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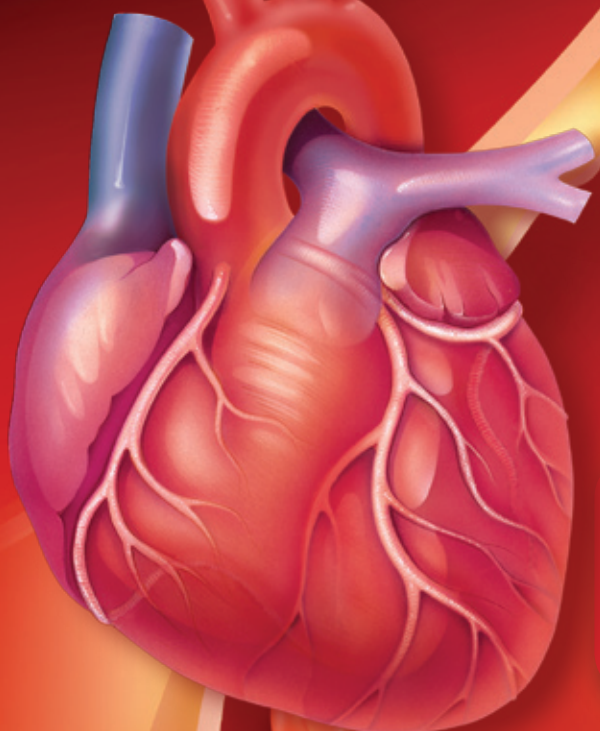
Cardiology – Treats and Tricks

- Update on Management of Heart Failure, *Dr Chu-Pak Lau (劉柱柏醫生)*
- Approaches to Carotid Stenosis, *Dr SL Li (李少隆醫生)*
- Utilizing Cardiac CT for the Detection of Coronary Artery Disease, *Dr Jennifer MF Kwok (郭妙芳醫生)*
- Heart Disease Does not Equate Coronary Artery Disease, *Dr CM Tam (譚志明醫生)*



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For the 30 days following the conclusion of treatment period (Day 1470) when, according to protocol, patients were discontinued from their study medication, the study revealed an 11% reduced risk reduction (p=0.086, intention-to-treat analysis).

References: 1. Tashkin DP, Celli B, Senn S, et al, on behalf of UPLIFT® study investigators. A 4-Year Trial of Tiotropium in Chronic Obstructive Pulmonary Disease. N Engl J Med. Vol 359;15: 1543-1554. 2. Global Initiative for COPD. Global strategy for the diagnosis, management, and prevention of COPD: executive summary. Updated 2007. http://www.goldcopd.com. Accessed September 5, 2008. 3. Data on file. Boehringer Ingelheim International GmbH; 2008.

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CONTENTS

Editorial

The Society of Physicians of Hong Kong has been established for over 50 years. It is one of the medical societies in Hong Kong with the longest history. In early years, it started as a social network for doctors in private practice.

In recent years, the Society has evolved rapidly into a learned professional body that conducts high level scientific meetings for the exchange and dissemination of medical knowledge. The Society is now one of the major providers of continuing medical education for doctors in Hong Kong. We have also seen growth of its membership to include many leading and distinguished members of the profession. We hope the Journal will provide a further platform for the promotion and exchange of medical knowledge, and will foster a closer link between the Society and the medical community in Hong Kong and overseas.

We want to thank all those who have contributed to bringing out the first issue successfully, especially the issue's editors Dr Cathy Lam (林紫芬醫生) and Dr Steven Li (李少隆醫生). The pioneers always work hardest so that those who follow will have an easier job.

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FHKAM (Psychiatry)
President

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FRCP, MD, FHKAM (Medicine)
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Update on Management of Heart Failure

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Chief Editor, Society of Physicians of Hong Kong

Table 1. New York Heart Association classification of dysponea due to heart disease

Class I	Heart disease but asymptomatic
Class II	Dyspnoea on moderate to severe exertion
Class III	Dyspnoea on mild exertion
Class IV	Dyspnoea at rest
↓ survival with a higher functional class (30% mortality in 1 year for class IV, 40% will die within 1 year after hospitalization)	

Heart failure (HF) is a progressive disease with significant mortality and morbidity, and its prevalence increases with age. For example, in patients with severe HF, the 1-year mortality despite optimal medical treatment (including beta-blockers, angiotensin-converting enzyme [ACE] inhibitors, and spironolactone) is about 30%, which is higher than that of some cancers. Thus, the goals of treatment are to reduce mortality, progression and complications, avoid hospitalization, and improve symptoms and quality of life.

The main complaint of a patient with HF is dyspnoea. It is important to assess its severity using the New York Heart Association (NYHA) classification. (Table 1) Signs such as elevated jugular venous pressure, ankle oedema and bilateral basal crackles may be present. Where in doubt, B-type natriuretic peptide (BNP) assay is a good method to exclude HF. A noninvasive test such as echocardiography is commonly needed to assess the severity of systolic dysfunction, or to make a diagnosis of HF with preserved ejection fraction (EF).

The treatment of HF starts with identifying reversible causes, such as coronary artery disease (CAD) and hypertension. Avoidance of cardiac risk factors and excessive salt and water consumption is essential. Commonly, patients are asked to keep an ideal dry weight, and either to increase or decrease the use of diuretics if body weight changes by more than 2 lb in 2 consecutive days. In patients with systolic dysfunction, medical treatment will in general be guided by the NYHA class. (Table 2) An ACE inhibitor is indicated in those with asymptomatic left ventricular dysfunction. In more advanced classes, after optimization of ACE inhibitor, a beta-blocker is added if there is no contraindication, and spironolactone is used for those with the worse NYHA class. In patients with sinus rhythm, digoxin may improve exercise capacity and reduce hospitalization, but not mortality. In patients with cough associated with ACE inhibitors, an angiotensin receptor blocker (ARB) is a good substitute, but it is important to note that only valsartan and candesartan have been shown useful for the treatment of HF. Regular assessment and patient education are essential for the early detection and prevention of complications (eg, hospitalization, atrial fibrillation and thromboembolism).

Sudden cardiac death occurs in up to one third of HF patients, and primary prevention of sudden death should be advocated in those with an EF <35%. Cardiac resynchronization therapy (ie, biventricular pacing) will improve the structure and function of the ventricle with left bundle branch block on electrocardiogram.

Finally, the treatment of HF is difficult and expensive. Prevention of risk factors will reduce the population at risk. Early recognition and management of hypertension, diabetes, cigarette smoking and CAD are critical in this approach. An ACE inhibitor or ARB may be useful for early cardiovascular protection.

