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The Cover Shot

照片攝於鳳凰古城附近的苗族山寨。時近農曆新年，2 位小女孩着上苗服在她們古舊村居的走廊玩耍時被拍下。走廊掛上笛族人風乾後的玉米和乾草。

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"To grow up" is one of the prime objectives of children. Parents would bring their children to professionals' attention when the pattern of growth deviates from normal expectation. Hormonal imbalance is often implicated but other organ system dysfunctions could also result in disturbance of growth, which by itself, is a sensitive indicator of health. It is of no wonder that paediatric consultations often begin with a measurement of growth.

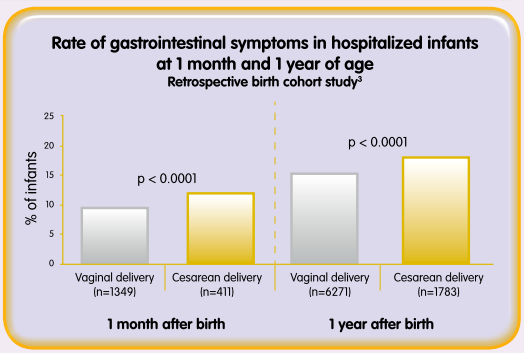
In this issue, we have five articles on the various aspects of growth or endocrine disturbance in children. Dr Betty BUT reviews the current indications in using growth hormone in children under the care of the Hospital Authority, the cost of treatment would be borne by public funding. Dr Aaron Yu reviews the clinical approach to pubertal disorders, which are increasingly aware of by both parents and professionals. Obesity as a non-communicable disease, continues to stress on our public health system. Dr Kin-tak Wong summarises the cardiovascular complications consequent to childhood obesity, providing useful information to health care workers in counselling patients suffering from the problem and their familes. Mr. Gordon Cheung, on the other hand, addresses the weight problem from an opposite direction and provides important tips in helping children with thriving problems. The debate on Calcium and Vitamin D metabolism remains as unresolved. Dr Sophie Leung, together with Dr Ruth Chan and Dr Warren Lee, summarise the current research findings on Calcium and Vitamin D requirements in Chinese children and the possible health consequences in deficiency, as well as in excess.

We take this opportunity to thank the authors for their contributions and the secretariat of the Hong Kong Federation of Medical Societies in preparing the scripts for printing.

Cesarean Delivery vs Vaginal Delivery - Are There Any Differences?

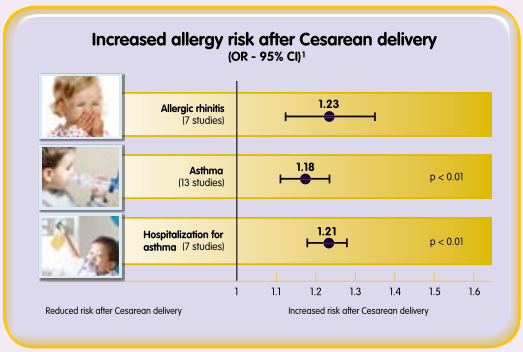
Gastrointestinal symptoms are more prevalent in Cesarean-born infants[3]

up to 1 year of age



A meta-analysis confirms that Cesarean delivery is a specific risk factor for allergies[1]

up to 23% more risk



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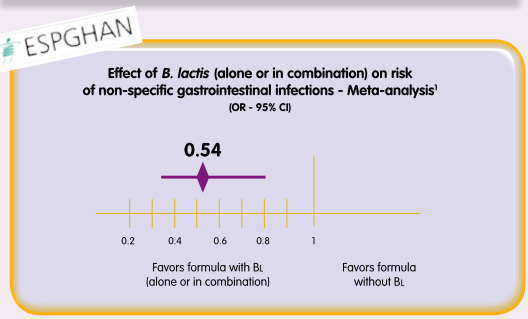

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Growth Hormone Treatment for children and adolescents in Hong Kong

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This article has been selected by the Editorial Board of the Hong Kong Medical Diary for participants in the CME programme of the Medical Council of Hong Kong (MCHK) to complete the following self-assessment questions in order to be awarded 1 CME credit under the programme upon returning the completed answer sheet to the Federation Secretariat on or before 31 May 2014.

Background

Human pituitary derived growth hormone (GH) was used to treat patients with growth hormone deficiency (GHD) and was provided by the Hong Kong Government from 1978 till 1985. The first case of Creutzfeldt-Jacob disease caused by prion-contaminated human pituitary derived GH was reported in 1985. Recombinant human GH was firstly approved for use in GHD by the Food and Drug Administration (FDA) of the United States (US) in the same year and it was used in Hong Kong since 1989. Approval for use was extended to patients with Turner Syndrome and chronic renal failure before transplantation by the Hospital Authority (HA) in Hong Kong since 1998. As it is used primarily for growth promotion, treatment should be stopped when the patient has nearly reached the final or target height. In recent years, more indications have been approved by the FDA and its use is supported by various governments (Table 1). Two additional indications for GH treatment including Prader Willi Syndrome (PWS) and short stature homeobox-containing (SHOX) gene disorders were approved by HA in 2012. Besides as a growth promoting agent, its use also aims at improving body composition for patients with PWS.[1] The objective of this paper is to discuss the use of GH in these conditions.

Table 1. Use of recombinant human growth hormone in various countries

FDA-US Year first approved	NICE-UK Recommended	PBS-Australia Eligible	Pharmac-NZ Eligible	HADF-HK Eligible
GH deficiency 1985	Yes	Yes	Yes	Yes
Adult GH deficiency 1996	Yes	–	Yes	–
CRI 1993	Yes	Yes	Yes	Yes 1998
Turner 1996	Yes	Yes	Yes	Yes 1998
Prader Willi 2000	Yes	Yes	Yes	Yes 2012
SGA (ht< -2 SD at 2yr) 2001	Yes (ht<- 2.5 SD at 4yr)	–	–	–
ISS (<- 2.25 SD & GV < 25% for BA) 2003		Yes (ht< 1% & GV < 25%)	Yes (ht< -3 SD & GV < 25%)	–
SHOX 2006	Yes	Yes	–	Yes 2012
Noonan 2007	-	–	–	–

Abbreviations: FDA, food and drug administration; US, United States; NICE, National Institute for Health and Clinical Excellence; UK, United Kingdom; PBS, Pharmaceutical Benefits Scheme; Pharmac, Pharmaceutical Management Agency; NZ, New Zealand; HADF, Hospital Authority Drug Formulary; HK, Hong Kong. GH, growth hormone; CRI, chronic renal insufficiency; SGA, small for gestational age; ISS, idiopathic short stature; SHOX, short stature homeobox-containing gene disorders; SD, standard deviation; ht, height; GV, growth velocity; BA, bone age; yr, year

Growth Hormone Deficiency

GHD occurs at an incidence of 1:4,000 – 10,000. Profound hypoglycaemia and prolonged jaundice during the neonatal period, micropenis, midline craniofacial abnormalities and a positive family history are conditions suggestive of congenital GHD. For acquired conditions, there may be history of brain tumour, cranial irradiation, head trauma, surgery or central nervous system (CNS) infection.

Workup of patients suspected of GHD includes clinical and auxological assessments as well as the exclusion of other systemic causes like hypothyroidism, chronic systemic disease, Turner Syndrome or skeletal disorder. Insulin-like growth factor -1 (IGF-1) may be measured but a low concentration is also found in children with hypothyroidism, malnutrition and liver disease. GH provocative tests can be performed with pharmacological agents such as glucagon, arginine, L-Dopa and insulin. In general, a peak GH concentration of less than 7-10 ug/L by two stimulation tests is considered abnormal although the cut-off levels are assays dependent.[2,3] In the presence of pathological causes such as brain tumour and multiple pituitary hormone deficiency, one abnormal provocative test is sufficient for diagnosing GHD. Sex hormone priming may be considered in girls aged >11.5 years and boys aged >13 years who are still in the prepubertal stage or have only early signs of puberty.[4] Magnetic resonance imaging (MRI) of the brain with particular attention to the hypothalamic-pituitary region should be performed in any child diagnosed with GHD.[2]

Studies have demonstrated that GH treatment increases the height velocity and final adult heights of children with GHD.[5] The recommended dose is 0.5 – 1 IU/kg/week (0.025-0.05 mg/kg/day). Although a higher

dose during puberty (2 IU/kg/week) may be considered in adolescents with late diagnosis and a diminished period of time for catch-up, it may not be necessary to increase the dosage if the height is maximised before the onset of puberty. The thyroid function should be monitored before and during treatment as central hypothyroidism may be unmasked by GH treatment and there may be increased conversion of T4 to T3.[6]

Turner Syndrome

Turner Syndrome (TS) is characterised by short stature, dysmorphism, cardiac and renal anomalies and primary hypogonadism in phenotypic females. It is a common chromosomal condition occurring at a frequency of about 1 in 2000-2500 live female births and is caused by partial or complete X chromosome monosomy.[7] Turner patients do not have GHD essentially but there is GH or IGF-1 resistance. It is believed that haploinsufficiency of one copy of the SHOX gene located within the pseudoautosomal region on the distal short arm of the X (and Y) chromosomes is primarily responsible for the growth problems.[8] Growth failure generally begins in utero, continues into infancy and childhood, and is exaggerated by the absence of pubertal growth spurt. The reported mean final height of Chinese patients with TS in Hong Kong was 142 cm[9] compared to 147 cm observed in Northern Europeans.[10]

Studies including randomised, controlled trials have repeatedly demonstrated that GH treatment is effective in promoting height gain and improves the final adult height. The average final height gain was around 5 to 8 cm over a treatment period ranging from 5.5 to 7.6 years although the response to treatment can be highly variable.[11,12] The recommended dose is 1 IU/kg/week divided daily (0.045-0.05 mg/kg/day).[13]

The optimal age of starting GH treatment has not been established. A recent randomised, controlled 2-year study demonstrated that early initiation of GH treatment as young as 9 months of age prevented growth failure that typically occurred in the first few years of life.[14] Hence, it is recommended that treatment with GH should be considered as soon as growth failure (decreasing height centiles) is noted.[15]

Hypothyroidism, glucose intolerance and scoliosis with or without kyphosis are more common in patients with Turner Syndrome. Assessment and monitoring of these conditions are required.

