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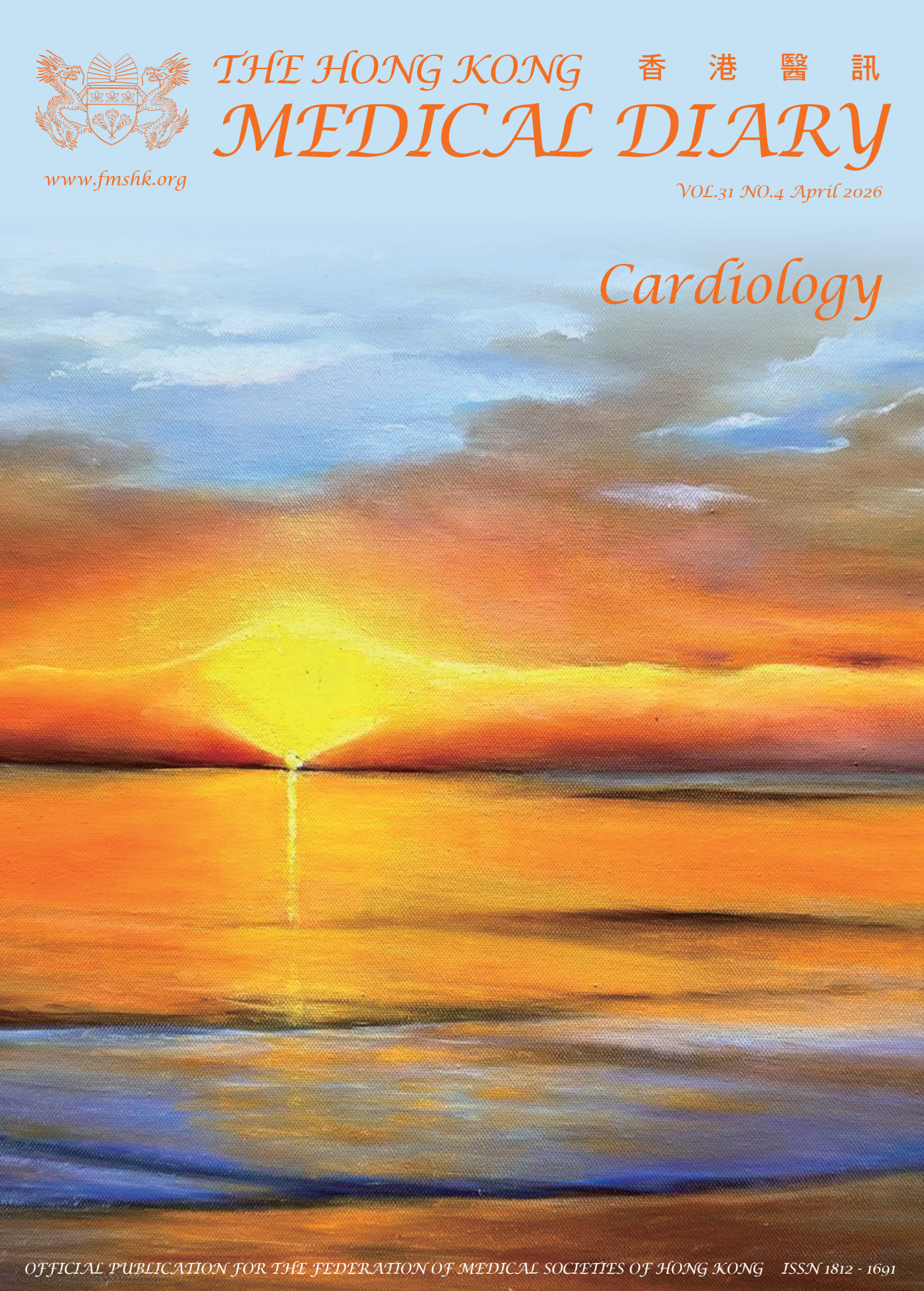
Cardiology



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Dr Victor YAN

MBBS (HK), MRACP, FRACP

Cardiologist

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across LVEF spectrum in HF[1,2]

Kidney protection
in the **broadest CKD population***[3-5]

Metabolic control
as **the only SGLT2i with CV death reduction**†[6-9]

* EMPA-KIDNEY study population included patients with CKD who had an eGFR of ≥20 to <45 ml/min/1.73 m², or who had an eGFR of ≥45 to <90 mL/min/1.73 m² with a UACR of ≥200 mg/g. The primary outcome was a composite of progression of kidney disease (end-stage kidney disease, a sustained eGFR decrease to <10 mL/min/1.73 m², a sustained eGFR decrease of ≥40% from baseline, or death from renal causes) or CV death; 13.1% (432/3,304) in JARDIANCE® group vs 16.9% (558/3,305) in placebo group (hazard ratio 0.72, 95% CI 0.64–0.82, P<0.001). Significant reduction of eGFR decline was observed across all categories of albuminuria.[4] DAPA-CKD study population included CKD patients with an eGFR of 25 to 75 mL/min/1.73 m² and UACR of 200 to 5,000 mg/g.[5]

† The only SGLT2i with CV death reduction in T2DM with eCVD.