Table 2. Treatment of systolic heart failure

Drug	NYHA class	Trials	Remarks
ACE inhibitors	II-IV	CONSENSUS, SOLVD, SAVE, etc	Captopril, enalapril, lisinopril, ramipril, trandolopril, perindopril
Diuretics	II-IV	--	Potassium-sparing diuretics increase arrhythmic death
Aldosterone antagonists	III-IV	RALES, EPHESUS	Spironolactone Eplerenone (post-MI)
Digoxin	II-III	DIG Study	AF – indicated SR – ↓ hospitalization but not mortality
Beta-blockers	I-IV	CIBIS II, MERIT-HF, US Carvedilol, COPERNICUS, COMET	Metoprolol controlled release Bisoprolol Carvedilol
ARB	II-III	Alternative VALIANT, CHARM-alternative Added VAL-HeFT, CHARM-added	Valsartan vs captopril (post-MI) Candesartan vs ACE inhibitors Valsartan (up to 320 mg) Candesartan (up to 32 mg)

ACE = angiotensin-converting enzyme; AF = atrial fibrilliation; CIBIS II = Cardiac Insufficiency Bisoprolol Study II; CHARM = Candesartan in Heart Failure – Assessment of Reduction in Mortality and Morbidity; COMET = Carvedilol or Metoprolol European Trial; CONSENSUS = Cooperative North Scandinavian Enalapril Survivial Study; COPERNICUS = Carvedilol Prospective Randomized Cumulative Survival Trial; DIG = Digitalis Investigators Group Study; EPHESUS = Eplerenone Post-AMI Heart Failure Efficacy and Survival Study; MERIT-HF = Metoprolol CR/XL Randomized Intervention Trial in Congestive Heart Failure; MI = myocardial infarction; NYHA = New York Heart Association; SAVE = Survival and Ventricular Enlargement Study; SOLVD = Studies of Left Ventricular Dysfunction; RALES = Randomized Aldactone Evaluation Study; SR = sinus rhythm; VAL-HeFT = Valsartan Heart Failure Trial; VALIANT = Valsartan in Acute Myocardial Infarction Trial

Approaches to Carotid Stenosis

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Key words:
Carotid stenosis (頸動脈狹窄), carotid angioplasty and stenting (頸動脈血管成形術及支架放置術)

Case 1

A 75-year-old gentleman experienced a 30-minute loss of power of his left upper arm while taking a flight from Shanghai to Hong Kong. He has had diabetes mellitus, hypertension and hypercholesterolaemia for more than 15 years. He has no history of transient ischaemic attack (TIA) or stroke. Exercise stress test and echocardiography, undertaken 1 year ago, showed normal results.

Physical examination revealed a right carotid bruit. There was no focal neurological sign, and his cardiac examination was unremarkable. His blood pressure was 150/90 mm Hg, and he was in sinus rhythm.

Questions

1. What further investigations would you arrange?
   a. Electrocardiogram
   b. Doppler carotid arteries
   c. Echocardiogram
   d. MRI brain
   e. All of the above

2. The patient's imaging result is shown in Figure 1. What is the abnormality?
   a. Vertebral artery stenosis
   b. Internal carotid artery stenosis
   c. Normal

3. What are the treatment options?
   a. Medical treatment
   b. Carotid endarterectomy
   c. Carotid angioplasty and stenting
   d. All of the above

Figure 1. Imaging result of the patient

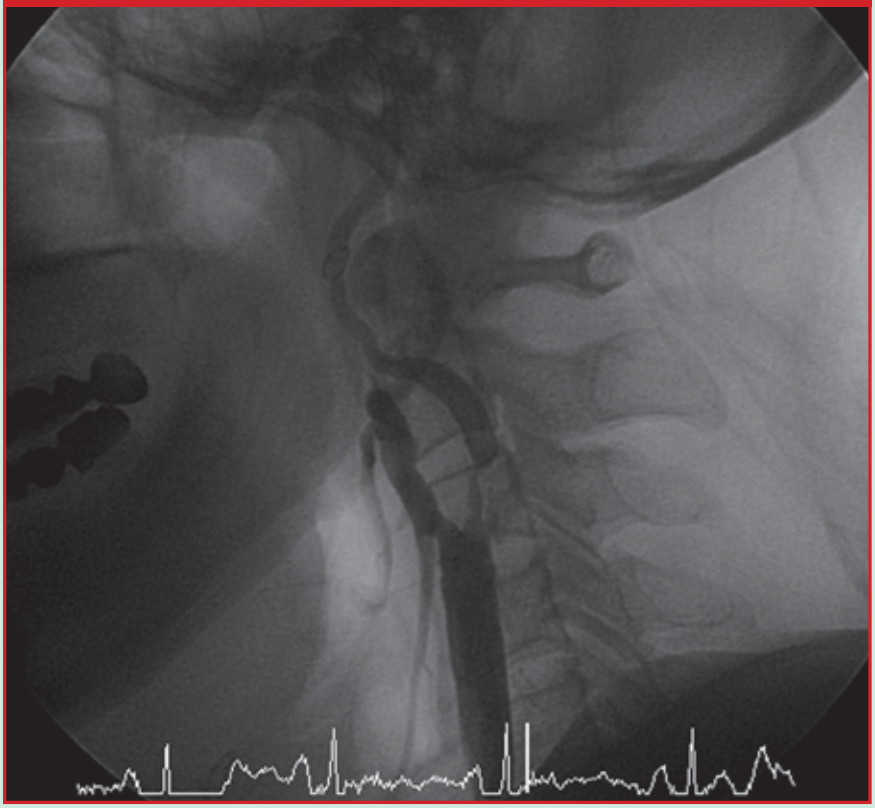
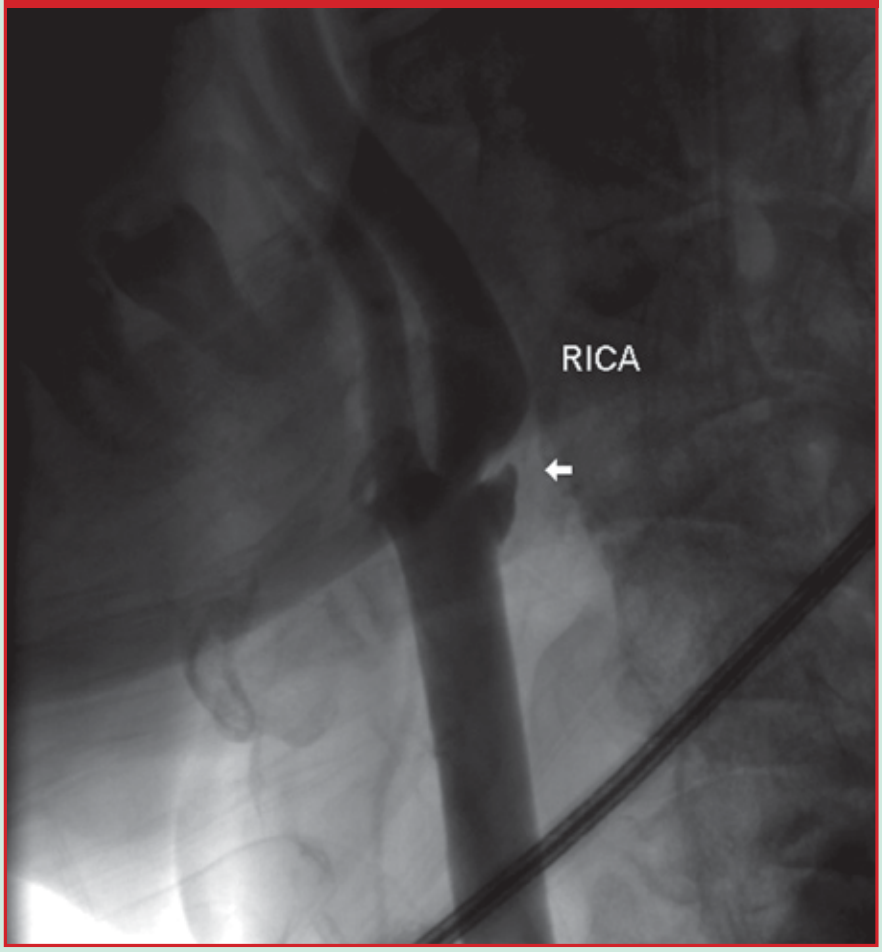