Chronic renal insufficiency before renal transplantation

Growth failure is common in patients with chronic kidney disease (CKD) and the adult height is less than 2 SD below the mean in about half of the patients. Many factors including protein-calorie malnutrition, acid-base disturbances, hyperparathyroidism, glucocorticoid treatment, derangements in the GH-IGF axis and GH insensitivity contribute to growth failure.[16]

GH given at a dose of 28-30 IU/m^2/week (0.045-0.05 mg/kg/day) is effective in improving the height velocity. The adult height may be improved by approximately 7 – 11 cm.[17] It is generally considered safe without any adverse effect on the renal function. However, careful monitoring of the renal function is mandatory. It is suggested to perform X-ray hips before initiating GH treatment and to stop GH in the presence of active renal osteodystrophy (hyperparathyroidism) as slipped capital femoral epiphysis is more common in patients with CKD. Clinical trials have demonstrated growth promoting effects and safety of GH therapy in children after renal transplantation. Recommendation on the duration and time of initiating GH requires further evaluation.[18]

Prader Willi Syndrome

Prader Willi Syndrome (PWS) is characterised by severe neonatal hypotonia, poor feeding early in life, short stature, hyperphagia after infancy leading to morbid obesity, learning disabilities, behavioural and psychiatric problems. It is also associated with scoliosis, sleep-disordered breathing and endocrine problems including growth hormone insufficiency, hypothyroidism, hypogonadism and adrenal insufficiency. It is caused by lack of expression of paternally inherited imprinted genes on chromosome 15q11-q13.[19] The incidence is around 1 in 2,5000 births.

Growth failure occurs early from the prenatal period and there is lack of a pubertal growth spurt in patients with PWS. It is generally believed that patients with PWS have disturbed hypothalamic control of GH secretion. The mean spontaneous adult height has been reported as 162 cm in boys and 150 cm in girls.[20] No local data on the prevalence and adult height are available in Hong Kong.

Studies including those on the long-term use of GH consistently show improvements in height, body composition and the lean body mass without significant adverse side effects.[21,22] GH therapy also improves the overall muscle tone, physical strength, and agility.[23] A recent study showed that GH treatment initiated prior to 2 years of age (range 4-32 months, mean 13 +/- 6 months) changed the natural history of PWS by improving the body composition, motor function, height and lipid profiles.[24] Cognitive development may also be improved by GH therapy.[25]

GH treatment is approved for those less than 18 years of chronological age. The recommended dose of GH in PWS is 21 IU/m2/week (1 mg/m2/day or ~0.035 mg/kg/day) with a maximum of 8 IU/day (2.7 mg/day). It is suggested to calculate the dose in these obese subjects based on the body surface area instead of the body weight to avoid excessive dosing of markedly obese subjects and to minimise the risk of side effects. For those with mature skeletons (bone age ≥ 14 years in female and ≥ 16 years in male), a dose of 0.12 IU/kg/wk is recommended.

PWS patients are at risk of obstructive sleep apnoea (OSA) and central hypoventilation during sleep. Unexpected deaths occur in PWS patients with or without GH treatment.[26] It is recommended to start with a low dose at 5-7 IU/m2/week and increased

gradually to 21 IU/m2/week over the first weeks and months. The insulin-like growth factor-1 (IGF-1) level was demonstrated to have a role in worsening OSA[27] and this should be monitored regularly and maintained within two standard deviations (SD) above the mean. A recent study reported that 13 of 15 children who did not show significant sleep-related disordered breathing at baseline or 6 weeks after initiation of GH therapy remained free of the disorder after 2 years of GH therapy.[28] In addition, improvements in arterial oxygenation and the cardiovascular function during sleep have been shown in PWS children treated with GH.[29] Nevertheless, it is important to monitor the GH effects clinically and watch out for development or worsening of sleep apnoea especially during the period of acute respiratory illness. Sleep studies and ear, nose and throat (ENT) evaluations should be performed before and ideally within 6 months after starting GH.[30]

Scoliosis with or without kyphosis is common in patients with PWS and is probably related to hypotonia and obesity. As GH promotes linear growth, there are concerns whether GH therapy will increase the incidence or severity of scoliosis. Controlled studies have demonstrated that GH does not significantly increase the risk of developing scoliosis.[31] However, regular assessments and monitoring for scoliosis are required in patients with PWS as they are at-risk.

Patients with PWS are at risk of developing Type 2 Diabetes Mellitus. No significant (but small) increase in fasting sugar and insulin has been reported in prepubertal patients with PWS treated with GH when compared with age-matched controls.[24] Another study on older patients showed the frequency of impaired glucose tolerance was decreased during 3 years of GH therapy.[32] Although GH is a counter-regulatory to insulin, improved body composition after GH therapy may potentially increase insulin sensitivity. It is unclear at present whether GH therapy will benefit glucose metabolism and further studies are required.

Other endocrine problems such as hypothyroidism, hypogonadism and adrenal insufficiency should be monitored and treated accordingly.

Short stature homeobox-containing (SHOX) gene disorders

The SHOX gene encodes a homeodomain transcription factor responsible for long bone growth and is located in the pseudoautosomal regions at the distal ends of the X and Y chromosomes.[8] Normal growth requires two functional copies of the gene. Hence, growth impairment can occur if one copy of the SHOX gene has been inactivated by mutation or deletion (haploinsufficiency). It is responsible for growth deficits in patients with Turner Syndrome, about 70% of patients with Leri-Weill dyschondrosteosis (LWD) and 2-3% of patients with idiopathic short stature.[33] The phenotypes of individuals with SHOX gene disorders can be very variable, ranging from short stature without obvious dysmorphism to severe mesomelic skeletal dysplasia (shortening and bowing of the forearms and lower legs) with Madelung deformity.[34] The overall prevalence of SHOX gene disorders in patients with short stature is 1 in ~2500. A scoring system based on the clinical features to identify the most appropriate subjects to test for SHOX deficiency has been developed.[35] No published data on prevalence are available in Hong Kong.

A randomised, controlled, multicentre study shows that GH treatment is effective in promoting height gain and its long-term effectiveness on SHOX gene disorders is similar to that on Turner patients. The adult height is increased by 1.1 SD when compared with baseline.[36,37] The recommended dose is 1 IU/kg/week (0.045-0.05 mg/kg/day).

Side effects of GH

Pseudotumor cerebri (benign intracranial hypertension) may develop and usually resolves after stopping GH.[38] When the patient has recovered, GH can be restarted at a lower dose (one fourth of the previous dose) and then stepped up gradually to full dose over a few weeks. Slipped capital femoral epiphyses and worsening of the existing scoliosis are more common in rapidly growing children and may require surgical correction. Continuation of GH treatment is recommended in general.[39] GH may induce carbohydrate intolerance in children with compromised insulin secretion. Other side effects include prepubertal gynaecomastia, oedema, arthralgia, myalgia and local reaction at the injection site.

There is always concern that GH treatment might increase the risk of tumour recurrence or progression or the appearance of a second neoplasm as GH and IGF-1 have mitogenic and anti-apoptotic activities.[40] At present, there is no conclusive evidence to support a role of GH in cancer pathogenesis. The Safety and Appropriateness of Growth hormone treatment in Europe (SAGhE) Study for the French population reported that the mortality rate was increased in adults treated as children with idiopathic isolated GH deficiency or childhood short stature particularly in those who had received doses greater than 0.05 mg/kg/day. Bone tumour-related and cerebrovascular disease-related mortality was increased.[41] Another paper from the SAGhE Study did not show the mortality rate was increased in patients treated with GH in Belgium, the Netherlands and Sweden.[42] Nevertheless, the presence of an active malignancy is a contraindication to GH treatment and it is recommended to start GH treatment one year after the completion of tumour treatment with no further evidence of tumour recurrence or growth. Careful follow-up of GH-treated patients, particularly those on higher doses is required. Monitoring of IGF-1 levels in patients treated with GH is recommended to ensure that they are maintained within age appropriate limits.[39]

Conclusions

In Hong Kong, GH treatment is approved for children and adolescents with GH deficiency, Turner Syndrome during the growth period, chronic renal failure before transplantation, Prader Willi Syndrome and SHOX gene disorders. Treatment with GH should always be initiated and monitored by a paediatrician with special expertise in managing growth hormone disorders in

children as GH is expensive and not without side effects. The multiple health problems faced by most of these patients also mean that a multi-disciplinary approach is required to reduce morbidity, mortality and to improve their quality of life.

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Growth Hormone Treatment for children and adolescents in Hong Kong." by Dr Betty WM BUT and complete the following self-assessment questions. Participants in the MCHK CME Programme will be awarded CME credit under the Programme for returning completed answer sheets via fax (2865 0345) or by mail to the Federation Secretariat on or before 31 May 2014. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary.