Abbreviations: CKD=Chronic kidney disease; CKM=Cardio-kidney-metabolic; CV=Cardiovascular; eCVD=Established cardiovascular disease; eGFR=Estimated glomerular filtration rate; HF=Heart failure; LVEF=Left ventricular ejection fraction; SGLT2i=Sodium-glucose cotransporter 2 inhibitor; UACR=Urine albumin-to-creatinine ratio.

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Bridging the Vascular Divide: A Call to Action on Cardiometabolic Syndrome, Peripheral Arterial Disease, Venous Thromboembolism, Atrial Fibrillation and Structural Heart Disease

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Issue Editor

Prof Bryan PY YAN

Cardiovascular disease remains the leading cause of death and disability worldwide, yet the breadth of vascular and cardiac conditions that contribute to this burden is often considered in silos. Peripheral arterial disease (PAD), cardiometabolic syndrome, venous thromboembolic disease (VTE), atrial fibrillation (AF), and structural heart disease share overlapping risk factors, pathophysiology, and opportunities for prevention and early intervention. Recognising and integrating these conditions into a unified public-health and clinical strategy is essential to reduce morbidity, preserve function, and save lives.

Cardiometabolic syndrome - characterised by central obesity, insulin resistance, dyslipidaemia, and hypertension - serves as the common soil from which many cardiovascular and thrombotic conditions grow. It amplifies the risk of PAD, AF, structural heart disease and VTE. Addressing cardiometabolic health requires population-level policy changes (healthy food environments, active transport, accessible preventive care) alongside individualised risk modification: weight management, glycaemic control, blood pressure and lipid lowering, and targeted pharmacotherapy where appropriate.

Peripheral arterial disease is underdiagnosed and undertreated. Many patients with PAD present with atypical leg pain or are asymptomatic, while still experiencing increased risk of myocardial infarction, stroke, and limb loss. Increasing awareness among clinicians and the public, and embedding PAD screening into routine vascular risk assessments, will identify patients who benefit from secondary prevention and timely referral for revascularisation when indicated.

Venous thromboembolic disease (VTE), encompassing deep vein thrombosis (DVT) and its most feared complication, pulmonary embolism (PE), is a major source of morbidity, long-term disability, and healthcare costs. While anticoagulation remains the cornerstone of acute management, a growing body of evidence supports endovenous interventions as a targeted strategy to reduce short- and long-term complications for selected patients. Appropriate patient selection is essential to balance benefits against bleeding risk and procedural complications. By combining precise selection, advanced techniques, and coordinated care pathways, we can reduce the downstream burden of VTE and restore function and quality of life for many who would otherwise suffer chronic venous sequelae.

Atrial fibrillation has emerged as a major contributor to stroke, heart failure, and cognitive decline. AF prevalence rises with age and cardiometabolic burden. Early detection - via opportunistic pulse checks, mobile rhythm monitoring for high-risk patients, or systematic screening in selected populations - coupled with guideline-concordant

stroke prevention (risk-score-based anticoagulation), rhythm control where appropriate, and aggressive risk-factor modification can lower the public health impact of AF.

Structural heart disease, including valvular heart disease and cardiomyopathies, operates at the nexus of ageing, metabolic stress, and vascular disease. Timely diagnosis through echocardiography and advanced imaging enables medical optimisation and, increasingly, catheter-based or surgical interventions that restore function and quality of life. Structural disease frequently coexists with PAD and AF - underscoring the need for coordinated care pathways among cardiology, vascular medicine, and primary care.

An integrated strategy to reduce the toll of these interrelated conditions should include: (i) routine vascular risk assessment in primary care that incorporates PAD screening, AF detection, and cardiometabolic evaluation; (ii) public-health policies that tackle obesogenic environments, improve access to healthy foods, and promote physical activity; (iii) education campaigns for clinicians and the public to recognise symptoms of PAD, VTE, AF, and structural heart disease; (iv) multi-disciplinary care models (cardio-vascular clinics, anticoagulation services, heart teams) to streamline diagnosis, management, and follow-up and (v) research investments into screening effectiveness, implementation science, and therapies that target shared mechanistic pathways (inflammation, thrombosis, metabolic dysfunction).