Figure 2. Finding of digital subtraction angiography



Case 2

Another 70-year-old gentleman presented with nonspecific dizziness. There was no history of TIA or stroke. Physical examination revealed no focal neurological abnormalities. Doppler ultrasound screening revealed moderate stenosis of his right internal carotid artery, and magnetic resonance angiography showed a moderately ulcerated plaque. Finding of digital subtraction angiography is shown in Figure 2.

Question

4. What is the best treatment option for this gentleman?
   a. Medical treatment with antiplatelet therapy and statin
   b. Carotid endarterectomy
   c. Carotid angioplasty and stenting

Answers: 1. e; 2. b; 3. d; 4. a

Discussion

Cerebrovascular accident is an important cause of morbidity and mortality. It can be due to ischaemic or haemorrhagic causes. Among ischaemic causes, extracranial carotid stenosis is a problem that can easily be identified.

Extracranial carotid stenosis is often asymptomatic clinically. Sometimes a carotid stenosis may be heard on physical examination. Carotid Doppler ultrasound study is the most convenient way to diagnose extracranial carotid stenosis. It allows assessment of the haemodynamic significance as well as the structural characteristics of the lesions (such as calcifications). MRI angiography provides a noninvasive means of delineating the whole extracranial and intracranial vasculature of the head and neck. In addition, it allows assessment of any old and silent cerebral infarcts, which may reflect the clinical significance of the carotid lesions concerned. Information about the status of intracranial vessels and vertebrobasilar vessels is important, especially when surgical and endovascular procedures are considered.

For asymptomatic patients, particularly those with less than critical stenosis, medical therapy alone appears to be the treatment of choice. While medical treatment with antiplatelet therapy such as aspirin and clopidogrel is an important modality of treatment, studies have shown that surgical treatment with carotid endarterectomy provides superior outcome compared with medical treatment for severe symptomatic stenosis, such as the patient in the first case study.[1-3] Although carotid endarterectomy has been the mainstay of surgical intervention for many years with proven results, it is relatively invasive and requires a longer hospital stay after the procedure. Many patients with extracranial carotid stenosis have significant comorbidities such as concurrent coronary artery disease, compromised left ventricular function, and coexisting organ failure such as impaired renal or pulmonary function, which will all increase the surgical risk of the procedure.

In recent years, carotid angioplasty and stenting has emerged as another

viable treatment option for carotid stenosis, especially in those subgroups of patients who are not ideal candidates for surgical treatment.[4] These include those with significant concurrent medical morbidities, severe coronary artery disease, contralateral obstruction, bilateral carotid stenosis, high carotid lesions above cervical level, and restenosis after previous endarterectomy. Compared with surgery, the endovascular approach with stenting is minimally invasive and requires a shorter hospital stay. Its success rate, complication rate and long-term outcome are also comparable with the conventional surgical method.

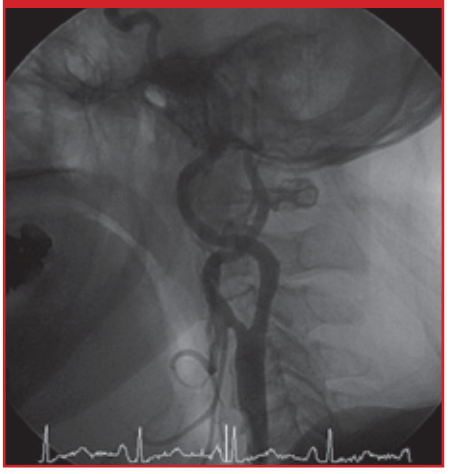
The patient in the first case went for carotid stenting, which was done uneventfully. At 3 years, he remained asymptomatic with no restenosis as assessed by Doppler ultrasound. His poststenting image is shown in Figure 3.

However, not all patients are suitable for carotid stenting. For example, patients with a calcified and elongated aortic arch, long calcified lesions and tortuous vessels carry a high risk for perioperative stroke. Therefore, carotid endarterectomy and carotid stenting are in fact complementary procedures, and together they provide alternative solutions to patients with different anatomical challenges.

Summary

In summary, all patients with ischaemic and thromboembolic stroke should be screened for extracranial carotid stenosis. Asymptomatic patients, particularly those with stenosis of less than 70%, may be treated medically. Symptomatic severe stenosis is best treated with revascularization, which includes carotid endarterectomy and carotid stenting. The choice of revascularization depends on the experience of the clinician and the institution, as well as the anatomical characteristics of the lesions and the surgical risk of the patients. In general, compared with carotid endarterectomy, carotid stenting is less invasive and requires a shorter hospital stay.