Questions 1-10: Please answer T (true) or F (false)

1. The incidence of Growth Hormone Deficiency is 1:25000
2. The incidence of Prader Willi Syndrome is 1: 25000
3. Neonatal hypotonia is a common feature in Prader Willi Syndrome
4. According to a Hong Kong study reported in 1997, the mean final adult height for Turner Syndrome in Hong Kong is 147cm
5. The Hong Kong Hospital Authority will provide Growth Hormone treatment to children with idiopathic short statures
6. The SHOX gene encodes a homeodomain transcription factor responsible for long bone growth and is located in an autosome
7. Small for gestational age is an approved indication for Growth Hormone treatment in the United Kingdom
8. Creutzfeldt-Jacob disease caused by prion-contaminated human pituitary derived GH was firstly reported in 1985
9. Monitoring of Insulin-like growth factor-1 (IGF-1) levels is not recommended in patients treated with Growth Hormone
10. Turner patients usually have GH deficiency and would be equally responsive to Growth Hormone treatment
11. Thyroid function should be monitored during treatment with Growth Hormone

ANSWER SHEET FOR MAY 2014

Please return the completed answer sheet to the Federation Secretariat on or before 31 May 2014 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Growth Hormone Treatment for children and adolescents in Hong Kong

Dr Betty WM BUT

MBBS (HK), FHKCPaed, FHKAM (Paed), FRCP (Edin)

Consultant Paediatrician, Queen Elizabeth Hospital, Hospital Authority, HK
President of the Hong Kong Society of Paediatric Endocrinology and Metabolism

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Answers to April 2014 Issue

Paediatric Neuroanaesthesia

1. e 2. e 3. e 4. a 5. d 6. e 7. d 8. d 9. d 10. e

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21 May	Systemic Lupus Erythematosus: What's new?	Dr. Priscilla WONG Specialist in Rheumatology
28 May	Advanced management of Rheumatoid Arthritis	Dr. Kelly CHAN Specialist in Rheumatology
18 June	Autoimmune Markers made easy	Dr. Cecilia O'young Specialist in Rheumatology
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16 June	**Breastfeeding** • Benefits of breastfeeding • Practical tips for successful breastfeeding • Problem shooting	Dr. Veronica HO Freelance Consultant in Nutrition/Dietetic and Community Health Education
23 June	**Weaning** • Food choice and preparation • Method of introduction • Feeding techniques • Problematic eating behaviors	Ms. Rhoda NG Internal Coordinator, HKNA Freelance Dietitian
30 June	**Growth assessment and its impacts on children's health** • Growth assessment and its interpretation - practice tips and common myths • Concept of early nutritional programming • Health consequences and risk factors for accelerated growth velocity in children	Mr. Gordon CHEUNG President-elect, HKNA Dietitian in Private Practice
7 July	**Children and adolescent nutrition** • Nutritional needs of children and adolescents • Eating disorders – overview, nutritional assessment and management • Building healthy eating habits • Use of nutrition labels and healthy lunch and snack ideas	Ms. Mandy MAN External Coordinator, HKNA Dietitian / Wong Tai Sin Hospital
14 July	**Management on overweight children** • Evidence-based approach on childhood obesity management • "Family-based and Multi-component Weight Management Program"	Ms. Sally POON Newsletter Editor, HKNA Dietitian in Private Practice

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Calcium and Vitamin D requirement in infancy and Childhood

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Dr Sophie SF LEUNG Dr Ruth SM CHAN Dr Warren TK LEE

Introduction

A recent study has shown that Hong Kong children consumed too much formula milk[1]. One of the reasons is that parents believed that drinking more milk is good for their children's health. Currently formula milks are marketed in Hong Kong as starting formula, No. 2, No. 3 and No. 4 for different stages of growth with increasing concentration of calcium, calorie, protein and sodium (Table 1).

Table1: Nutrient contents of some milk formulae marketed in Hong Kong compared to those of human milk

Per 100ml	Human milk	Starting formula,0-6 m	No.2 formula, 6-12 m	No.3 formula, 1-3 yr	No . 4 formula , 4-7 yr
Energy, Kcal	67	68	71	81-100	105
Protein, g	1.5	1.55	2.8	3.1	3.9
Carbohydrate, g	7.0	7.7	8	10-14	12-14
Fat, g	3.8	3.5	3.1	3.3	3.3-4.2
Sodium, g	18	16	34	38	55
Calcium, mg: phosphorus, mg	34 : 14	42 : 24	103 : 70	123 : 88	142 : 107

Assuming a 6 months old baby consuming No.2 formula 800 ml/d would have about 800 mg/d calcium while a breast fed one may have only about 280 mg/d. For a 5 years old , a child consuming 720 ml daily No.4 formula would have about 1000 mg/d while a child who does not consume any milk might have 300-500 mg/d calcium. Is it true that breast milk can no longer support the calcium need of an infant beyond 6 months of age? Is it true that children should not stop drinking milk even at the age of 5 years in order to get enough calcium?

Mineralisation of bone

Calcium is an important nutrient, both for bone growth and for cellular function. Skeletal calcium accretion begins in the third trimester of foetal life, continues through infancy and childhood and peaks at adolescence. Consolidation of bone mass has been found to occur in late adolescence and early adulthood after cessation of bone growth[2], and then stops at adulthood, followed by bone loss in late adulthood (menopause in females). Such process is a slow one, regulated not so much by dietary calcium but by insulin growth factors, parathyroid hormone-related peptides, growth hormone, oestrogen and testosterone. Fig.1 shows that the mean spinal bone mineralisation as measured by Dual Energy X-ray Absorptiometry (DXA) in Hong Kong children and adults changed with age even though the dietary calcium for all the age groups were in the same range of 500 mg/d[3]. The role of hormones far exceeds that of calcium in modifying bone development and mineralisation.

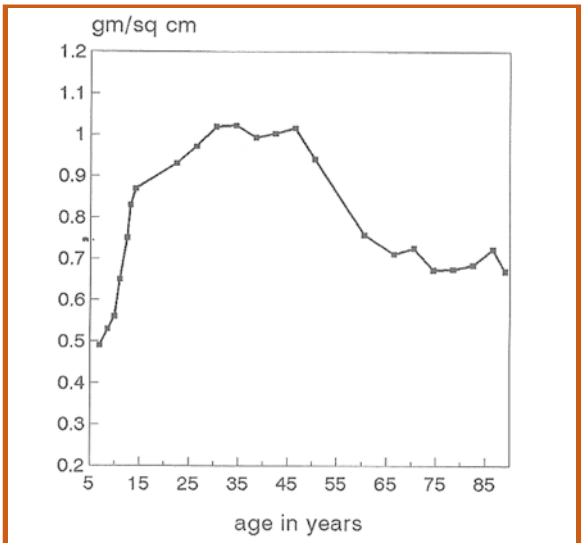

Fig.1: Mean bone mineral content of spine (L2-L4) of Hong Kong Chinese females from childhood to elderly

Calcium absorption and supplementation studies in Chinese children

Would a higher calcium intake in childhood enhance the mineralistion with the hope of preventing osteoporosis in later life? This possibility has been examined by a series of local studies:

1. Hong Kong children born in the eighties were mostly bottle fed during infancy. Milk consumption continued throughout their childhood. The calcium intake from 2 to 5 years old were of the range of 500 mg/d. The efficiency of calcium absorption (approximately 60%) measured by doubly labelled calcium stable isotope was double that of the Caucasian population[4]. This implies an adaptation in our body mechanism for an habitual 'lower' calcium intake practised by our ancestors for thousands of years.

2. Children in the Mainland who did not consume formula milk in infancy and childhood had only 300 mg calcium per day.

3. Supplementation of the Hong Kong and Mainland Chinese children with an extra 300 mg calcium a day at the age of 7 years did show a more rapid increase in bone mineral density[5,6]. But once the supplementation was stopped after 18 months, the rate of increase slowed down[7,8].

This implies that the strongest factor to determine the peak bone mass is most likely to be genetic. The benefit of having a higher calcium intake to prevent osteoporosis in late adulthood has not yet been confirmed by studies.

Calcium requirements

Approximately 1 kg of calcium is deposited in the skeleton between birth and adulthood. The daily calcium accretion rate is around 150-200 mg/d assuming the growth rate being constant throughout this period. On top of this there is obligatory loss through the gut, urine and skin[9]. During the process of bone growth, there is continuous bone remodelling to adapt mobility and mechanic stress in the skeletal support. This corresponds to the high fluxes of calcium in and out of the bone.

Calcium in the diet is mainly absorbed in the proximal small intestine. Active absorption is accomplished by a tight regulation of 1,25(OH)2D and parathyroid hormone (PTH) to maintain the ionised calcium in the extracellular fluid within normal. If the intake is too low (<200mg/day) even the maximum activity of the Vit D dependent intestinal active transport system cannot provide enough net calcium absorption to replace obligatory losses. Increased blood levels of PTH and 1,25(OH)2D would be secreted to activate osteoclastic bone resorption and to enhance absorption in the gut. Calcium intake greater than 4 g/d would force passive intestinal absorption on top of the down regulation on the intestinal active transport and renal tubular calcium re-absorption. This can cause hypercalciuria, nephrocalcinosis, progressive renal failure and hypercalcaemia[10]. However, intake should not be above 2000 mg as this has been shown to be associated with prostate cancer[11].

The efficiency of absorption of calcium varies with the food sources. The bioavailability of calcium in human milk and in green leafy vegetables is much higher than that in cow milk or cow milk formulae. Calcium absorption can be reduced in the presence of high phosphate(as in soft drinks), high animal protein, high sodium, high caffeine etc. In setting up a national recommendation for daily calcium intake adjustment is required to take into consideration of the customary dietary habits of the population. Such difficulty can be reflected in setting up the calcium requirement. Say for a 5 years old child, it can range from 450 mg/d in the United Kingdom[12] , 600mg/d in WHO[13] and Singapore[14], 800 mg/d in China[15] and 1000mg/d in the United States[16] (Table 2). During lactation, mothers have to provide 200 mg calcium in the daily milk supply. Their bone mineral density would fall but then regain to the baseline once breast feeding is stopped[17]. Supplementation of calcium during lactation did not show much difference. Once again, it shows how hormones play a major role in the determinant of bone accretion and bone loss.

Table 2. Comparison of the recommended dietary calcium requirements, mg/day of infants and children below 5 years of age in some countries

	WHO	US	UK	China	Singapore
2 m	Human milk:300 Cow milk:400	200	525	300	Human milk :300 Cow milk 400
6 m	Human milk:300 Cow milk:400	200	525	400	Human milk:300 Cow milk:400
12 m	400	200	525	600	500
24 m	500	700	350	600	500
36 m	500	700	350	600	500
48 m	600	1000	450	800	600
60 m	600	1000	450	800	600

Vitamin D requirement

Vitamin (Vit) D and its metabolites should be considered as hormones and not just nutrients. With adequate exposure to sunlight the body is able to manufacture Vit D for pregnant women, infants, children and adults. By exposing the face and arms under the sun for 15 minutes three times per week should be an effective way to ensure an adequate amount of Vit D in the body. In response to ultraviolet radiation on the skin Vit D can be formed from 7-dehydrocholesterol. It is then converted to 25 hydroxyvitamin D [25(OH)D] in the liver for transport and storage. When necessary, it would then be converted to the active form of 1,25(OH)2D in the kidney under the regulation of PTH. There seems to be no problem with excessive sunlight exposure related to the usual daily outdoor activities. Populations who are in the tropics are protected by their dark skin. Problems arise when these populations immigrate to a temperate region, even worse if there is air pollution. Infants and children may then suffer from rickets - a form of Vit D deficiency in children prior to epiphyseal fusion. With the changing of lifestyle and the fear of skin cancer, people are not getting enough sunlight, therefore the oral form of Vit D in foods (Vitamin D2 from plant source and Vitamin D3 from animal source) or supplementation have to be considered. Serum level of 25(OH)D <15 ng/ml in adults and < 10ng/ml in infants were shown to be associated with increasing PTH levels and lower bone density so these levels were taken as the cutoff for Vit D deficiency. It is very rare to have Vit D overdose. In case of excessive Vit D, there would be an increase in urine calcium excretion >250 mg/24hrs. Theoretically the amount of Vit D consumed should be that to keep urine calcium within the range of 100-250 mg/24hrs. For a long time the recommendation for oral Vit D is 400 iu/d. Currently there is some suggestion to increase to 800 iu/d. because even a suboptimal Vit D deficiency may not be beneficial to health.