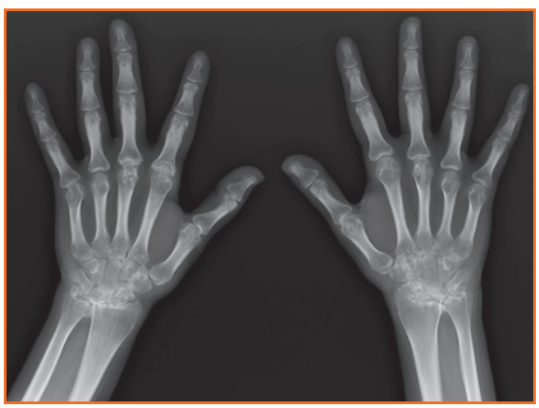
We cannot continue treating these conditions as separate entities. A systems-level approach - rooted in prevention, early detection, and coordinated care - can reduce the overlapping burdens of PAD, cardiometabolic syndrome, VTE, AF, and structural heart disease. Clinicians, policymakers and communities must act together to bridge the vascular divide and deliver better cardiovascular health for all.

Radiology Quiz

Dr Janet Hin-man WONG

MBBS, FRCR

Dr Janet Hin-man WONG



A 32-year-old lady presented with subacute onset of bilateral wrists and small joint pain for a few years.

Questions

1. What are the radiological findings?
2. What is the most likely diagnosis?
3. What is the next step of management?

(See P. 36 for answers)

Managing Cardiovascular-kidney-metabolic (CKM) in the General Practice Setting

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Prince of Wales Hospital, Chinese University of Hong Kong

Dr Guang-ming TAN

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 30 April 2026.

INTRODUCTION

Cardiovascular-kidney-metabolic (CKM) syndrome represents a novel framework that highlights the interconnected pathophysiology among cardiovascular disease (CVD), chronic kidney disease (CKD), and metabolic diseases such as diabetes and obesity. CKM syndrome is categorised into five progressive stages, with higher stages correlating with escalating risks of adverse cardiovascular and renal outcomes. The CKM health model promotes systemic screening, risk stratification, and targeted management to facilitate early detection and mitigate disease progression. First introduced by the American Heart Association (AHA) in 2023[1], the Hong Kong College of Physicians (HKCP) has developed a position statement to adapt this CKM framework to Hong Kong's healthcare system[2]. This adaptation considers local needs, resource constraints, financing mechanisms, care delivery models, and emerging initiatives such as the Primary Healthcare Blueprint and the Chronic Disease Co-Care (CDCC) Pilot Scheme[3].

CHALLENGES IN GENERAL PRACTICE SETTING

CKM syndrome seeks to identify at-risk individuals for timely interventions to halt disease progression. Nonetheless, several challenges remain. A recent Hong Kong population survey showed widespread unawareness of overweight/obesity, hypertension, diabetes, and hypercholesterolaemia[4]. The ageing population further strains healthcare resources to manage CKM-related end-organ complications. Although promising policies like the Primary Healthcare Blueprint and the 3-year CDCC Pilot Scheme (launched November 2023) aim to bolster prevention[3], specialist-primary care integration remains insufficiently leveraged, and awareness among primary physicians remains limited.

SCREENING RECOMMENDATIONS

Screening for metabolic risks, including overweight/obesity, central adiposity, hyperglycaemia, hypertension, and dyslipidaemia, is a cornerstone of the CKM framework. Assessment should begin early, starting at age ≥ 21, with annual body mass index (BMI) and waist circumference checks, plus blood pressure, lipid profile, and glycaemia testing. Screening Intervals vary by stage: every 3 - 5 years for stage 0 (healthy/lean individuals), 2 - 3 years for stage 1 (overweight/obese or prediabetes), and annually for stage 2 (diabetes, hypertension, dyslipidaemia). Uncertainty remains regarding Asian-specific BMI thresholds for overweight and obesity[5]. In this regard, waist circumference or relative fat mass (RFM) index may serve as an alternative for detecting adiposity[6]. In Hong Kong, the early screening programme is supported by the CDCC Pilot Scheme[3], which subsidises private-sector screening for residents ≥ 45 without known diabetes/hypertension, starting with BP and HbA1c, followed by lipids, eGFR, and urinalysis if initial results are abnormal. The diagnostic threshold for hypertension has been updated to ≥ 130/80 mmHg, replacing the previously used ≥ 140/90 mmHg, both home and office monitoring are encouraged.

ROLES OF PRIMARY CARE PHYSICIANS

The primary care physicians play a pivotal role in the early detection and management of asymptomatic cases of CKM syndrome. For CVD risk assessment, the AHA's PREVENT equation may be used to estimate 10-year risk in asymptomatic 30-79-year-olds without established CVD or HF, incorporating CKM factors (preferred over Pooled Cohort Equations)[7]. However, this needs to be applied with caution in Asian patients due to the potential risk of overestimation[8]. Additionally, Coronary artery calcium (CAC) scoring can support shared decision-making regarding statin and aspirin use for primary prevention, though routine testing should be undertaken with caution due to testing burdens and the lack of follow-up programmes. Cardiac biomarkers, such as BNP/NT-proBNP and high-sensitivity troponin, can aid detection of subclinical heart failure. However, their utility caution is questionable since Guideline-directed management (GDMT) for HF, such as angiotensin-converting enzyme inhibitors (ACEi) and angiotensin receptor blockers (ARBs) , are already the first-line medication for HT. Screening for eGFR and urine albumin-to-creatinine ratio (ACR) in at-risk groups, including those

with dyslipidaemia, metabolic syndrome, diabetes, and hypertension is often overlooked. It should be reinforced as a key strategy for early detection.

