which stimulates periosteal bone formation) is not known. Similarly, the mechanism of catch-up growth in bone width is unknown. This study also argues against the widely stated notion that there is a 'golden window of opportunity' to lay down bone during adolescence. Contrary to this notion, when exposure to sex steroids occurs on the later side of the normal range, catch-up occurs, ultimately resulting in a skeleton that is normal, or nearly so.

Is early puberty triggered by catch-up growth following undernutrition?

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Int J Environ Res Public Health 2012; 9: 1791–1809

Summary

This paper reviews catch-up growth after undernutrition and the relationship between catch-up growth and early pubertal development. Catch-up growth can occur following intrauterine growth restriction or after postnatal undernutrition (e.g. Crohn's disease, celiac disease or eating disorders) as well as after combined fetal-postnatal undernutrition when the subsequent nutritional intake is adequate. Catch-up growth may be associated with earlier

timing of puberty after intrauterine growth retardation and after combined fetal-postnatal undernutrition. The authors state that catch-up growth following undernutrition that occurs only postnatally has not been reported to be associated with earlier pubertal development. Catch-up growth occurs when children are adopted from developing countries to developed countries and is associated with earlier puberty than that of the reference population. The mechanisms responsible for the association between catch-up growth and early puberty remain unknown. For international adoption, the hypotheses include both shifts in nutritional status and also endocrine-disrupting chemicals from pesticides.

Comments

This review provides a valuable synthesis of studies linking catch-up growth and early puberty. One particularly interesting conclusion of this review is that early puberty has been observed after prenatal growth restriction or after combined pre- and postnatal restriction, but not after isolated postnatal undernutrition. Of course, lack of supporting evidence is not the same as evidence to the contrary. We do not have good studies of pubertal timing after transient postnatal growth restriction. In fact, even finding the right model to study a possible association is not easy. Most disorders that cause postnatal nutritional problems, such as gastrointestinal disease, may not resolve completely. International adoption can allow a recovery from postnatal undernutrition and is associated with early puberty in girls. Although exposure to endocrine-disrupting pesticides has been suggested as a possible etiology, the review mentions that, in one study, the girls with the most pronounced stunting and fastest catch-up growth had the lowest age of menarche, implicating the growth restriction in the etiology. The review points out that many international adoptees also had a low birth weight and thus were subject to combined fetal-postnatal undernutrition. However, not mentioned in the review is a study of 276 internationally adopted girls where those with a birth weight <2,500 g and those with a birth weight >2,500 g showed similar early pubertal development [3]. In our opinion, these data suggest, but do not prove, that early pubertal development is associated not only with catch-up growth after prenatal growth restriction but also after postnatal undernutrition.

Revisiting the relationship of weight and height in early childhood

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Adv Nutr 2012; 3: 250–254

Background

Although progress is being made in decreasing undernutrition in low- and middle income countries, wasting (weight-for-height z-score <−2) and stunting (height-for-age z-score <−2) during childhood continue to burden the poorest regions in the developing world. Ponderal and linear growth of children has been widely studied. However, epidemiologic evidence of a relationship between the two is inconsistent. Understanding the relationship between

weight-for height and height-for-age will allow organizations to better design and evaluate programs to improve childhood nutrition.

Results

At a cross-sectional level, there appears to be a limited relationship between ponderal and linear growth. Individual level, cross-sectional studies have found little association between weight-for height and height-for-age. Cross-sectional, population-level studies have demonstrated that height for age decreases throughout the first 2–3 years of life in many developing countries, whereas weight-for-height tends to falter during a more limited age window in the first year of life, after which weight-for-height stabilizes or increases. The precise timing of the weight-for-height faltering varies according to the country-specific age of weaning and other local factors. The geographic differences observed in linear and ponderal growth are most probably due to a combination of factors, including maternal nutritional status, exposure to and treatment of infectious diseases as well as dietary factors. If a child has an acute illness or dietary deficiency that result in weight loss, linear growth may slow down or cease until weight is recovered. Once the child regains weight, linear growth will continue and, given adequate nutritional resources and no further infections, catch-up growth may occur, returning the child to the original growth trajectory. However, children in developing countries often experience multiple insults with limited recovery time, leading to persistent height deficits.