Figure 3. The patient in case 1 poststenting



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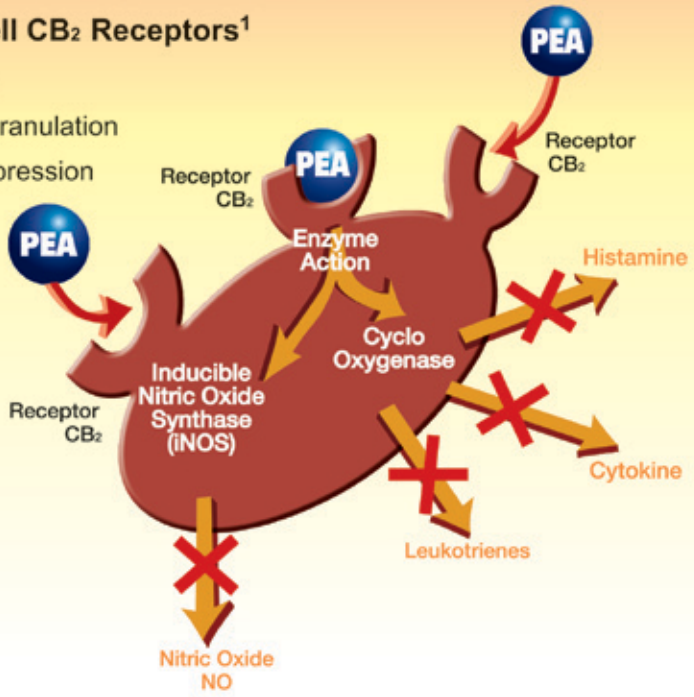

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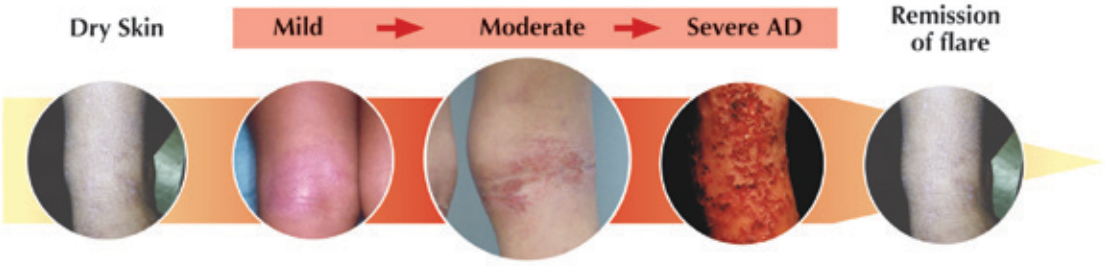

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References:
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Utilizing Cardiac CT for the Detection of Coronary Artery Disease

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Key words:
Cardiac CT (心臟電腦掃描),
coronary artery disease (冠心病)

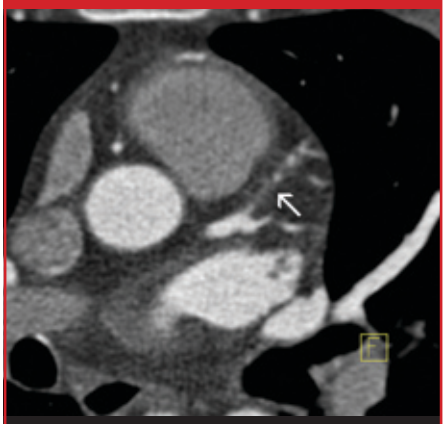
Noninvasive coronary angiography has become possible as a result of improved temporal and spatial resolution with multidetector computed tomography (MDCT). The following case is an example of using 64-slice MDCT for the detection of coronary artery disease. The basic techniques of reading cardiac CT pictures will be demonstrated.

The patient is a 46-year-old man, a nonsmoker. He has no history of diabetes mellitus or hypertension. His cholesterol level is elevated. His total cholesterol is 6.9 mmol/L, LDL cholesterol is 5.48 mmol/L, and HDL cholesterol is 0.69 mmol/L. He has no chest pain, and has good exercise tolerance during daily activities. Electrocardiography has been performed as a preoperative assessment before his ganglion operation. His electrocardiogram shows Q waves in V1-4. Echocardiography has been performed, which shows a dilated left ventricle with moderate global hypokinesia. His left ventricular ejection fraction is 40%. Cardiac CT is then performed to look for coronary artery disease.

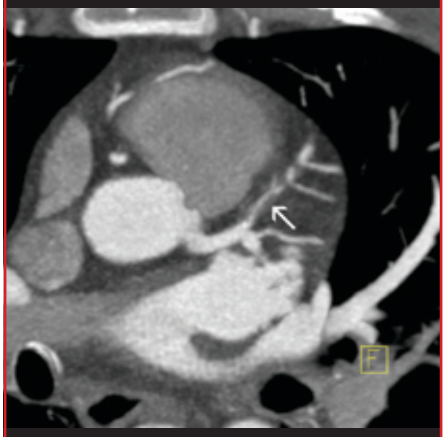
To analyze coronary arteries by cardiac CT, a systematic approach should be followed. An overview of the data set should be obtained to detect gross abnormalities and artefacts. Postprocessing techniques including axial image scrolling, multiplanar reconstruction (MPR), maximum intensity projection (MIP) and curved mutiplanar reconstruction (cMPR) should be mastered for accurate assessment of lesions.

It is recommended to review the axial image first. When scrolling through the axial images from the cranial to the caudal direction, one's eyes should follow one coronary artery each time. One may look at the left anterior descending artery (LAD) first, followed by the left circumflex artery (LCx), and then the right coronary artery (RCA). For this patient, by scrolling through the axial image, we can see severe stenosis in the proximal LAD. (Figure 1A) Severe stenosis in the proximal segment of the RCA and total occlusion in the mid to distal segment of the RCA are also seen. The axial plane is especially useful for the evaluation of the proximal LAD. MPR in the axial plane shows the proximal LAD lesion clearly. (Figure 1A)

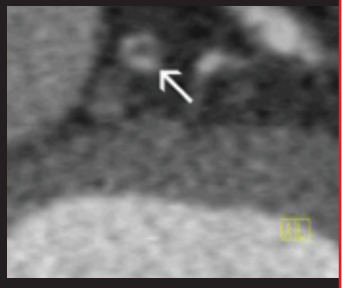
Figures 1A-1C



(1A)



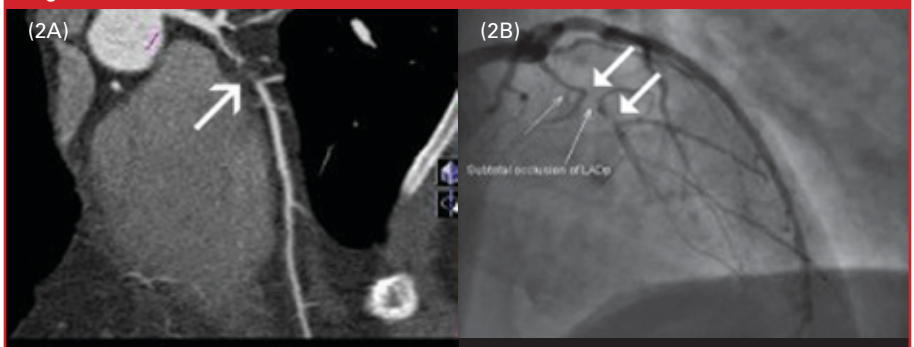
(1B)