CLINICAL MANAGEMENT: INTERDISCIPLINARY MODEL

GDMT, with appropriate adjustment for Asians population, should be applied in the management of CKM complications. In particular, referral for weight management therapy, such as bariatric surgery or a Glycogon-Like Peptide 1 Receptor Antagonist (GLP1RA), should be considered for patients with BMI ≥ 27.5 kg/m² or ≥ 25 kg/m² in those with type 2 diabetes. Cardioprotective antidiabetic therapy, such as metformin, should be prioritised if HbA1c < 7.5 % with optimisation of glycaemic control for cardiorenal benefits. Statins and aspirin should be considered for primary prevention of CVD. Intensification of lipid control using a combination of statins, ezetimibe, and proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors may be required to keep LDL < 1.8 mmol/L in high-risk individuals. For patients with HF (HF with reduced or preserved ejection fraction), sodium–glucose cotransporter-2 (SGLT2) inhibitors and non-steroid mineralocorticoid receptor antagonist (Finerenone) should be considered. Kidney-protective therapies, including ACEi, ARB, SGLT2i, GLP-1 receptor agonists, or Finerenone, are recommended across all stages of CKD. Referral to specialist care is warranted for patients who develop advanced CKM complications, such as severe HF, end-stage renal failure or severe CVD.

CONCLUSION AND WAY FORWARD

CKM syndrome carries substantial implications for patient outcomes. Effective prevention of complications requires integrative care that bridges primary care and specialist services. Conditions such as hyperglycaemia, dyslipidaemia, obesity, kidney insufficiency, and hypertension should be treated as chronic diseases warranting early intervention rather than merely risk factors. Proactive screening for asymptomatic individuals and timely initiation of GDMT are crucial to mitigate the adverse sequelae of CKM syndrome.

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MCHK CME Programme Self-assessment Questions

Please read the article entitled "Managing Cardiovascular-kidney-metabolic (CKM) in the General Practice Setting" by Dr Guang-ming TAN 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 answer link: https://forms.gle/tJZgcHdDa3fK6wiu8 or by mail to the Federation Secretariat on or before 30 April 2026. Answers to questions will be provided in the next issue of The Hong Kong Medical Diary. (Address: Duke of Windsor Social Service Bldg., 4/Fl., 15 Hennessy Rd., Wan Chai. Enquiry: 2527 8898)

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

1. Screening for CKM syndrome should begin only at age 45.
2. Waist circumference and Relative Fat Mass (RFM) index may be more useful than BMI in detecting adiposity especially in Asian populations.
3. The PREVENT equation is preferred over the Pooled Cohort Equations for estimating 10-year cardiovascular risk in asymptomatic individuals.
4. Coronary artery calcium (CAC) scoring is recommended as a routine test for all patients in primary care.
5. Screening for eGFR and urine albumin-to-creatinine ratio (ACR) is often overlooked but should be reinforced in at-risk groups.
6. GLP 1 receptor agonists and bariatric surgery may be considered for weight management in patients with BMI ≥ 27.5 kg/m^2.
7. NP/NT-proBNP and high-sensitivity troponin are routinely recommended for all patients in primary care to detect subclinical heart failure.
8. Checking blood test for renal function annually is adequate to detect the kidney involvement of CKM syndrome.
9. SGLT2 inhibitors and Finerenone are recommended therapies for patients with heart failure and CKD.
10. All patients with CKM syndrome should receive statin and aspirin.

ANSWER SHEET FOR APRIL 2026

Please return the completed answer sheet to the Federation Secretariat on or before 30 April 2026 for documentation. 1 CME point will be awarded for answering the MCHK CME programme (for non-specialists) self-assessment questions.

Managing Cardiovascular-kidney-metabolic (CKM) in the General Practice Setting

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Prince of Wales Hospital, Chinese University of Hong Kong

Answer Link

1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10 ☐

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Answers to March 2026 Issue

Out-of-Hospital Cardiac Arrest in Hong Kong: Current Challenges and Future Strategies

1. T 2. F 3. T 4. F 5. T 6. T 7. T 8. F 9. T 10. T

Your CKD and HF patients win twice when you manage hyperkalaemia long term* and maintain RAASi cardiorenal protection[1,2]

Rapid serum K^+ reduction **within 1 hour** of a single dose[2]

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*Up to 12 months based on the prespecified exploratory analysis of changes in RAASi dose during the maintenance phase of a phase III, open-label, single-arm, 12-month study.