Hong Kong infants born in 1984 had a Vit D level above 10 ng/ml[18] . None had any clinical features of rickets. These studied infants were brought up in Shatin. Many of them were brought to the playground for outdoor activities. These infants were fed with milk formulae fortified with Vit D. Over the last thirty years, Hong Kong's lifestyle and child care practices have changed. It seems more likely for pregnant mothers to hide themselves away from the sun by working long hours, taking the underground transport , using more sunscreens and spending weekends in shaded shopping centres. Likewise many infants and children had their out of home activities indoor.

Conclusion

While there is a need for ongoing research on the optimal requirement for calcium and Vit.D in children and adults there is no doubt that the optimal infant food is human milk, not just for the first 6 months but for the following months up to two years, complimenting a variety of solid foods. The bioavailability of calcium is good enough in spite of its lower calcium content compared to cow milk or milk formulae. Adequate sunlight exposure and outdoor physical activity should be encouraged among parents, especially pregnant mothers,similarly for infants and children.

It is worth noting that in populations whose ancestors did not drink milk habitually, like those in most Asian countries, their calcium intake would be lower than the cow milk drinking population. Their bodies have already got the compensating mechanism. It is tempting to comment such populations as calcium deficient. This is one of the reasons why milk formulae have been so aggressively marketed in Hong Kong and the nearby Asian countries. Apart from calcium, milk formulae contain other components-lactose, extra calorie, extra fat, extra animal protein and possibly hormones. These children have to face all these associated consequences as well. A balanced diet for a 2-5 years child should include plenty of natural plant based diet-rice, vegetables and fruits, including some calcium rich foods[19,20] (Table 3). If the consumption of cow milk or milk formulae would compromise the children's appetite for family food and their health (eczema, chronic constipation, frequent coughs and colds, etc) there is no reason why milk cannot be stopped. In case of doubt parents can consult a physician or a dietitian for the adequacy of nutrients.

Table 3: Amount of calcium in mg per 100 gm of some calcium rich foods in Hong Kong

Foods	Calcium, mg/100g	Foods	Calcium, mg/100g
Cow milk	120	Pak Choi	140
Cheese, cheddar	800	Choi Sum	140
Tofu	240	Watercress	220
Tofu sheet	300	Broccoli	100
Sesame	540	Sardine	550
Fig, dry	280	Shrimp, dry	880
Spinach	159	Small fish, dry	761

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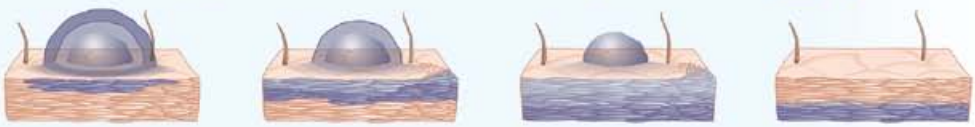

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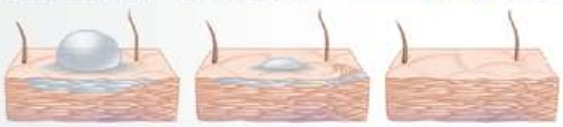

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Cardiovascular Dysfunction in Obese Children

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Cardiovascular (CV) diseases become more common in parallel with the rise in childhood obesity[1]. Obese children are prone to increased risks of CV morbidity and mortality in adulthood. Recent studies demonstrate that obese children show early signs of CV dysfunction. This article sought to address the CV abnormalities in obese children and highlights the importance and need for early detection and intervention so as to alleviate this potentially severe health problem.

Obesity in children is defined as a body mass index (BMI) at or above the 95th percentile for age and sex[2]. An alarming increase in obesity has been noticed amongst children and adolescents. A recent study reports that 17.9% of students are obese in Hong Kong[3]. Childhood obesity is highly predictive of adult obesity. Being obese in childhood increases the risk of CV morbidity in adulthood[4]. Childhood obesity is accompanied by concurrent abnormal CV changes, suggesting an emerging problem requiring immediate attention to prevent progressive CV damage from childhood.

Cardiac abnormalities and dysfunction

There are increased metabolic demands due to greater adipose tissue, lean mass and expanded blood volume in obese subjects[5]. Studies have reported that the cardiac mass, left atrial and left ventricular dimensions are significantly greater in obese children[6]. The thickness of the epicardial fat is established to be a CV disease risk predictor in adults[7]. It may also be a useful tool for the assessment of CV risks in children. Thicker epicardial fat has been reported in obese children than lean children[6].

Increased heart size in the obese children results in greater cardiac output than lean subjects[8]. Despite these indicators of augmented heart size and output, obese subjects often demonstrate evidence of impaired myocardial function[5]. Subclinical depression in left ventricular function among obese children is observed[9]. The cardiac dysfunction, as a consequence of chronic volume overload, is related to the severity and chronicity of obesity. Studies of obese children have reported both impaired systolic and diastolic dysfunction[10]. These abnormalities can be better detected by tissue Doppler, strain rate analyses and speckle tracking echocardiography[10]. Hence conventional methods have limited roles in recognition of early ventricular dysfunction, and therefore the severity of cardiac dysfunction in childhood obesity may be underestimated.

Obesity is obviously detrimental to an endurance exercise test. Among obese children, there is a negative correlation between BMI and distance on a 12-minute walk test[11]. When exercise capacity is measured by maximal oxygen consumption relative to body mass, an adverse effect of obesity is also observed[12].

Vascular abnormalities and dysfunction

Endothelial dysfunction and arterial stiffness in obese children present from early life. Post-mortem examinations on children who died from other unrelated causes report fatty streaks and fibrous plaque lesions in the aorta[13]. This suggests that arterial wall damage begins during childhood. Several tools can analyse functional and morphologic characteristics of arteries in adults. These methods have been utilised to analyse vascular function in the paediatric population.

The carotid intima-media thickness (IMT), measured by ultrasonography, is a marker of pre-clinical atherosclerosis. IMT is predictive of CV morbidity and mortality in adults[14]. Studies have reported an increased carotid IMT in obese children when compared with lean controls[15].

Arterial stiffness correlates closely with early atherosclerosis in obese children[16]. It can be estimated using pulse wave velocity (PWV). PWV represents the time that the pulse wave takes to travel a given distance along the vasculature. The faster is the PWV, the greater is the arterial stiffness. A significant positive correlation was observed between the degree of obesity and PWV in adults[17]. PWV is predictive of CV morbidity and mortality in adults[18]. It was positively correlated with BMI in adolescents[19]. Greater arterial stiffness has been reported in obese children[15].

Flow-mediated dilatation (FMD) is a measure of the endothelial function. FMD is expressed as the percentage change in the brachial artery diameter from baseline in response to increased flow.Damage to the endothelium, assessed by brachial artery flow-mediated dilation (FMD), is an early clinical indicator of atherosclerosis and vascular damage in adults[20]. FMD may be useful in identifying those children with early signs of atherosclerotic development. Researches have reported that obese children have significantly lower FMD compared with lean children[15].

Effects of Interventions

Previous studies in adults have shown that the left ventricular dimension and function improve with weight reduction through diets with or without physical activities[21]. The link between improvement of vascular function and dietary weight loss has been established in adults[22].

Current guidelines advocate lifestyle, dietary, and exercise interventions for the prevention and management of childhood obesity using BMI as an outcome measure[23]. More aggressive approaches such as pharmacotherapy and bariatric surgery are reserved for seriously obese adolescents who have failed conventional interventions[24]. Exercise programmes have positive effects on BMI and measures of adiposity over short-term[25] and medium-term[26]. Some studies have demonstrated improved endothelial function without reduction of BMI[27]. Therefore using BMI alone as the only outcome measure may underrate the effectiveness of the intervention.

One local study has reported that 6 weeks' dieting alone or dieting plus exercise programmes are both associated with improvements in FMD and IMT in obese children. Changes are significantly greater after dieting plus exercise[28]. Exercise continued for 1 year results in further improvements in FMD and regression of IMT. Thus a combination of optimal diet and exercise training may successfully reverse vascular damges. Longitudinal randomised control studies with long-term follow-ups are required to formulate the most effective interventions.

Practical issues

Childhood obesity is associated with various quantifiable changes in CV structure and function. In clinical settings, these changes are not routinely measured. Thus, the related CV changes are likely underrated, and therefore sub-optimally handled. A standardised clinical protocol for CV evaluation of obese children is required. In view of the reversibility of such changes, this protocol should include early detection and evaluation of subclinical cardiac dysfunction. However, incorporating these assessments into clinical practice is demanding because specialised equipment and personnel are required. Furthermore, there is a lack of a normal range for reference, and acquisition of this reference data is essential for setting up of clinical guidelines.

On the other hand, utilising biomarkers for assessment of CV diseases seems to be a simple solution for clinical practice. Nonetheless, there are insufficient data to recommend the use of any biomarkers in screening children for CV diseases[29]. Therefore, identifying biomarkers for obesity-related CV diseases may provide cost-effective measures that can be used to screen obese children for CV dysfunction. Furthermore, future researches should integrate all clinically important parameters for evaluating the success of an intervention, instead of BMI alone.