(1C)

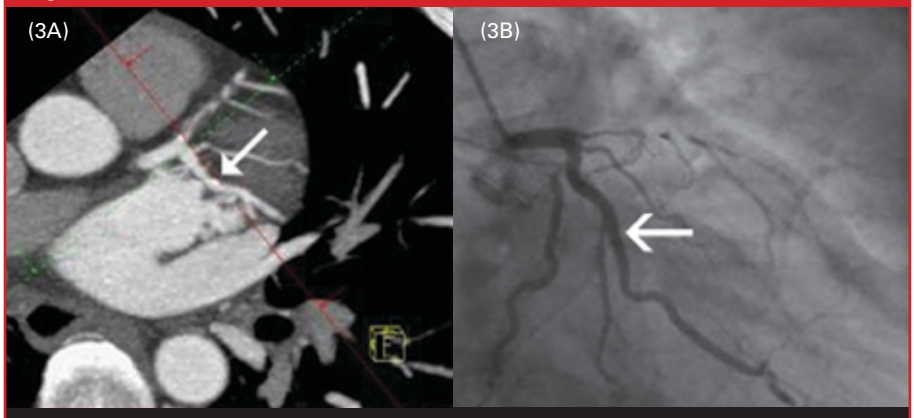
(1A) Severe stenosis in the proximal LAD. (1B) MIP illustrates the length of the lesion and the relation of the lesion to the diagonal branches. (1C) Subtotal occlusion in the proximal and mid LAD.

Figures 2A-2B



(2A) Subtotal occlusion of the proximal and mid LAD in cMPR. (2B) Subtotal occlusion of the proximal to mid LAD on conventional coronary angiography.

Figures 3A-3B



(3A) A small calcified plaque in the mid LCx. (3B) Small lesion in the mid LCx.

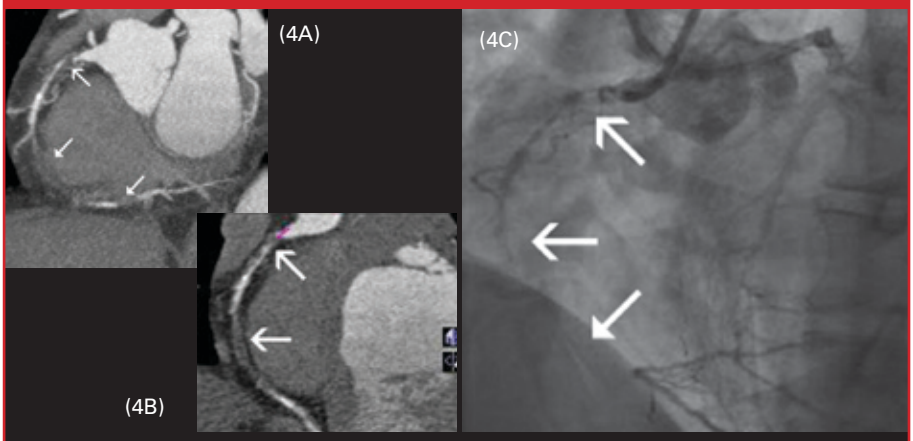
Another reconstruction method is MIP. The highest intensity voxels are used to reconstruct the two-dimensional projection image. It is similar to the principle of conventional coronary angiography, in which the highest attenuation value is projected on the image. Compared with MPR, MIP shows longer lengths of vessels, and the relation of the main vessel to the branches can also be seen. However, the problem is that high-density structures may obscure low-density structures, so that lesions may be missed. For this patient, the length of the lesion and the relation of the lesion to the diagonal branches can be illustrated in MIP. (Figure 1B) The LAD runs in the interventricular groove, so that the axial and parasagittal planes will help in the evaluation of LAD lesions. Tracing along the MIP picture of the LAD in parasagittal view can demonstrate the entire course of the LAD. Dedicated planes parallel and orthogonal to the lesion can be generated.

When suspected lesions are detected, the segment of interest should be magnified, and orthogonal views of the vessel should be created. Orthogonal views create images similar to that of intravascular ultrasound, so that the patency of the vessel can be evaluated. Subtotal occlusion is demonstrated in the proximal and mid LAD. (Figure 1C)

cMPR can be generated using a manual method or automatically. Using the Circulation Software provided by Siemens, we start with the volume data set of the heart and the rib cage. The heart is then spontaneously isolated from the volume data set. After processing the data with blood pool removal and angioview algorithm, the three-dimensional view of the coronary arteries that resembles conventional coronary angiography is generated. We can rotate the heart in every direction to create pictures comparable to conventional coronary angiography in usual views. The cMPR picture of each vessel can be generated and rotated in every direction. We need to rotate and look at the pictures in every direction to see eccentric lesions. Subtotal occlusion of the proximal and mid LAD is well demonstrated in cMPR. (Figure 2A) Conventional coronary angiography shows a similar picture as the cardiac CT, which demonstrates subtotal occlusion of the proximal to mid LAD. (Figure 2B)

By rotating the MIP picture in the axial view, we can see the proximal to

Figures 4A-4C



(4A) Subtotal occlusion in the proximal segment and total occlusion in the mid to distal segment on the MIP picture of RCA. (4B) Same findings on the MPR picture. (4C) Bilateral injection using conventional coronary angiography shows retrograde collaterals and the lesions.

mid segment of the LCx artery. Figure 3A shows a small calcified plaque in the mid LCx. The orthogonal view of the LCx can then be created, which demonstrates the small calcified plaque without significant stenosis in the mid LCx. The cMPR picture of the LCx is then created. Conventional coronary angiography cannot show the small lesion in the mid LCx. (Figure 3B) This demonstrates that cardiac CT is more sensitive in picking up small calcified lesions than conventional coronary angiography.

While RCA runs in the atrial ventricular groove, placing the reference line in the atrial ventricular groove will show the paracoronal plane. The MIP picture of RCA shows subtotal occlusion in the proximal segment and total occlusion in the mid to distal segment, as well as retrograde collaterals. (Figure 4A) The MPR picture shows the same findings. (Figure 4B) Conventional coronary angiography shows again subtotal occlusion in the proximal segment, total occlusion in the mid to distal segment, and bridging collaterals. Bilateral injection using conventional coronary angiography shows retrograde collaterals and the lesions. (Figure 4C)

This case shows that when reading cardiac CT pictures, different postprocessing methods have their own advantages. Each lesion should be assessed with different methods, including MPR, MIP and cMPR. As a result, the severity of the lesion and the relation of the lesion to the branches can be assessed.