^73.5% of participants in the ZORA study stabilized or uptitrated their RAASi dose with LOKELMA™. ZORA was a multi-country, observational cohort study comparing the likelihood of maintained RAASi therapy at 6 months following an episode of hyperK in patients with CKD and/or HF. Participants either received no K^+ binder prescription, or had ≥120 days of LOKELMA™ treatment.

†Long-term is defined as LOKELMA™ treatment of over 90 days.

#Risk reduction is compared with patients who had received LOKELMA™ treatment of ≤90 days.

‡Only the 5g and 10g doses of LOKELMA™ has been approved in Hong Kong. The 15g dose of LOKELMA™ is not available in Hong Kong.

Study design: HARMONIZE (ZS004) is a phase III, multicentre, multiphase, placebo-controlled study in 258 patients with hyperK. Open-label phase: LOKELMA™ 10g TID, administered for 48 hours, at which time patients (n=237) with normokalaemia (3.5–5.0mmol/L) were randomised to LOKELMA™ or placebo, 5g, 10g or or 15g‡ QD for 28 days. Primary endpoint: mean serum K^+ level with LOKELMA™ vs. placebo on days 8–29. Eligible patients then continued treatment with LOKELMA™ 10g QD.[2] In a single-arm open-label extension study, patients from HARMONIZE with K^+ 3.5-6.2mmol/L received LOKELMA™ 5-10g QD for ≤337 days. In the CP, patients with K^+ >5.5mmol/L received LOKELMA™ 10g TID before entering the MP while patients with K^+ 3.5-5.5mmol/L immediately entered the MP phase and began LOKELMA™ 10g QD. Endpoints included achievement of mean serum ≤5.1mmol/L (primary) or ≤5.5mmol/L (secondary).[2]

Study design: The RECOGNIZE I is a retrospective noncomparative study using claims data from the HealthVerity warehouse, which included outpatients in the United States who had ≥6 months of continuous LOKELMA™ treatment. They continued to receive 6 months of LOKELMA™ for a total coverage of 12 months. The study aimed to describe healthcare resource utilization with long-term (>90 days' supply; n=405) and short-term (<90 days' supply; n=748) LOKELMA™ and identify characteristics associated with long-term vs. short-term therapy.[4]

Abbreviations: AE: Adverse event; CKD: Chronic kidney disease; CP: Correction phase; HF: Heart failure; HyperK: Hyperkalaemia; HypoK: Hypokalaemia; K^+: Potassium; MP: Maintenance phase; QD: Once daily; RAASi: Renin–angiotensin–aldosterone system inhibitors; TID: 3 times daily.

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Peripheral Arterial Disease Awareness: Clinical Importance, Gaps, and Strategies for Improvement

Prof Bryan PY YAN

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Peripheral arterial disease (PAD) is a common manifestation of systemic atherosclerosis characterised by arterial stenosis or occlusion of the lower extremities, leading to ischemia and increased risk of cardiovascular morbidity and mortality. Despite its prevalence - affecting an estimated 200 million people worldwide - PAD remains underdiagnosed and undertreated, partly due to limited awareness among patients and healthcare providers, atypical symptom presentation, and gaps in screening practices[1,2]. Enhanced awareness is essential for early diagnosis, risk modification, and the reduction of adverse limb and cardiovascular outcomes.

EPIDEMIOLOGY AND CLINICAL SIGNIFICANCE

PAD prevalence increases with age and is strongly associated with traditional cardiovascular risk factors, including smoking, diabetes mellitus, hypertension, and dyslipidaemia[3]. Importantly, PAD is a potent marker of systemic atherosclerotic burden: patients with PAD face a two- to threefold increased risk of myocardial infarction, stroke, and cardiovascular death compared with age-matched controls[4]. Critical limb-threatening ischemia (CLTI), the most severe PAD phenotype, carries high rates of limb loss and mortality if not promptly recognised and managed[5]. The burden of PAD is disproportionately higher in populations with limited access to care and among patients with diabetes, underscoring health equity considerations.

BARRIERS TO AWARENESS AND DIAGNOSIS

Multiple factors contribute to the under-recognition of PAD. First, symptoms may be absent (asymptomatic PAD) or atypical; classic intermittent claudication is reported in only a minority of patients[6]. Second, primary care clinicians often underutilise simple diagnostic tests such as the ankle-brachial index (ABI), due to time constraints, lack of equipment or training, and variable guideline implementation[7]. Third, patient-level factors - including misattribution of leg pain to ageing or musculoskeletal disease, low health literacy, and limited access to vascular specialty services - delay presentation and diagnosis. Our group recently conducted a local survey, which identified a critical gap in PAD awareness in Hong Kong[8]. Of 1,008 participants, only 36.7 % were aware of PAD, substantially lower than awareness for diabetes, hyperlipidaemia, hypertension, coronary artery disease and cerebrovascular disease. Among PAD-aware patients, 84 % associated the disease with walking difficulties, but only 50.0 % acknowledged that PAD could be fatal. The elderly, less-educated, and pre-atherosclerotic patients exhibited the least awareness of PAD. These barriers result in missed opportunities for secondary prevention and timely limb preservation strategies.