Conclusions

The progression of CV dysfunction in obese children is influenced by several genetic and environmental factors. Other co-morbidities such as hypertension and dyslipidaemia further add intricacy to the mechanism of obesity-related CV dysfunction[30]. Childhood obesity not only increases CV risks in adulthood, but is also associated with CV damages during childhood. Hence there is a compelling need for prevention and treatment protocols designed for obese children. Longitudinal studies are required to delineate the progression of CV abnormalities and evaluate the effectiveness of intervention. These results will be useful for formulation of evidence-based protocols to provide best treatment options.

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Faltering Growth in Local Infants and Young Children: From a Dietetic Perspective

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Growth is a unique and dynamic process in children which reflects invaluable information about the child's health and well-being. In clinical settings, a normal growth pattern helps rule out multiple endocrine and non-endocrine disorders and indicates nutrition adequacy. Recent scientific evidence also points out the impact of early nutrition and growth, as well as the health of later life. For the parents and caregivers, a baby or child failing to grow as people expected might lead to pejorative comments on their caring practices and possible harm to health and development. The anxiety derived from such a situation perpetuates and creates a stressful caring environment, and ruins the psychosocial development and parent-child bonding, as well as imposing the risk of eating behavioural problems. Traditionally, the term 'failure to thrive' (FTT) was widely used to describe the failure to achieve an expected growth in weight and/or height. This phrase has been criticised being too negative and induced blame on the parents, and 'faltering growth' is now a widely accepted alternative to characterise this childhood condition[1].

What Is Faltering Growth?

It is very important to recognise that "faltering growth" is a sign or growth pattern rather than a diagnosis or disease[2,3]. Although the concept of faltering growth or FTT was widely adopted, no consensus exists concerning the specific anthropometrical criteria to define this description[4,5]. Thus, it has been used to cover a broad range of different anthropometric indicators, usually based on growth charts for weight and/or height. Early studies defined it simply on the basis of being or falling below a low centile line[6], but it tends to emphasise low birth weight rather than poor postnatal weight gain. Serial measurements of weight and height and identifying children dropping through major centile spaces are now regarded as a preferable way, but it may over-identify large newborns with regression to the mean (i.e. smaller babies tend to grow faster than larger babies and both tend to move towards the mean weight). In clinical practice, a weight that crosses more than two major centile spaces downwards would be considered as the threshold for concern[7], but other anthropometric parameters such as weight-for-height, height relative to parental height, clinical history are also useful in identifying faltering growth.

Causes of Faltering Growth

It is rational that faltering occurs as a consequence of inadequate nutrition, since the energy requirements in infancy are very high. The reasons of the uncoupled energy intake and requirement are complex and usually multifactorial. The conventional classification of organic and non-organic causes of faltering growth is overly simplified and stresses too much on the organic causes. Studies found that only 5-10% of children with faltering growth had substantial organic diseases[8,9]. Many other contributing factors such as progression of weaning, feeding difficulties[10], maternal dietary constraint[11] and depression[12], family problems[13], neglect and abuse[14], and behavioural feeding problems like learned food aversionare common among children with faltering growth. A large population study found that weight faltering seen in the first 2 weeks of life was associated with perinatal factors such as preterm birth and maternal smoking, while later onset was associated with organic diseases and feeding problems[15].

Assessments of Infants and Children with Faltering Growth

The locally developed growth charts based on a cross-sectional survey in 1993 is routinely used in Hong Kong for growth assessment[16]. Although WHO advocates the use of universal growth references across different populations[17,18], Hong Kong Chinese toddlers are shorter in general as the epigenetic constraints on growth limit our infants and children to reach their full genetic height potential[19]. The use of WHO growth references thus probably exaggerates the local situation of faltering growth with stunted cases being over-reported. Weight and height/length should be measured accurately at every visit and plotted on the growth charts, with the adjustment for prematurity up to 2-year-old. Plotting the parental height on growth charts at adult age may also be informative for children with slow growth (both weight and height/length centiles are low) to estimate the genetic potential. In affluent societies with a low prevalence of undernutrition, stunting would be more likely due to constitutional factors or organic diseases rather than poor nutrition[20].

Dietary assessment of infants and children with faltering growth is often quite challenging. Only a small portion of them showed dietary intake well below the adequate level, combination of nutritional assessments would be needed to reveal the actual situation. To construct a complete picture of the child's feeding, the dietary assessment should include early feeding history from birth, dietary recall of present intake, mealtime routine and feeding behaviour, the range and types of foods taken, food diaries, and observation(s) of feeding if possible. It should be conducted by dietitians, preferably with specialised training in paediatrics with

proper interviewing and probing skills for infants and childrens' diet. The records of the assessments by paediatricians, clinical psychologists and speech therapists within the multidisciplinary team are also very useful to rule out any possible organic cause of the condition, the possible behavioural feeding problem(s) and the oro-motor function disorders to tailor dietary advices according to individual needs.

Dietetic Management of Faltering Growth

The aims of dietetic management are (i) to improve energy intake; (ii) to promote catch-up optimum growth; (iii) to correct nutritional deficiencies and achieve an adequate nutritional intake; and (iv) to empower and support parents through dietary changes[21]. The ideal weight gain velocity should be individualised to balance between the potential benefits and the deleterious effects of accelerated weight gain (e.g. improved intellectual development[22] versus the increased risk of the cardiovascular disease[23] and obesity[24,25] in the later stage of life).

Dietary advices should be personalised according to the cause, growth status and lifestyle. Usually elevated energy and protein intakes are advised in a short-run for catch-up growth. In general, it could be achieved by having frequent snacks and increasing the energy density of usual foods by fortifying foods with oil and cheese on an interim basis, but it should be taken into account of the food availability and preparation, palatability of foods, as well as the possible effects on delayed gastric emptying and increased regurgitation. The growth velocity should be monitored closely and the caloric and protein prescription should be tailed down towards normal requirements for the age when the growth has improved to avoid imposing unnecessary cardiometabolic risks in the adulthood. Milk intake should be limited according to the local health authority guidelines and increasing number and variety of foods offered usually help to increase solid food intakes. Practise dietary advices on proper weaning, food choices and preparation with recipes, and the possible behavioural modification with simple positive reinforcement strategies are helpful to the parents and caregivers (Fig.1). Although high energy supplement drinks are available in the market, evidence suggests that they do not improve weight gain and may even depress solid food intake[26,27]. The use of dietary supplements is also not recommended for non-organic cases as it may medicalise the problem and the parents may overlook the roles in helping their child to improve nutritional intake[21].

Conclusion

Multidisciplinary efforts from physicians, dietitians, nurses, speech therapists, clinical psychologists and social workers are the key to the early diagnosis of underlying causes, assessment of nutritional status and successful management of infants and children with faltering growth. It is vital to acknowledge the concerns of parents, avoid blame and work with the families to optimise their children.

Fig.1 Possible strategies for increasing energy intake in children aged over 9 months[2]

Dietary:

- Include three meals and two snacks each day
- Increase the number and variety of foods offered
- Increase energy density of usual foods, such as adding cheese, margarine or cream
- Limit milk intake to 500 ml per day†
- Avoid excessive intake of fruit juice and squash‡

Behavioural:

- Offer meals at regular times with other family members.
- Praise when food is eaten, but ignore when not.
- Limit a meal's time to 30 minutes.
- Parents should eat at the same time as the child.
- Mealtime conflict should be avoided.
- The child should never be force-fed.

† Family Health Service of the Department of Health recommends milk intake of 360-480ml per day for young children over 1 year-old

‡ It may also apply to the soup or other low energy-density drinks

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Dermatological Quiz

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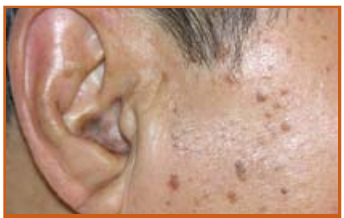

Fig. 1a: Multiple brownish warty papules over the cheek

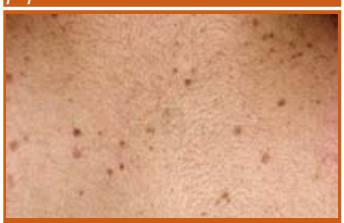

Fig. 1b: Numerous similar lesions over the trunk

This middle aged man developed multiple asymptomatic pigmented skin lesions over his face (Fig.1a) and trunk (Fig.1b). These brownish warty papules gradually increased in number. He was told by a beautician in a salon and also reassured by a doctor next door that these were plane warts. He was told that these were contagious and should be removed. His past health was good. His deceased father also had a similar condition at an advanced age.

Questions:

1. What are your diagnosis and main differential diagnosis?
2. How do you differentiate between the two?
3. What is another common reason for referral to a dermatologist if the lesion increases in size and pigmentation?
4. How do you treat this condition?

(See P.32 for answers)

Puberty and Pubertal Disorders

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Puberty is the transition from childhood to adulthood, with development of secondary sexual characteristics, in association with a growth spurt and acquisition of maturation of reproductive potential. The period usually lasts for 3-4 years. The onset of puberty is heralded with an increase in the nocturnal pulsatile secretion of gonadotrophin releasing hormone from the hypothalamus, resulting in a series of hormonal and physical changes[1]. However, the exact trigger for such pulsatile secretion is still unknown.

While there were a plethora of reports on the secular trends in the normal age of onset of puberty in boys and girls across different populations of the developed countries documenting earlier onset of puberty, the sequence and tempo of pubertal events remained largely unchanged from what was described by Marshall and Tanner in the 1960s.[2,3] Breast development and testicular enlargement are the first signs, followed by a defined pattern of secondary sexual characteristics together with maturation of the sexual organs and reproductive potential. Conventional definition of abnormal early puberty is the presence of the first sign of puberty by 8 years in girls and 9 years in boys, while delayed puberty by no breast development by 13 year in girls and no testicular enlargement by 14 year in boys.