Conclusions

Decreased weight-for-height and height-for-age are both important risk factors for illness and death during childhood, and changes in weight appear to have a lagged effect on height during early childhood. Further research is needed to identify the factors associated with recovery of linear growth after a child experiences an insult with decreased weight-for-height. A better understanding of these relationships will enable program managers to design improved strategies to intervene in order to improve childhood nutrition, growth and health.

Comments

In the present review the authors discuss the interpretation of different variables of the growth charts and the relationships between them. It is an important paper since it highlights the strength and limitations of growth charts and the information that can be derived and used from such data both on different populations and on an individual child. About 200 million children under the age of 5 in the developing world are stunted, most likely due to inadequate nutrition, and suffer from repeated episodes of infectious diseases. The growth chart is a very important tool for the pediatrician when following an individual child and for health authorities when following pediatric populations. Understanding the relationships between height-for-age, weight-for-age and weight-for-height in developing countries and their changes during intervention programs are extremely important and should be studied in prospective studies since better and cheaper tools for assessing nutritional interventions in different parts of the world do not exist.

Linear growth and final height characteristics in adolescent females with anorexia nervosa

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Objective

Malnutrition often results in growth deceleration, while nutritional rehabilitation results in catch-up growth that is often incomplete, resulting in compromised final adult height. Anorexia nervosa (AN) provides a model for studying the effect of caloric restriction during adolescence on growth and final height. Several studies reported growth failure or short stature in patients with AN. However, data regarding final height of patients with AN is scarce and inconclusive. The aims of the study were to evaluate the prevalence of growth retardation in a cohort of female adolescent patients with AN in order to assess the effect of weight restoration on catch-up growth and final height and to identify factors affecting catch-up growth and final height.

Methods

All female patients with AN (n = 211), hospitalized in an inpatient eating disorders department from January 1, 1987 to December 31, 1999, were included in this study and their medical charts were retrospectively reviewed. After assessment of the nutritional status, a nutritional rehabilitation program geared toward weight gain of 0.5–1.0 kg/week was constructed. Target weight was established according to age and the estimated potential height. Patients were discharged upon reaching their target weight and maintaining it for at least 2 weeks. After discharge, patients were followed until reaching the age of 18. Target weight was readjusted every 3 months during follow-up in patients who had not finished growing, and was increased gradually to allow for the expected height gain, based on the potential height. Height and weight were assessed at admission and thereafter routinely during hospitalization and follow-up. Final height (defined as height at age 18 or older, and at least 3 years after menarche) was measured in 69 patients 2–10 years after discharge.

Results

The mean age of the patients on admission was 16.6 ± 4.2 years; their mean BMI was 15.7 ± 1.02 , and their mean age at menarche was 12.7 ± 2.4 years. Patients' height standard deviation scores (SDS) on admission (-0.285 ± 1.02) and discharge (-0.271 ± 1.02) were significantly lower than expected in normal adolescents ($p < 0.001$). The extent of growth impairment was more severe among patients admitted at the age of 13 years on both

admission and discharge than patients admitted at an older age ($p = 0.03$), and patients admitted less than 1 year after menarche had more severe growth impairment on admission than patients admitted more than 1 year after menarche (height SDS -0.38 ± 1.01 vs. -0.19 ± 0.92 , $p = 0.03$). Final height SDS, available for 69 patients, was -0.258 ± 1.04 , significantly lower than expected in a normal population ($p = 0.04$), and was more severely compromised in patients who were admitted less than 1 year from their menarche. Height SDS on admission was a strong predictor of final height SDS ($p < 0.001$). BMISDS upon admission was negatively correlated with the change in height SDS between admission and final height ($r = -0.306$, $p = 0.011$). Other factors, including weight gain during hospitalization, duration of hospitalization, number of hospitalizations, and duration of AN prior to hospitalization had no significant effect on final height SDS. Complete growth data (i.e. pre-morbid, admission, discharge, and final height) was available for 29 patients. In these patients the preMalnutrition and Catch-Up Growth during Childhood and Puberty 95 morbid height SDS was not significantly different from the expected (-0.11 ± 1.1), whereas heights at the other time points were significantly lower (-0.56 ± 1.2 , -0.52 ± 1.2 , and -0.6 ± 1.2 , respectively, $p = 0.001$).