DIAGNOSTIC STRATEGIES

The ABI remains a validated, inexpensive, and non-invasive screening test for PAD and should be performed in patients with exertional leg symptoms, nonhealing lower extremity wounds, or age- and risk factor-based thresholds (e.g., age > 65 years; > 50 years with history of smoking or diabetes)[9]. An ABI ≤ 0.90 is diagnostic of PAD, while ABI values > 1.30 may indicate medial arterial calcification, particularly in diabetes and chronic kidney disease, necessitating further evaluation with toe-brachial index (TBI) or duplex ultrasonography[10]. Duplex ultrasound, CT angiography, and MR angiography are used for anatomical mapping when revascularisation is considered. Awareness initiatives should emphasise appropriate use and interpretation of these diagnostic modalities.

MANAGEMENT AND SECONDRARY PREVENTION

Management of PAD involves symptom control, functional improvement, risk factor modification, and timely revascularisation when indicated. Medical therapy with antiplatelet agents (e.g., aspirin or clopidogrel) and high-intensity statin therapy is foundational for reducing cardiovascular risk[11]. Structured exercise programmes, particularly supervised exercise therapy, are effective in improving walking distance and quality of life in patients with claudication[12]. Revascularisation - endovascular or surgical - should be considered in patients with lifestyle-limiting claudication refractory to conservative therapy or in those with CLTI after multidisciplinary evaluation.

STRATEGIES TO IMPROVE AWARENESS

Improving PAD awareness requires coordinated actions at the patient, clinician, and health system levels. Patient education campaigns should highlight PAD symptoms, risk factors, and the prognostic significance of diagnosis. Our group recently demonstrated strong

diagnostic accuracy in detecting symptomatic PAD using a self-administered claudication plantarflexion test (CPT), a combination of a single claudication question ("Do you have pain in either leg or calf muscle on walking?") and an active pedal plantarflexion test, which involves 30 seconds of repeated active ankle plantarflexion while standing[13]. As the CPT requires no specialised equipment or training, it holds potential for large scale screening programmes and may serve as a valuable triage tool for prioritising specialist referrals for PAD diagnosis. Use of targeted screening protocols in primary care - incorporating ABI measurement or CPT for high-risk groups - can improve case detection; training and incentivising primary care teams to perform ABI may overcome barriers[7,13]. Clinical decision support integrated into electronic health records can prompt screening and guideline-concordant therapy. Multidisciplinary limb preservation programmes that link primary care, podiatry, vascular surgery, and wound care facilitate timely referral and coordinated management, reducing amputation rates[14]. Finally, public health initiatives should address disparities by allocating resources to underserved communities and promoting tobacco cessation and diabetes control.

RESEARCH AND POLICY PRIORITIES

Research priorities include implementation studies to identify effective models for ABI or CPT screening in primary care, cost-effectiveness analyses of screening in different risk groups, and trials testing interventions to improve referral pathways and minimise time to revascularisation for CLTI. Policy efforts should support reimbursement for ABI testing in primary care, fund workforce training in vascular diagnostics, and incentivise development of integrated limb care networks.

CONCLUSION

PAD is a common, high-risk manifestation of systemic atherosclerosis that is frequently under-recognised. Improving awareness through education, targeted screening, guideline-based secondary prevention, and integrated care models can reduce cardiovascular events and limb loss. Concerted action at clinical and health system levels is required to close current gaps in diagnosis and management and to improve outcomes for patients with PAD.

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Advances in Atrial Fibrillation Management

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Atrial fibrillation (AF) remains the most prevalent sustained arrhythmia worldwide and continues to impose a significant burden through increased risks of stroke, heart failure, hospitalisation, and impaired quality of life[1,2]. As populations age and cardiovascular risk factors become more common, AF management has undergone substantial evolution. In the past decade alone, technological innovation, deeper understanding of disease mechanisms, and new therapeutic modalities have transformed clinical practice. The following discussion highlights five major areas where progress has been particularly impactful - diagnostics and monitoring, stroke prevention, catheter ablation, risk factor modification, and integrated models of care. Each of these domains contributes to a paradigm shift toward earlier detection, personalised therapy, and comprehensive management.

MODERN DIAGNOSTIC AND MONITORING TECHNOLOGIES

One of the most significant transformations in AF management has been the advancement of diagnostic tools. Traditional monitoring approaches, such as 24- hour Holter monitors, often miss paroxysmal episodes or asymptomatic arrhythmia, leading to underdiagnosis and delayed treatment. Today, an expanded ecosystem of digital health devices has revolutionised detection. Wearable sensors - including smartwatches, fitness trackers, smartphone connected ECG modules, and adhesive patch monitors - enable continuous or intermittent rhythm surveillance over days to weeks[3]. These devices capture rhythm disturbances in real time and can detect subtle, transient episodes previously missed by conventional methods.