Precocious puberty:

The incidence of precocious puberty is estimated to be between 1:5000 and 1:10000, with female: male ratio of between 3:1 to 23:1. The 1993 Hong Kong Growth Study recorded the progression of puberty from cross sectional observations in about 7500 students.[4,5] Stage II breast development was observed in 3% of girls at 7.1 yr and 10% of girls at 8 years had entered puberty by definition, which implied that over-investigation and unnecessary treatment might happen if precocity was cut off at 8 year.[4]

Classification of Precocious Puberty:

Table 1: [6] Four groups of premature sexual development:

1.	Premature activation of the hypothalamus-pituitary-gonadal axis or central precocious puberty (CPP) or Gonadotrophin Dependent Precocious Puberty (GDPP)
2.	Abnormal patterns of gonadotrophin secretion (premature thelarche, thelarche variant)
3.	Excess adrenal androgens (premature adrenarche, congenital adrenal hyperplasia, adrenal tumours)
4.	Secretion of sex steroids independent of the HPG axis, or Gonadotrophin Independent Precocious Puberty (GIPP)

The purposes of evaluation in premature onset of puberty are : (i) to distinguish among these various possibilities, (ii) to identify the underlying causes if possible, (iii) to estimate the effects on physical growth and psychosocial adjustment and finally (iv) to determine if treatment would be beneficial or not.

Investigations of precocious puberty:

Hormonal tests:

Because of the pulsatile nature of gonadotrophin secretion, a single random level is not usually helpful to differentiate the precocious from the prepubertal range. Serial measurements after challenge with Gonadotrophin Releasing Hormone is necessary to diagnose CPP. An elevated baseline LH > 0.3 IU/L as measured by ultra-sensitive assay (immuno-chemiluminometric)[7], exaggerated elevation of LH compared to baseline after stimulation or LH/FSH ratio more than 1 is suggestive of pubertal response.

Imaging:

Pelvic ultrasonic examination of ovary and uterus would provide information on the sexual organ maturation and adrenal pathology. The cut – off length for uterine length ranges from 3.4 to 4.0 cm, the presence of an endometrial echo is highly specific but less sensitive. The cut-off for a pubertal ovarian volume ranges between 1 and 3 ml.[8]

X ray of the left hand is usually taken to compare the bone age with the chronological age. Adult height prediction by the Tanner and Whitehouse Method (version 3) would help to determine the effects on subsequent growth. To rule out cranial pathology, all boys with CPP and girls with CPP presenting at < 6 years of age should have a cranial MRI.[9]

Treatment of Precocious Puberty

Current treatment of CPP is by monthly injection of Gonadotrophin Releasing Hormone Agonist (GnRHa). The drug has been commercially available since the 1980s, firstly as a daily intranasal spray, subsequently through the daily subcutaneous route and finally by monthly depot intramuscular injections. Only those showing progressive pubertal development and growth acceleration should be treated. In general, girls with onset of puberty before 6 years, treatment would result in an average gain of 9 – 10 cm while those with onset between 6 and 8 years have variable benefits ranging from 4.5 +/- 5.8 cm to 7.2 +/- 5.3 cm.[9] Those with non-progressive early onset of puberty usually have less improvement in adult height.

Treatment should be considered for all boys with onset of puberty before 9 years of age. Body proportions among untreated children with precocity are affected by early skeletal maturation of long bones, resulting in progressively shorter arms and legs in relation to the total height[10]. With treatment, these proportions could be normalised[10]. The dosage of GnRHa in the treatment of CPP ranges from 3.75 mg to 15 mg monthly. In order to reduce the burden of injection and clinic visit, three-monthly preparations were available. A recent randomised trial had demonstrated efficacy of the three-monthly preparations, but a higher dosing level may be necessary to achieve adequate LH suppression.[11,12]

GnRHas are well tolerated apart from local tenderness on injection. Bone mineralisation and body fat compositions may be adversely affected on prolonged treatment and should be monitored during the treatment period. Follow-up of treated or untreated girls with CPP into the mid-teenage years suggests that the development of polycystic ovary morphology is not clearly different from that in the general population[9]. A longitudinal case-control study from Italy demonstrated that GnRHa treatment might be an independent risk for development of Polycystic Ovary Syndrome in adolescence.[13]

The age of discontinuation of GnRHa therapy is arbitrary, and should be decided jointly with the parents and the patient.[14] Typically it would be continued until the normal age of puberty. Menarche would occur in 9 – 18 months after stopping treatment. Gonadal function is not impaired in girls treated with GnRHas Combined treatment with Human Growth Hormone and GnRHas in order to improve the final adult height by delaying puberty had been experimented with no definite benefits demonstrated. An on-going clinical trial from the Belgian Study Group for Pediatric Endocrinology focusing on the Efficacy and Safety of a 4 Year Combination Therapy of Growth Hormone and Gonadotropin- Releasing Hormone Agonist in Children with a short predicted height (boys with a bone age between 11 and 13 years and a predicted adult height below 163 cm or girls in early puberty with a bone age between 10 and 12 years and a predicted height under 151 cm) had just completed the case recruitment, the result of this study would be available in 2017.

Delayed Puberty

Delayed puberty was defined by the absence of breast development by 13 years, or absence of menarche by 16 years in girls and absence of testicular enlargement by age 14 years in boys, or failure of appropriate progression of secondary sexual development in both sex.[14]

Table 2: [15]

Pubertal delay
Constitutional growth and pubertal delay (CGDP)
Delayed secondary to chronic illness
Hypogonadotrophic hypogonadism
Defect in the hypothalamopituitary region
Secondary to radiotherapy/chemotherapy
Hypergonadotrophic hypogonadism
Secondary to gonadal failure (Turner's syndrome)
Secondary to radiotherapy/chemotherapy

Delayed puberty is more common in boys, with the great majority of cases due to simple delay in growth and onset of puberty (CGDP). A history and initial investigations will help to aim to identify those with pathological conditions involving the hypothalamus or pituitary function presenting with low levels of LH and FSH, or those with primary gonadal dysfunction with LH and FSH way above the normal range. Further investigations include bone age assessment, chromosome / genetic study, other pituitary hormone secretion, general health status including thyroid function.

Treatment is often necessary in delayed puberty, especially in boys, with hormonal induction for spontaneous onset of puberty to hormonal replacement therapy, in order to foster appropriate physical growth, secondary sexual characteristics development, cardiovascular and bone health as well as psychological well being.

Conclusion:

Puberty is hormonally controlled but it is also affected by various genetics and environmental factors. Disorders in puberty can often be diagnosed after a careful history, physical examination and simple investigations including growth charts or bone age. In both early or delay in puberty, appropriate referrals for assessments are important as effective treatments are available to improve or alter the abnormal progression of puberty for the benefits of the affected children.

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"Travel Health Advices for Cruise Travellers & Cruise Seminar"

On 24 Mar 2014, a free seminar on "Travel Health Advices for Cruise Travellers & Cruise Seminar" was held at the FMSHK Lecture Hall. The Federation was glad to have Dr Pang-yung FAN, Founder Member of the Faculty of Travel Medicine, RCPSG, UK to deliver a talk on travel medicine. Dr Fan updated the latest on pre-travel vaccination, medication and international requirements. Another talk was given by Mrs Nancy CHUNG, Asia Regional Director of Carnival Corporation HK Ltd on cruise travel, covering the cruise history, and the selected luxury itineraries and life style on board. A short quiz with lovely gifts was also held at the end of the lectures. As cruise holiday is becoming a hot trend for holidays, the Federation is delighted to organise this seminar for our professionals, with delivery of the necessary health information and precaution in travelling abroad, especially in the prevention of vector-borne diseases, the theme of World Health Day 2014.

Queen Elizabeth Ship Visit

The Queen Elizabeth Ship Visit was successfully held on 28 Mar 2014. We were privileged to have friends of the Federation & Foundation, Presidents of Member Societies and BMA(HK) members to join us on board. The participants had an enjoyable evening with a comprehensive ship tour and fine dining in the exquisite Verandah restaurant. Our President, Dr. Raymond LO marked the opening with a warm welcome speech. The talk from our Hon. Secretary, Dr. Mario CHAK updated our members and guests on recent activities of our bereaved children charity project and other coming activities. The event was enriched with a short singing performance by Ms Annabel CHOY with her sweet angelic voice. We also thanked Dr Sek-hong CHEUNG for capturing the joyous moment for us.

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Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
				1	★ Joint Surgical Symposium – Breast surgery 2	3
4	★ HKMA Council Meeting ★ Monthly meeting of Hong Kong Urological Association 5	6	7	★ HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2014 – Injuries in Hiking 8	★ HKMA Shatin Doctors Network – Advancement of Meningococcal Conjugate Vaccine & Global Practice 9	★ HKMA CME – Refresher Course for Health Care Providers 2013/2014 10
11	12	★ HKMA Yau Tsim Mong Community Network – Acne Management Review ★ HKMA Kowloon West Community Network – Sarcopenia in Elderly ★ HKMA Tai Po Community Network – Importance of Muscle Training for Adults ★ Annual Scientific Meeting 2014 of the Hong Kong Surgical Laser Association cum Joint Scientific Meeting of The Hong Kong Surgical Laser Association and The Hong Kong Medical Association ★ FMSHK Officers' Meeting 13	★ Hong Kong Neurosurgical Society Monthly Academic Meeting ★ HKMA Central, Western & Southern Community Network 14	★ HKMA Kowloon East Community Network & United Christian Hospital – Certificate Course for GPs 2014 – Update on Hypertension Management ★ HKMA Hong Kong East Community Network – Do Patient Characteristics Influence Choice of DPP-4 Inhibitor? ★ HKMA New Territories West Community Network – Update on Management of Diabetic Nephropathy 15	16	17
18	19	20	★ Medical Exchange Tour in Yunnan ★ HKMA Shatin Doctors Network – Update on Invasive Meningococcal Disease and Prevention 21	★ Medical Exchange Tour in Yunnan ★ HKMA Kowloon City Community Network – Diagnosis and Treatment of Axial-Spondyloarthropathy (Axial-SpA) ★ FMSHK Executive Committee and Council Meeting ★ HKMA Kowloon East Community Network - Updates on the Management of Non-Alcoholic Fatty Liver Disease (NAFLD) 22	★ Medical Exchange Tour in Yunnan ★ HKMA Shatin Doctors Network - New Era of Diagnosis and Treatment of Rheumatoid Arthritis 23	★ Medical Exchange Tour in Yunnan 24
★ Medical Exchange Tour in Yunnan 25	26	★ HKMA Kowloon West Community Network - Managing Common Gastrointestinal (GI) Disturbance in Infants and Young Children ★ HKMA Tai Po Community Network - The Importance of Overall Efficacy and Cross Protection of Cervical Cancer Vaccines 27	★ HKMA Central, Western & Southern Community Network - Update in Acne Treatment 28	★ HKMA New Territories West Community Network - Treatment and Prevention of Pneumococcal Disease for Elderly Patient 29	30	31