Conclusions

The findings of the study show that whereas the pre-morbid height of female adolescent with AN is normal, linear growth retardation is a prominent feature of the illness. Weight restoration is associated with catch-up growth, but complete catch-up is often not achieved with impaired final height. The findings emphasize the importance of early detection of AN, as growth retardation occurring during a critical growth period during puberty may be irreversible. Weight restoration geared towards restitution of height to the pre-morbid percentile for age should be initiated as early as possible.

Comments

This is a relatively large study that assesses the effect of undernutrition during puberty on growth rate and final adult height. AN provides an opportunity to study the 'pure' interaction between nutrition and growth in the adolescent age group since AN is not associated with other diseases which might affect growth, such as malnutrition in the developing countries. Adolescent girls with AN do catch-up during nutritional rehabilitation despite the fact that they usually present at a relatively 'old' age (mean age 16.6 ± 4.2 years). It is an important study despite the fact that it is of retrospective nature, thus it is associated with many limitations. It is possible that earlier admission could have a better outcome despite the authors' observation that patients admitted under the age of 13 years or less than 1 year after menarche were more severely affected than patients admitted at an older age. It is possible that patients who were admitted in an earlier age were more severely affected by the disease already at its earlier stages and started their AN at an earlier age than the patients who were admitted at an older age. It is clear that a prospective study is crucial to better understand the interaction between nutrition and growth in the adolescent age group using AN as a model. In such a study, information on the age when first symptoms appear, previous growth data, bone age, parents' height and family history should be collected and analyzed and the 'best' rehabilitation program should be studied. A well-designed study

could potentially teach us more about the interaction between nutrition and growth than any large retrospective study.

Catch-up growth after long-term implementation and weaning from ketogenic diet in pediatric epileptic patients

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Clin Nutr 2013; 32: 98–103

Objective

Ketogenic diet (KD) is a potent, accepted treatment in childhood epilepsy resistant to other medications. Previous studies have reported significant reductions in height and weight gain among children after implementation of KD. However, long-term studies on growth following dis96 Jee/Baron/Phillip/Bhutta continuation of long-term KD are still lacking. The aim was to analyze the presence of growth delay among children with epilepsy during implementation of long-term KD, and to assess the presence of catch-up growth 1 year after diet discontinuation.

Methods

Data were retrospectively collected from chart review of 40 children (20 males) with intractable epilepsy who began long-term KD, followed by successful weaning after more than 2 years of diet implementation. Inclusion criteria consisted of follow-up at the outpatient clinic with well documented epidemiologic and clinical data (including height and weight measurements) at the onset of the KD, 2 years with the diet, and 1 year after weaning. Growth retardation was defined as a statistically significant drop in mean z-scores of clinical parameter as compared to baseline. If growth retardation was followed by return to baseline z-scores or was followed by significant increase in z-scores of the clinical parameters after diet discontinuation, it was considered as catch-up growth.

Results

The median age at seizure onset was 0.79 ± 3.31 years (range 0.01–10.67), and the median age at start of KD was 4.58 ± 5.29 years (range 0.58–15.52). Subjects continued the diet for a mean duration of 2.35 ± 0.36 years (range 2.00–3.26). Mean period of follow-up after the diet was 1.70 ± 0.43 years (range 1.00–2.42). A significant reduction was found in both height and weight gain after prolonged KD. After a year of diet discontinuation, significant catch-up growth was evident in both height and weight. When comparing the growth patterns among subsets of the study patients, ambulation had favorable influence on growth

during KD and after diet discontinuation. Uncontrolled epilepsy and younger age at the start of KD contributed a negative impact on growth pattern.