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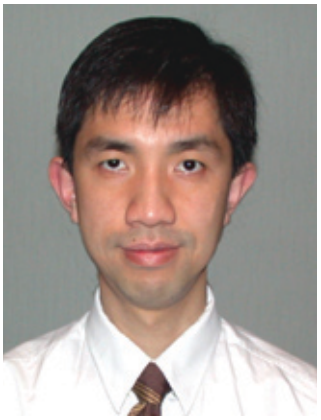

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Key words:
Apical hypertrophic cardiomyopathy (心尖肌肉肥厚), diastolic heart failure (舒張性心力衰竭), echocardiogram (心臟超聲檢查)

Case History

A 40-year-old male smoker presented to his doctor for recent-onset nonexertional vague chest discomfort. He also noticed subjective worsening of exercise tolerance. He denied any symptom of palpitation, orthopnoea or ankle swelling. He does not have any family history of coronary artery disease. His fasting blood sugar was 5.4 mmol/L , HDL cholesterol was 0.9 mmol/L, and LDL cholesterol was 3.6 mmol/L.

Physical examination showed normal blood pressure at 126/70 mm Hg, with a regular pulse at 64/min. Precordial and chest examination was unremarkable. His ECG is shown in Figure 1. He was suspected to have coronary artery disease, and a CT coronary angiogram was arranged.

Figure 1. ECG of the patient

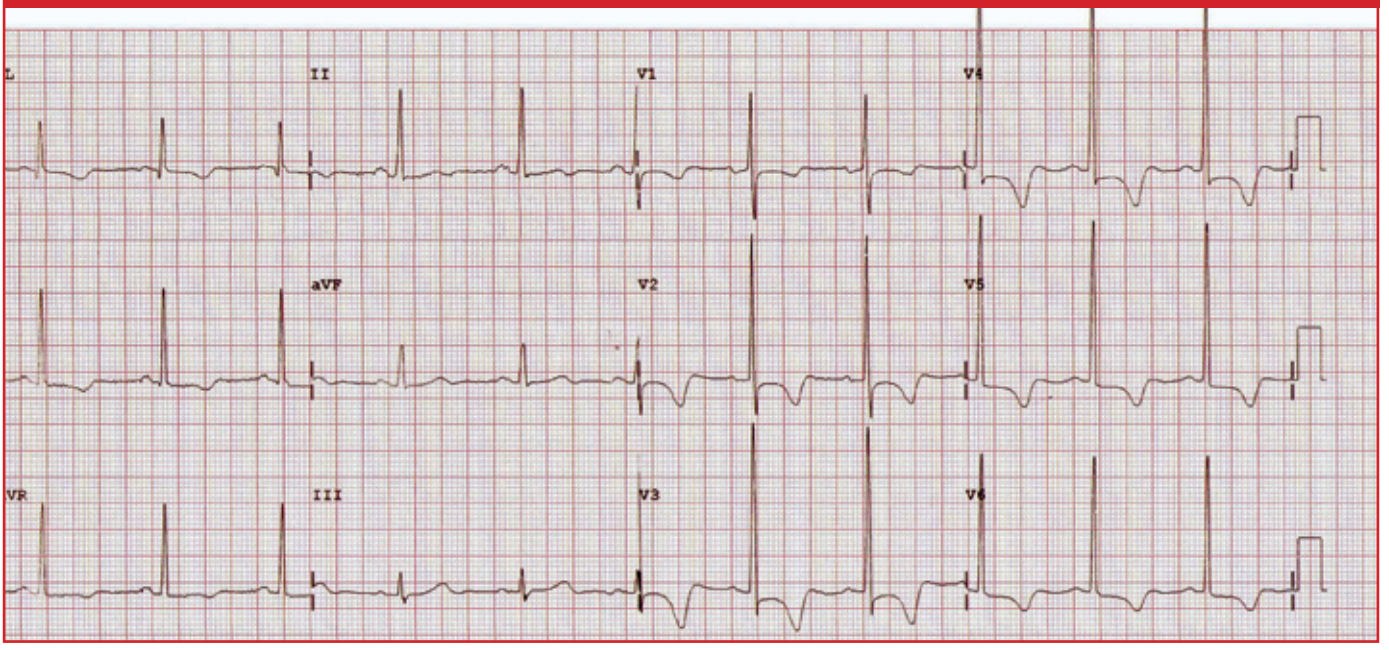


Figure 2. CT coronary angiogram of the patient

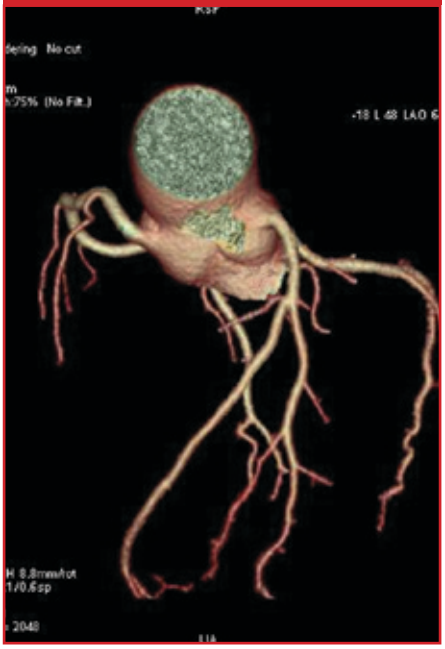
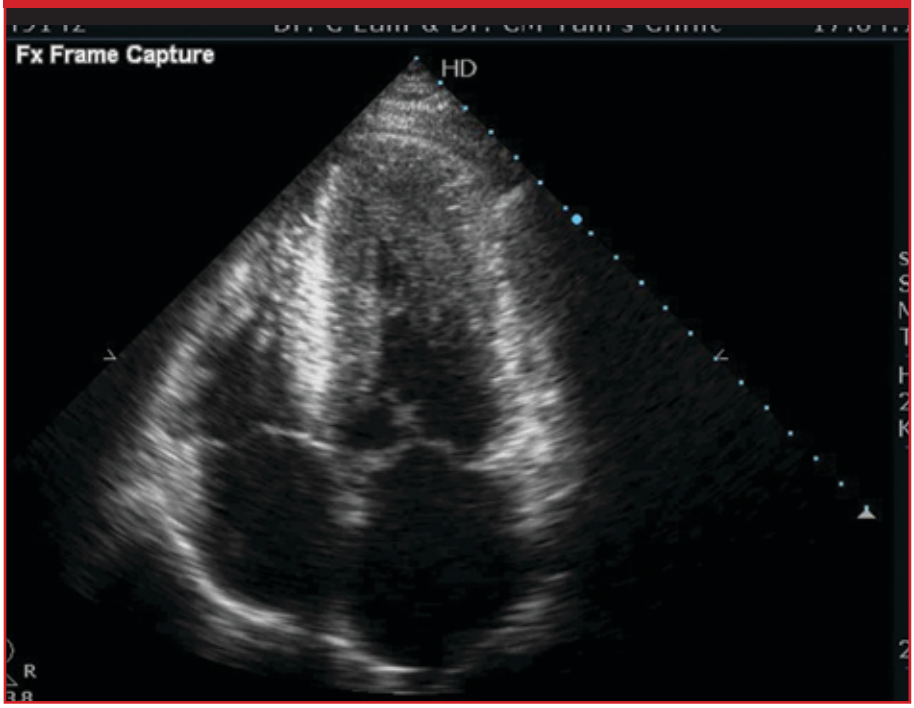