Artificial intelligence has further amplified diagnostic precision[4]. Machine learning algorithms embedded and consumer and medical devices can differentiate between noise and true arrhythmia, detect irregular ventricular response patterns, and identify "ECG fingerprints" associated with future AF risk even during sinus rhythm. These predictive capabilities are especially important for early intervention, offering opportunities to prevent progression from brief paroxysms to more sustained AF. Expanded screening initiatives - whether opportunistic, targeted to high risk groups, or implemented at the population level - have also improved case detection, helping clinicians intervene earlier with anticoagulation and rhythm control strategies. Overall, modern monitoring technologies not only enhance diagnostic yield but also increase patient engagement by allowing individuals to take an active role in their own cardiac surveillance.

STROKE PREVENTION AND EVOLVING ANTICOAGULATION STRATEGIES

Stroke prevention remains a cornerstone of AF management, and major advances have been made in this field[1,2]. The introduction and widespread adoption of direct oral anticoagulants (DOACs) have transformed treatment. Agents such as apixaban, rivaroxaban, dabigatran, and edoxaban offer several advantages over warfarin: predictable pharmacokinetics, no need for routine INR monitoring, fewer dietary interactions, and reduced rates of intracranial haemorrhage. These benefits have improved patient adherence and outcomes, simplified long term anticoagulation for both patients and clinicians.

However, oral anticoagulation is not suitable for all patients - especially those with high bleeding risk or contraindications. In such cases, left atrial appendage occlusion (LAAO) devices have emerged as an essential alternative[5]. Technological refinement has resulted in more secure implant designs, improved deployment techniques, and lower procedural complication rates. Newer generations of LAAO devices provide reliable stroke prevention comparable to anticoagulation in selected patients. The growing body of evidence supporting LAAO has expanded its use from a niche therapy to a mainstream consideration within individualised stroke prevention pathways.

Additionally, improvements in risk stratification tools allow clinicians to more precisely balance stroke risk against bleeding risk. Biomarkers, imaging markers such as atrial fibrosis, and refined clinical scoring systems enhance decision making and contribute to a more personalised anticoagulation strategy[6]. Together, these advancements signify a major step forward in safeguarding patients from one of AF's most devastating complications.

BREAKTHROUGHS IN CATHETER ABLATION, PARTICULARLY PULSED-FIELD ABLATION

Catheter ablation has increasingly become central to rhythm control therapy, especially for patients with symptomatic, recurrent AF. Although radiofrequency (RF) and cryoballoon ablation long served as the primary energy modalities, the recent emergence of pulsed field ablation (PFA) marks a paradigm shift[7]. Unlike thermal ablation, which carries risks of collateral damage to the oesophagus, phrenic nerve, and pulmonary veins, PFA uses high frequency electrical pulses to induce targeted electroporation. This mechanism selectively affects myocardial tissue while sparing adjacent structures, dramatically improving procedural safety. However,

haemolysis and coronary damage are two potential complications unique to PFA[8].

Early multicentre trials and real world experience indicate that PFA is associated with shorter procedure times, faster lesion formation, lower complication rates, and quicker patient recovery[7,9]. These characteristics not only improve the patient experience but may also allow electrophysiology centres to treat more individuals efficiently. Furthermore, the consistency of lesion creation reduces variability and may improve long-term rhythm outcomes.

Beyond PFA, advancements in high resolution electroanatomic mapping systems, contact force sensing catheters, and imaging integration (e.g., CT or MRI fusion) have enhanced procedural precision. The growing trend toward earlier ablation - supported by clinical evidence showing that early rhythm control can prevent AF progression and reduce cardiovascular events - reflects an important evolution in management philosophy. In combination, these procedural and technological innovations have elevated catheter ablation into a more effective, safer, and more accessible treatment option. Combination with LAAO implantation within a same procedure has become a viable approach[10,11].

RISK-FACTOR MODIFICATION AND COMORBIDITY MANAGEMENT AS CORE THERAPY

A major insight over the last decade is the recognition that AF is not merely an electrical disorder but also the product of structural, metabolic, and inflammatory processes[12]. As such, targeting underlying risk factors has become a central pillar of contemporary AF management.

Weight reduction, particularly through structured programmes involving dietary modification and increased physical activity, has demonstrated profound benefits. Studies show that even modest weight loss can reduce AF burden, improve symptom severity, and enhance ablation success. Similarly, hypertension control - especially through ambulatory blood pressure monitoring, combination therapy, and treatment of resistant hypertension - is essential, given the strong association between elevated blood pressure and atrial remodelling.