Conclusions

KD is an effective treatment modality in intractable epileptic patients, and decreased growth velocity is a risk that most patients and guardians are willing to take. This preliminary study of long-term growth pattern among children treated with KD will aid in planning for long-term care extending beyond the duration of dietary treatment.

Comments

In recent years, the use of KD to treat patients with resectable epilepsy regained popularity among physicians since its effectiveness was shown to exceed in some cases even to most modern medications. The effect of KD on weight and height was described in the literature before but the present study allows us to get information of children's growth with the use of currently used KD protocols. The authors confirmed that indeed the use of KD for 2 years interferes with weight and height gain of children in different age groups. There was a significant reduction in height and weight z score after 2 years on DK with a statistical improvement in these parameters after a year of regular diet. It is an important study even though the number of patients studied was small and the follow-up period was limited (only 1 year on 'regular diet') since it confirms previous observations of attenuated growth during KD and suggests catch-up growth thereafter. However, it does not shed light on the mechanism by which KD influences growth and therefore does not contribute to our understanding of the mechanism of that phenomenon. It also does not teach us which diet was used during the year of catch-up. It seems like 2 years of KD is safe in most age groups since catch-up does occur during the year following the intervention period (except for the young age group) but what is the longest 'safe' period of KD in the different pediatric age groups? In addition, only 1 year of follow-up after KD was documented. The present study confirms the need for a prospective study with more patients and for a longer period of time in order to better understand the long-term effect of KD in the pediatric age group.

Catch-up growth in children born growth-restricted to mothers with hypertensive disorders of pregnancy

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Arch Dis Child 2013; 98: 30–35

Background

Fetal growth restriction (FGR) occurs frequently in preterm hypertensive disorders of pregnancy. This may vary in different regions and have long-term implications. The authors examined growth prospectively in a cohort of 135 children born to mothers who were admitted before 34 weeks' gestational age with a severe hypertensive disorder of pregnancy and evaluated height, weight, body mass index (BMI), head circumference (HC), SD scores (SDS) at 3 months and 1 and 4.5 years of age, and complete catch-up growth (height SDS-target height SDS >-1.6).

Results

The investigators found that on average growth scores were lower compared to Dutch growth curves, except for BMI at 3 months and girls' HC at all ages. Mean height SDS increased over time from -1.4 to -0.5 at 4.5 years, with 94% having complete catch-up growth. Mean BMI SDS also decreased from -0.2 at 3 months to -1.0 at 1 year, and was -0.8 at age 4.5. Mean HC SDS did not change over time and -0.3 at 4.5 years.

Conclusion

The authors concluded that although the majority of children born growth-restricted had catch-up growth of height within the normal range at 4.5 years of age, they remained smaller and lighter compared to Dutch growth charts. The customized birth weight ratio, as a measure of the degree of FGR, was related to all growth SDS at 4.5 years, while no association was seen with prematurity expressed by gestational age at birth.

Comments

The finding that fetal growth restriction or intrauterine growth retardation was associated with long-term effects on linear growth and body size is well recognized and reported across various countries and contexts. In a recent analysis of factors affecting growth across the world [4], close to 20% of all stunting at 24 months of age was attributed to being born small for gestational age (SGA). The fact that the degree of FGR was associated with all growth outcomes among the Dutch growth cohort is consistent with these observations and confirms the association. Recent data [5] also confirm the association of prematurity and fetal growth retardation in a subset of newborn infants in various geographies. The authors did not study this prematurity-SGA interaction which might have been of interest. There is also no information provided on metabolic factors or body composition at any of the time points and hence it is difficult to comment on associations with body fat distribution with status at birth or varying patterns of linear growth and catch-up in infancy and early childhood. The association of long-term outcomes with patterns of linear growth in early childhood is well recognized [6] and one hopes that this particular cohort would also be tracked to adult life.