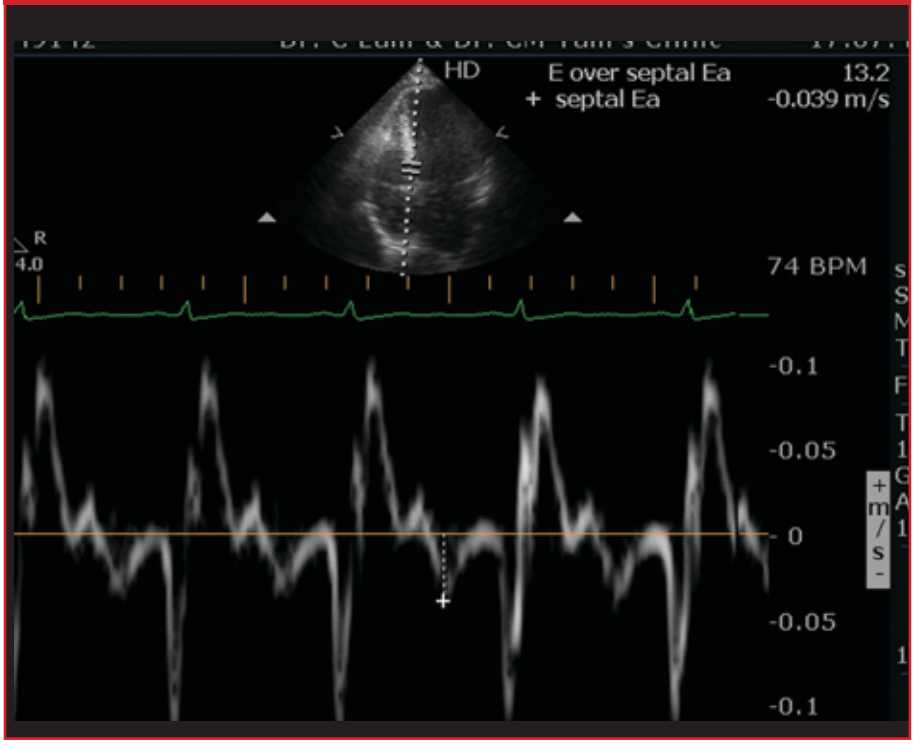
Figure 3. Echocardiogram showed features of apical hypertrophic cardiomyopathy



The CT coronary angiogram was normal without any significant plaque or stenosis. (Figure 2) The patient was reassured and discharged.

The patient sought a second opinion from another doctor for his persistent symptom. Echocardiogram was arranged for his abnormal ECG and symptom. Echocardiogram showed features of apical hypertrophic cardiomyopathy, and there was Doppler evidence of left ventricular diastolic dysfunction. (Figures 3 and 4) His CT coronary angiogram was thus reviewed, and it confirmed the diagnosis of apical hypertrophic cardiomyopathy. (Figure 5)

Figure 4. Tissue Doppler evidence of left ventricular diastolic dysfunction



Discussion

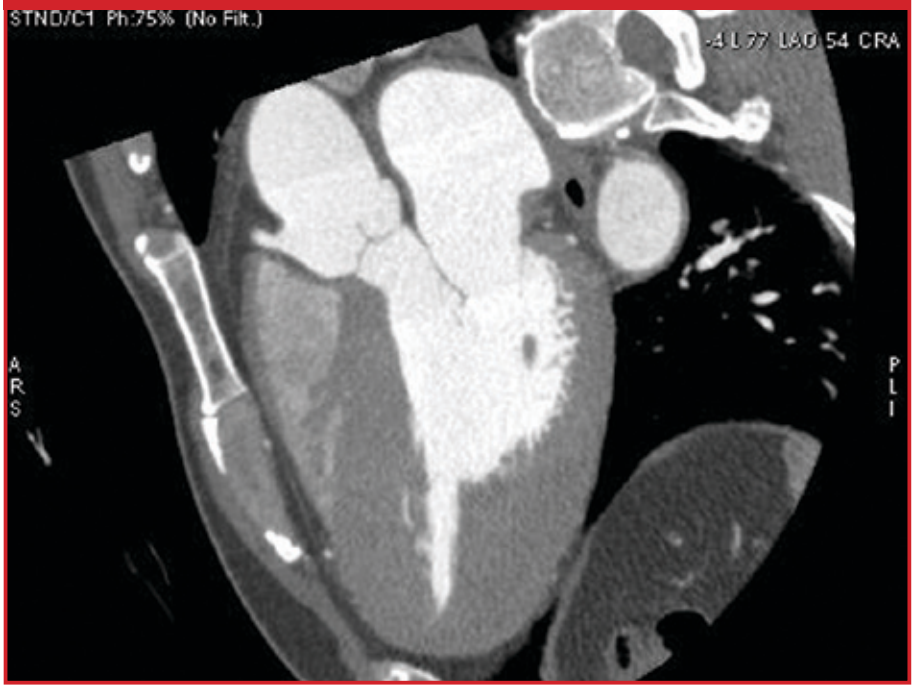
When a patient complains of chest pain, most doctors would like to rule out coronary artery disease, which is always considered the most dangerous cause. In this case, atypical chest pain in a middle-aged man represents an intermediate pretest likelihood of coronary artery disease. Usually, an exercise stress test will be a logical next step for subsequent risk stratification. However, the severely abnormal baseline ECG will preclude any meaningful conclusion from an exercise ECG test. A simple echocardiogram to assess the cause of the abnormal ECG and exertional shortness of breath will help to confirm the diagnosis early. A functional imaging test such as stress echocardiogram or stress MRI, or an anatomical imaging test such as CT coronary angiogram will be adequate to confirm the diagnosis. The pitfall of this case is the presumption of coronary artery disease over other causes of heart disease in the setting of chest pain and abnormal ECG.