Date / Time	Function	Enquiry / Remarks
2 FRI 8:00am	**Joint Surgical Symposium - Breast Surgery** Organisers: Department of Surgery & The University of Hong Kong & Hong Kong Sanatorium & Hospital, Chairman: Dr. CHAN Yu-Wai, Speakers: Dr. Polly CHEUNG & Dr. Dacita SUEN, Venue: Hong Kong Sanatorium & Hospital	Department of Surgery, Hong Kong Sanatorium & Hospital Tel: 2835 8698 1 CME Point
5 MON 8:00am	**HKMA Council Meeting** Organiser: The Hong Kong Medical Association, Chairman: Dr. TSE Hung Hing, Venue: HKMA Head Office (5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong)	Ms. Christine WONG Tel: 2527 8285
7:30pm	**Monthly meeting of Hong Kong Urological Association** Organizer: Hong Kong Urological Association, Chairman: Dr. Sidney YIP, Speakers: Dr Raymond KAN, Dr Ringo CHU, Dr Ngan Ho-yin, Dr CHENG Cheung-hing, Dr Edmond WONG, Venue: Multi-disciplinary Simulation and Skills Centre, 4/F, Block F, QEH	Ms. Tammy HUNG Tel: 96096064 1 CME Point
8 THU 2:00pm	**HKMA Structured CME Programme with Hong Kong Sanatorium & Hospital Year 2014 – Injuries in Hiking** Organiser: Hong Kong Medical Association & Hong Kong Sanatorium & Hospital, Speaker: Dr. Jimmy Wai Kwok WONG, Venue: HKMA Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	HKMA CME Dept. Tel: 2527 8452 1 CME Point
9 FRI 1:00pm	**HKMA Shatin Doctors Network – Advancement of Meningococcal Conjugate Vaccine & Global Practice** Organiser: HKMA Shatin Doctors Network, Chairman: Dr. MAK Wing Kin, Speaker: Dr. CHAN Keung Kit, Venue: Jasmine Room, Level 2, Royal Park Hotel, 8 Pak Hok Ting Street, Shatin	Ms. Vinki CHEUNG Tel: 3189 8734 1 CME Point
10 SAT 2:15pm	**HKMA CME – Refresher Course for Health Care Providers 2013/2014** Organiser: Hong Kong Medical Association, HK College of Family Physicians & HA-Our Lady of Maryknoll Hospital, Speaker: Ms. KWAN Yee Mei, Venue: Training Room II, 1/F, OPD Block, Our Lady of Maryknoll Hospital, 118 Shatin Pass Road, Wong Tai Sin, Kowloon	Ms. Clara TSANG Tel: 2354 2440 2 CME Point
13 TUE 1:00pm	**HKMA Yau Tsim Mong Community Network – Acne Management Review** Organiser: HKMA Yau Tsim Mong Community Network, Chairman: Dr. LAM Yick Wang, Clement, Speaker: Dr. CHUNG Chun Kin, Alex, Venue: Pearl Ballroom, Level 2, Eaton, Hong Kong, 380 Nathan Road, Kowloon	Ms. Candice TONG Tel: 2527 8285
1:00pm	**HKMA Kowloon West Community Network – Sarcopenia in Elderly** Organiser: HKMA Kowloon West Community Network, Chairman: Dr. CHAN Ching Pong, Speaker: Dr. YIP Wai Man, Venue: Crystal Room I-III, 30/F, Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, NT	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
1:45pm	**HKMA Tai Po Community Network – Importance of Muscle Training for Adults** Organiser: HKMA Tai Po Community Network, Speaker: Dr. CHAN Hoi Chung, Samuel, Venue: Chiuchow Garden Restaurant (潮江春) Shop 001-003, 1/F, Uptown Plaza (新達廣場), No.9 Nam Wan Road, Tai Po	Ms. Kate NG Tel: 6323 7932 1 CME Point
7:00pm	**Annual Scientific Meeting 2014 of the Hong Kong Surgical Laser Association cum Joint Scientific Meeting of The Hong Kong Surgical Laser Association and The Hong Kong Medical Association** Organisers: Hong Kong Medical Association & HK Surgical Laser Assn, Speaker: Dr. TSE Tak On; Dr. FUNG Ming Kit; Dr. YEUNG Chun Chun, Venue: Shanghai Room 8/F, Langham Place Hotel, Mongkok.	Ms. Jacqueline SHUM Tel: 2632 2879 1 CME Point
8:00pm	**FMSHK Officers' Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Gallop, 2/F., Hong Kong Jockey Club Club House, Shan Kwong Road, Happy Valley, Hong Kong	Ms Nancy CHAN Tel: 2527 8898
14 WED 7:30am	**Hong Kong Neurosurgical Society Monthly Academic Meeting –Primary central nervous system lymphoma : current treatment strategies** Organiser: Hong Kong Neurosurgical Society, Speaker: Dr TSE Po Ki, Teresa, Chairman: Dr CHAN Kam Tong, Tony, Venue: Seminar Room, Ground Floor, Block A, Queen Elizabeth Hospital	Dr. LEE Wing Yan, Michael Tel: 2595 6456 1.5 CME Point
1:00pm	**HKMA Central, Western & Southern Community Network – Practical Consideration in the Use of Novel Oral Anticoagulants (NOACs) in Stroke Prevention in AF Patients** Organiser: HKMA Central, Western & Southern Community Network, Chairman: Dr. POON Man Kay, Speaker: Dr. LI Shu Kin, Venue: HKMA Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
15 THU 1:00pm	**HKMA Kowloon East Community Network & United Christian Hospital – Certificate Course for GPs 2014 – Update on Hypertension Management** Organiser: HKMA Kowloon East Community Network & United Christian Hospital, Chairman: Dr. Danny MA, Speaker: Dr. LEUNG Kwok Fai, Venue: East Ocean Seafood Restaurant (東海海鮮酒家), Shop 137, 1/F, Metro City Plaza 3, 8 Mau Yip Road, Tseung Kwan O, Kowloon	Ms. Polly TAI /Ms. Cordy WONG Tel: 3513 3430 / 3513 3087 Fax: 3513 5505 1 CME Point
1:00pm	**HKMA Hong Kong East Community Network – Do Patient Characteristics Influence Choice of DPP-4 Inhibitor?** Organiser: HKMA Hong Kong East Community Network, Chairman: Dr. KONG Wing Ming, Henry, Speaker: Dr. IP Tai Pang, Venue: HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Hong Kong	Ms. Candice TONG Tel: 2527 8285 1 CME Point
1:00pm	**HKMA New Territories West Community Network – Update on Management of Diabetic Nephropathy** Organiser: HKMA New Territories West Community Network, Chairman: Dr. CHAN Lam Fung, Lambert, Speaker: Dr. LEE Hoi Kan, Achilles, Venue: Plentiful Delight Banquet (元朗喜尚嘉喜酒家), 1/F., Ho Shun Tai Building, 10 Sai Ching Street, Yuen Long	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
21 WED 7:30am (22-25)	**Medical Exchange Tour in Yunnan** Organiser: HKMA Youth Committee, Chairman: Dr. LAM Tzit Yuen, David Dr. PONG Chiu Fai, Jeffery.	Miss Phoebe WONG Tel: 2527 8285
1:00pm	**HKMA Shatin Doctors Network – Update on Invasive Meningococcal Disease and Prevention** Organiser: HKMA Shatin Doctors Network, Chairman: Dr. MAK Wing Kin, Speaker: Dr. LEE Cheuk Hon, Venue: Jasmine Room, Level 2, Royal Park Hotel, 8 Pak Hok Ting Street, Shatin	Ms. Jude LEUNG Tel: 2506 8345 1 CME Point
22 THU 1:00pm	**HKMA Kowloon City Community Network – Diagnosis and Treatment of Axial-Spondyloarthropathy (Axial-SpA)** Organiser: HKMA Kowloon City Community Network, Chairman: Dr. CHIN Chu Wah, Speaker: Dr. SUNG Chi Keung, Venue: Spotlight Recreation Club (博藝會), 4/F., Screen World,Site 8, Whampoa Garden, Hunghom, Kowloon	Ms. Candice TONG Tel: 2527 8285