Health profile of young adults born preterm: negative effects of rapid weight gain in early life

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J Clin Endocrinol Metab 2012; 97: 4498–4506

Background

The association of early postnatal weight gain with risks of developing cardiovascular disease (CVD) and type 2 diabetes mellitus (DM2) in adults born at term is well recognized. However, relatively little is known of such risks in infants born preterm. The authors investigated the association of weight gain and weight trajectories during early childhood among infants born prematurely with determinants of CVD and DM2 in early adulthood; specifically they assessed growth in infancy and determinants of CVD and DM2 among 162 young adults (18–24 years) born preterm (gestational age <36 weeks) in comparison with data of 217 young adults born at term.

Results

The authors demonstrated that early gain in weight for length in the period from preterm birth up to term and the 3 months thereafter was positively correlated with % body fat, waist circumference, total cholesterol and low-density lipoprotein cholesterol levels at 21 years of age. These effects were more marked in subjects with the highest weight gain in weight from birth to term age and rapid catch-up in weight thereafter in the first 3 months had significantly higher body fat percentage, waist circumference, acute insulin response and disposition index in early adulthood than subgroups with moderate and slower catch-up in weight.

Conclusions

The authors concluded that accelerated neonatal gain in weight relative to length after preterm birth (immediately after birth and during the first 3 months after term age) may be associated with metabolic changes suggestive of excessive risk CVD in early adulthood.

Comments

These findings present associations of early weight gain in the first few months of life with biomarkers of potential CVD (body fat, waist circumference and lipid levels). Given that linear growth data were not a focus of attention and that 'rapid growth' was largely defined as gains in weight and weight for length, it is difficult to ascertain the subset who gained in both length and weight ('optimal growth'). Early weight gain in the first 2 years of life has been shown to be appropriate and safe in evaluation of long-term outcomes in multi-country cohorts [7] and linear growth has been shown to be of benefit in recent evaluations based on pooled analysis of the same populations [6]. These findings must therefore be taken in the context of existing data and further information sought on linear growth as well as growth patterns in the 0- to 24-month window to assess the validity of this association. The issues in relation to the lack of information on patterns of intrauterine growth and standards are valid and could well be resolved by availability of intrauterine growth standards from the Intergrowth 21st study later this year.

Associated factors for accelerated growth in childhood: a systematic review

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Background

The authors conducted a systematic review of factors associated with accelerated growth, or catch-up growth based on studies (English, Spanish or Portuguese) in the Medline/PubMed database. The term ‘catch-up’ has been used for the accelerated growth of children who have suffered from restricted intrauterine growth based on either maternal nutritional factors or morbidity such as hypertensive disease. The authors focused on studies of outcomes in children 0–12 years of age who had either accelerated growth or catch-up growth studied after birth.

Results

There was a remarkable paucity of literature and out of the 2,155 articles highlighted, only 9 were considered suitable for further study. The authors found no uniformity in the operational definition of accelerated growth, or in the concept of catch-up. Factors associated with accelerated growth included primiparity, maternal smoking during pregnancy, lower birth weight, and early weaning.

Conclusions

The authors identified major limitations in the studies as lack of adequate controls and high rates of loss to follow-up. They called for further studies as they were unable to find convincing risk factors associated with accelerated growth in the available literature.

Comments

This review is illustrative of the state of the current evidence around early child growth and development with relatively few prospective observational studies and trials. As evidenced by data from the COHORTS studies, there is paucity of information on intrauterine and postnatal growth patterns, especially in relation to gestational age. The latter has been difficult to standardize in studies based on recall of maternal menstrual dates and expected date of delivery. Improvements in technology, notably ultrasound assessment of gestational age, have made it possible to assess fetal maturity with confidence and also assess phenotypes with a greater degree of confidence [8, 9]. Future studies also need to include biomarkers [10] in addition to growth and development parameters.

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