Apical Hypertrophic Cardiomyopathy

Apical hypertrophic cardiomyopathy is not an uncommon disease. Western and Chinese literature reported a prevalence rate of 0.2% of hypertrophic cardiomyopathy in these populations.[1] Hypertrophic cardiomyopathy affecting mainly the mid to apical left ventricular cavity (ie, apical hypertrophic cardiomyopathy) is first reported in Japan in the 1970s, and is found to be more common in the oriental population.[1] While Western series reported a prevalence rate of 9% of the apical variant, Japanese data reported a prevalence rate of 13% to 25%, and a local series reported the highest prevalence rate of 41%.[2,3] Hypertrophic cardiomyopathy is caused by mutation of different myosin genes and it runs in the family.

Apical hypertrophic cardiomyopathy is considered to be more benign and carries a better prognosis compared to the classic outflow tract obstructive hypertrophic cardiomyopathy. The annual mortality reported from a 13-year cohort was 0.1% (versus 1.4% to 4 % for classical hypertrophic cardiomyopathy).[3] Since it mainly affects the mid to apical left ventricular cavity, it causes no obstruction to the left ventricular outflow tract and thus less severe symptoms of heart failure. It also carries a lower risk of sudden death due to mechanical obstruction or life-threatening arrhythmia. However, even though there is no mechanical outflow tract obstruction, the severe form of apical hypertrophic cardiomyopathy can cause disabling symptoms due to severe left ventricular diastolic dysfunction. The thick and non-compliant left ventricular muscle diminishes the left ventricular volume, causing severe restriction during diastole. The small and restricted left ventricle causes a fixed small stroke volume and high filling pressures (restrictive physiology). Upon exercise, cardiac output can only be increased by increasing the heart rate. However, tachycardia will further compromise left ventricular filling due to short filling time and underlying impaired relaxation. Therefore, the patient will experience varying degrees of heart failure symptoms, ranging from effort intolerance to resting dyspnoea, depending on the different stages of diastolic dysfunction. The local series also reported a high incidence of paroxysmal atrial fibrillation (12%), which is likely due to left atrial enlargement secondary to diastolic dysfunction.[2]

Figure 5. CT coronary angiogram confirmed apical hypertrophic cardiomyopathy



Diagnosis

The diagnosis of apical hypertrophic cardiomyopathy is mainly by echocardiography. The sensitivity of echocardiography in detecting apical hypertrophic cardiomyopathy can be further increased by the use of echo contrast, as certain patients could be very non-echogenic due to underlying small left ventricular volume. Cardiac MRI and cardiac CT are two other newer tests which can help such diagnosis. Cardiac MRI is very useful especially when the patient is non-echogenic and echo contrast is not available. It can also study cardiac wall motion, but cannot assess left ventricular diastolic function and obstructive gradient as echocardiogram. Cardiac CT can help to rule out concomitant coronary artery disease in the setting of abnormal ECG, but apical hypertrophy may be missed if it is not particularly looked for. Overall, echocardiography is still the most useful investigation to confirm the diagnosis and assess the anatomical and functional involvement of apical hypertrophic cardiomyopathy.

Treatment

Treatment of apical hypertrophic cardiomyopathy is mainly to alleviate heart failure and arrhythmia. If there is a family history of sudden death and unexplained syncope, the patient is at high risk of malignant arrhythmia. An internal defibrillator may be indicated to prevent sudden death. Medical treatment of diastolic dysfunction includes beta-blockers and diuretics. Anticoagulation may also be needed for thromboembolism prophylaxis.

Conclusion

Heart disease does not equate coronary artery disease alone. Apical hypertrophic cardiomyopathy is not an uncommon cause of heart failure and abnormal ECG. Basic investigation such as echocardiogram is indispensable for making the diagnosis and functional assessment.

References:

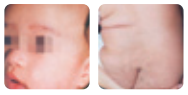
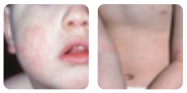
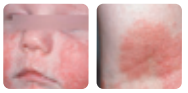
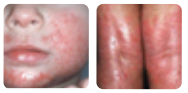
1. Wigle ED. Cardiomyopathy: The diagnosis of hypertrophic cardiomyopathy. Heart 2001;86:709-714.
2. Ho HH, Lee KL, Lau CP, Tse HF. Clinical characteristics of and long-term outcome in Chinese patients with hypertrophic cardiomyopathy. Am J Med 2004;116:19-23.
3. Eriksson MJ, Sonnenberg B, Woo A, et al. Long-term outcome in patients with apical hypertrophic cardiomyopathy. J Am Coll Cardiol 2002;39:638-645.

*Adapted from NICE clinical guideline 57, NHS UK, 2007

Atopic Dermatitis Stepped-care plan*

Diagnosis - Physical assessment

Diagnosis:

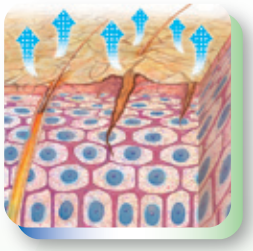
Clear	Mild	Moderate	Severe
			

Treatment:

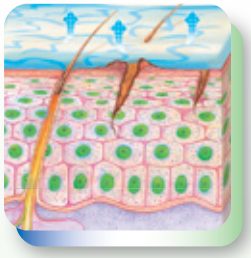
Emollients	Emollients	Emollients	Emollients
	+ Mild potency topical corticosteroids	+ Moderate potency topical corticosteroids + Topical calcineurin inhibitors + Bandages	+ Potent topical corticosteroids + Topical calcineurin inhibitors + Bandages + Phototherapy + Systemic therapy

Treat areas of differing severity independently
Step treatment up or down according to physical severity

Dry Skin Problems

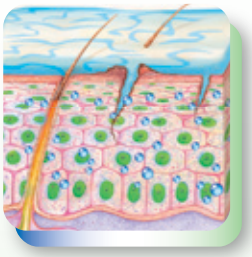


The Power of Occlusives



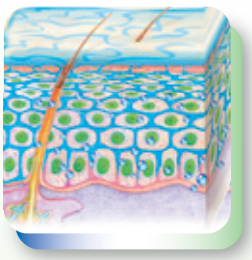
Polyglycerylmethacrylate forms a "reservoir" (a hydrophobic film on the skin), which restricts water across the epidermis

The Power of Emollients



Fill the spaces between desquamating corneocytes to improve the appearance and feel of the skin

The Power of Humectants



Draw water up from the dermis into the stratum corneum

	Cetaphil Moisturizing Cream	Cetaphil Moisturizing Lotion
Emollient	Propylene Glycol	Dimethicone
Humectant	Glycerin	Glycerin
Occlusive	Polyglyceryl -methacrylate	Silicon Wax (Stearoxy -trimethylsilane & Stearyl Alcohol)

Cetaphil moisturizers are enriched with occulsives, humectants and emollients to help your patients with dry and sensitive skin

Cetaphil®
Every Age. Every Stage. Every Day.™

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