Date / Time	Function	Enquiry / Remarks
22 THU 1:00pm	**HKMA Kowloon East Community Network - Updates on the Management of Non-Alcoholic Fatty Liver Disease (NAFLD)** Organiser: HKMA Kowloon East Community Network, Chairman: Dr. MA Ping Kwan, Danny, Speaker: Dr. LAI Sik To, Thomas, Venue: East Ocean Seafood Restaurant (東海海鮮酒家), Shop 137, 1/F, Metro City Plaza 3, 8 Mau Yip Road, Tseung Kwan O, Kowloon	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
7:00pm	**FMSHK Executive Committee and Council Meeting** Organiser: The Federation of Medical Societies of Hong Kong, Venue: Council Chamber, 4/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	Ms Nancy CHAN Tel: 2527 8898
23 FRI 1:00pm	**HKMA Shatin Doctors Network - New Era of Diagnosis and Treatment of Rheumatoid Arthritis** Organiser: HKMA Shatin Doctors Network, Chairman: Dr. MAK Wing Kin, Speaker: Prof. TAM Lai Shan, Venue: Jasmine Room, Level 2, Royal Park Hotel, 8 Pak Hok Ting Street, Shatin	Ms. Zoe CHAN Fax: 2219 7397 1 CME Point
27 TUE 1:00pm	**HKMA Kowloon West Community Network - Managing Common Gastrointestinal (GI) Disturbance in Infants and Young Children** Organiser: HKMA Kowloon West Community Network, Chairman: Dr. LEUNG Kin Nin, Kenneth, Speaker: Dr. CHOW Wing Cheong, Venue: Crystal Room I-III, 30/F, Panda Hotel, 3 Tsuen Wah Street, Tsuen Wan, NT	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
1:00pm	**HKMA Tai Po Community Network - The Importance of Overall Efficacy and Cross Protection of Cervical Cancer Vaccines** Organiser: HKMA Tai Po Community Network, Speaker: Dr. WONG To, Venue: Chiuchow Garden Restaurant (潮江春) Shop 001-003, 1/F, Uptown Plaza (新達廣場), No.9 Nam Wan Road, Tai Po	Ms Yvonne YEUNG Tel: 3189 8626 1 CME Point
28 WED 1:00pm	**HKMA Central, Western & Southern Community Network - Update in Acne Treatment** Organiser: HKMA Central, Western & Southern Community Network, Chairman: Dr. YIK Ping Yin, Speaker: Dr. CHAN Pui Yiu, Nicola, Venue: HKMA Central Premises, Dr. Li Shu Pui Professional Education Centre, 2/F, Chinese Club Building, 21-22 Connaught Road, Central, Hong Kong	Miss Hana YEUNG Tel: 2527 8285 1 CME Point
29 THU 1:00pm	**HKMA New Territories West Community Network - Treatment and Prevention of Pneumococcal Disease for Elderly Patient** Organiser: HKMA New Territories West Community Network, Chairman: Dr. CHAN Siu Chung, Speaker: Dr. TAI Kian Bun, Venue: Plentiful Delight Banquet (元朗喜尚嘉喜酒家), 1/F., Ho Shun Tai Building, 10 Sai Ching Street, Yuen Long	Miss Hana YEUNG Tel: 2527 8285 1 CME Point

Upcoming Meeting

28-30/6/2014	**4th IDKD Intensive Course in Hong Kong "Musculoskeletal Diseases"** Organiser: IDKD, HKU & HKCR, Venue: Hong Kong Convention & Exhibition Centre (HKCEC), 1 Expo Drive, Wanchai, Registration: www.idkd.org

香港鐳射醫學會
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Annual General Meeting 2014 of the Hong Kong Surgical Laser Association cum Joint Scientific Meeting of the Hong Kong Surgical Laser Association and The Hong Kong Medical Association

香港醫學會
THE HONG KONG MEDICAL ASSOCIATION

Date : **13 May 2014 Tuesday**

Venue : **Shanghai Room, Level 8, Langham Place Hotel, Mongkok**

7:00 pm : **Annual General Meeting 2014**

7:30 pm : **Scientific Meeting**

Topics : **1. Lasers in Contemporary Esthetic and implant dentistry**
Dr Tse Tak On (Bachelor of Dental Surgery)
2. Treatment of varicose veins in Hong Kong after 2003
Dr Fung Ming Kit (Specialist in General surgery)
3. Laser in Ophthalmology
Dr Yeung Chun Chun, Jane (Specialist in Ophthalmology)

8:45 pm : **Dinner**

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Answers to Dermatological Quiz

1. Seborrhoeic keratosis. The main differential diagnosis is plane wart.

   In Hong Kong, this condition is now commonly misdiagnosed as plane warts by beauticians in beauty salons and also by doctors (including some dermatologists). Seborrhoeic keratosis has multiple confusing names like seborrhoeic wart, "Longevous mole" or "Old aged mole" among Chinese. These were misnomers as they are neither viral warts nor melanocytic naevi. In dermatology, other names include keratosis pigmentosa, verruca senilis and dermatosis papulosis nigra in Black.

   Although seborrhoeic keratosis is the most common benign tumour in old individuals, it is also common in the middle-aged, especially in those who have multiple lesions with autosomally dominant mode of inheritance. The sign of Leser-Trélat refers to the sudden eruption of multiple seborrhoeic keratoses associated with internal malignancy. However, the validity of this sign has been challenged and its existence is really doubtful.

2. Seborrhoeic keratoses have a "stuck-on" appearance, hyperkeratotic surface, and usually have a light brown to deep black pigmentation. They can occur at almost any site of the body, with the exception of the palms and soles and mucous membranes. They usually occur in the elderly or middle-aged. Plane warts (also known as flat warts, verruca plana) typically have flat or slightly elevated flesh-coloured papules that may be smooth, arranged in a grouped, confluent pattern or in a linear distribution after scratching or injury (Koebner phenomenon). The face, hands, and shins are the common areas. They usually occur in children or adolescents. Both of them can vary from a few to numerous in number. In case of difficulties in diagnosis, a skin biopsy will differentiate between the two, though in practice this is seldom done.

3. Seborrhoeic keratosis has a variety of clinical appearances which vary greatly in pigmentation, thickness, size and number. Sometimes individual lesions can grow rapidly to an alerting size and become deeply pigmented. In such circumstance, the patient is often referred to a dermatologist with a suspicion of malignant melanoma or pigmented basal cell carcinoma. In case of doubt, a skin biopsy should be done.

4. Seborrhoeic keratosis will not transform into malignancy. In asymptomatic elderly patients, they should be reassured of its benign nature and the lesion can be left untreated. If treatment is needed, it can easily removed by cauterisation and curettage, shave excision, cryotherapy or laser surgery.

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THE FEDERATION OF MEDICAL SOCIETIES OF HONG KONG
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Annual Scientific Meeting 2014

Care for Our Older Population

Date: 1 June 2014 (Sunday) Time: 09:30 AM – 09:00 PM
Venue: Ballroom, 3rd Floor & Sung room I-II, 4th Floor
Sheraton Hotel, 20 Nathan Road, Tsim Sha Tsui

Opening Ceremony

• Plenary Session

Elderly Population - Challenges and Opportunities
Mr. Richard YUEN, JP *Permanent Secretary for Food and Health (Health)*

中國國內老人醫學發展
李小鷹教授 中華醫學會老年病學分會主任委員

• Session I: New Guidelines on Cardiovascular Diseases and Diabetes in Applications for Older People

Update on Hypertension: Blood Pressure Goal and Management of Refractory Hypertension
Prof. Chu-pak LAU *Past President, The Hong Kong College of Cardiology*

Risk Stratification and Personalized Care in Diabetes
Prof. Juliana CHAN *Professor, Department of Medicine & Therapeutics, The Chinese University of Hong Kong*

How to Make Sense of the New Cholesterol Guidelines
Prof. Kathryn TAN *Sir David Todd Professorship in Medicine, Department of Medicine, The University of Hong Kong*

• Session II: Luncheon Symposium on Vaccination

How can we Prevent Herpes Zoster and its Complications
Dr. Thomas SO *Executive Committee Member, The Federation of Medical Societies of Hong Kong*

• Session III: Advance in Surgical Operations in Older People

Title to be confirmed
鄭民華教授 上海瑞金醫院

Urological Problem in Older People
Dr. Chi-wai MAN *Executive Committee Member, The Federation of Medical Societies of Hong Kong*

• Session IVa: Geriatric Stroke

Hyperacute Treatment of Ischemic Stroke in Geriatric Patient
Dr. Mang-ho YUEN *Specialist in Neurosurgery*

Geriatric Stroke - a Surgical Perspective
Dr. Dawson FONG *Specialist in Neurosurgery*

Update on Endovascular Treatment of Stroke
Dr. Pui-wai CHENG *Radiologist-in-charge, Scanning Department of St. Teresa's Hospital*

• Session IVb: Geriatric Diseases

Common Retinal Diseases in the Elderly and Recent Advances in Management
Dr. Vincent LEE *President, The Hong Kong Ophthalmological Society*

Hearing Problems in the Older Population
Prof. Michael TONG *Chairman, Hear Talk Foundation*

Functional Rehabilitation of the Edentulous Elderly with Dental Implants
Dr. Philip LEE *Specialist in Oral and Maxillofacial Surgery*

• Session Va: 3Ds - Dementia, Depression and Delirium

Diagnosis and Assessment of Dementia in the Community
Prof. Linda LAM *Professor and Chairman, Department of Psychiatry, Faculty of Medicine, The Chinese University of Hong Kong*

Recent Advances in the Management of Late Life Depression
Dr. Wai-chi CHAN *Clinical Associate Professor, Department of Psychiatry, The University of Hong Kong*

Detection and Management of Delirium
Prof. Timothy KWOK *Professor, Department of Medicine and Therapeutics, The Chinese University of Hong Kong*

• Session Vb: Ageing - Related Aesthetic Medicine

Skin Ageing and The Use of Botulinum Toxin
Dr. Kingsley CHAN *Executive Committee Member, The Federation of Medical Societies of Hong Kong*

Management of Skin Pigmentation in Elderly: Diagnosis and Laser Applications
Dr. Lai-shan CHIU *Specialist in Dermatology and Venereology*

Tissue Filler : Which to Use
Dr. Daniel LEE *President, Association for Integrative Aesthetic Medicine, Hong Kong Limited*

• Session VI: Round Table Discussion

安老服務及院舍於香港的未來發展
Future Development on Elderly Services and Residential Care Homes in Hong Kong
Venue: Sung Room I-II, 4th Floor, Sheraton Hotel
Language: Mandarin

• Session VII: Joint Dinner Symposium Visual Impairment and Falls in Older People

Time: 07:00 PM- 09:00 PM
Venue: Sung Room I-II, 4th Floor, Sheraton Hotel

Organizers:
- The Federation of Medical Societies of Hong Kong
- The College of Ophthalmologists of Hong Kong
- The Hong Kong Ophthalmological Society

Cataract Surgeries - What are the New Challenges?
Dr. CHOW Pak Chin *President, The College of Ophthalmologists of Hong Kong*

Advances in Diagnosis and Management of Glaucoma
Dr. Nancy YUEN *Vice-President, The College of Ophthalmologists of Hong Kong*

Fall Preventions in Older People
Dr. Raymond LO *President, The Federation of Medical Societies of Hong Kong*

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HK$200 Non-member
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First come first served

Registration

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Remarks: No CME/CPD/CNE points will be obtained if applicants join Session VI: Round Table Discussion only
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