Obstructive sleep apnoea (OSA) is another major contributor to AF initiation and recurrence. Continuous positive airway pressure (CPAP) therapy significantly decreases AF recurrence after ablation and improves overall cardiovascular outcomes. Optimising glycaemic control in diabetes, moderating alcohol consumption, and promoting cardiovascular fitness are part of an expanded management approach that aims to address modifiable drivers of AF.

This holistic perspective signifies a major shift from symptom-driven management to disease-modifying therapy. Integrated risk factor modification not only reduces arrhythmia burden but also improves broader cardiometabolic health.

INTEGRATED CARE MODELS AND PATIENTS-CENTRED MANAGEMENT

The final major advance in AF care relates to how services are organised and delivered. Increasingly, AF is managed not in isolated specialty clinics but through multidisciplinary, integrated care pathways[1,2]. These models involve close collaboration among electrophysiologists, cardiologists, primary care physicians, sleep specialists, dietitians, and lifestyle intervention teams. Evidence demonstrates that such integrated systems improve survival, reduce hospitalisation, enhance guideline adherence, and improve patient satisfaction.

Telehealth and digital platforms further support integrated care by enabling remote rhythm monitoring, virtual follow up visits, digital symptom diaries, and continuous communication between patients and their clinical teams. This infrastructure fosters long term engagement and allows rapid adjustments to management as symptoms evolve.

Equally important is the rise of shared decision-making, which ensures that patients understand the risks and benefits of treatment options such as anticoagulation, ablation, or lifestyle modification. With an expanding array of therapies available, aligning medical decisions with patient preferences and life goals is essential. This patient-centred approach improves adherence, empowers individuals, and ultimately enhances outcomes.

CONCLUSION

In summary, AF management has advanced dramatically across five major domains: innovative diagnostic tools, improved stroke prevention strategies, safer and more efficient catheter ablation technologies, comprehensive risk factor modification, and integrated multidisciplinary care. Collectively, these developments mark a shift toward proactive, personalised, and holistic management. As technologies continue to mature and clinical evidence expands, AF care is poised to become even more precise, effective, and patient centred - ultimately improving outcomes for the millions affected worldwide.

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Endovenous Intervention: From the Legs to the Lungs

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INTRODUCTION

Venous diseases encompass a wide array of vascular pathologies that affect the venous vasculature in the circulatory system. While venous thromboembolism (VTE) makes up a majority of the conditions presented to physicians, venous disease can also come in the form of vascular stenosis or vascular compression. Venous diseases can be classified by their chronicity into acute or chronic, by their underlying pathology into thromboembolic and stenotic, and by their location into peripheral, central, or pulmonary. While acute pulmonary embolism (PE) represents the most urgent type of venous disease, other forms of venous disease, such as chronic venous insufficiency (CVI) or chronic thromboembolic pulmonary hypertension (CTEPH), can be associated with significant morbidity and impairment of quality of life[1]. Significant advancements in technology and technique of endovenous intervention have enabled safe and effective treatments of various venous pathologies. In this review, we will discuss the general principles of endovenous intervention and how to apply them in the management of various venous diseases[1-4].

PRINCIPLES OF ENDOVENOUS INTERVENTION

Endovenous intervention works on the basis of endovascular interventions with a few key differences from arterial intervention, stemming from differences in venous vascular structure and underlying pathology from the arterial system.

A vein is a thin-walled vascular structure, and a low-pressure system. Intrinsic vascular support is relatively weaker than the arterial system, and is prone to extrinsic compression, especially by pulsatile arterial structures. Therefore, plain-old-balloon-angioplasty (POBA) tends to yield less durable luminal gain, especially in chronically occluded veins, and a metallic scaffold is often needed to provide extrinsic support and to maintain luminal patency. Intraluminal venous pressure is usually close to 0 mmHg at the peripheral, and the draining of blood inside the vein depends greatly on the patency of the flow circuit and the normal functioning of venous valves, which are present in the peripheral veins outside the thoracoabdominal cavity. Restoration of the patency of the venous conduit with approximation to normal circumference is therefore important to re-establish normal venous circulation. Intravascular imaging, such as intravascular ultrasound (IVUS), has been shown to be useful in determining the appropriate stent size and identifying extrinsic compression in endovenous intervention[1]. Unlike atherothrombotic occlusion in the arterial system,which is a progression of arterial intimal atherosclerosis, the occlusion in the venous system is usually the result of acute thromboembolism or failure of thrombus resolution in the chronic stage. While thrombotic occlusions are soft and can be easily recanalised in the acute stage, chronic organised venous thrombus are fibrinous and hard, making wires crossing and recanalisation more challenging. Moreover, chronic venous occlusion is usually associated with extensive fibrotic changes and scarring of the diseased segment, further mandating metallic scaffolding in maintaining luminal patency. Most of the stents used in endovenous intervention are self-expanding metallic stents, which have high radial force for scaffolding support and flexibility to accommodate the tortuous venous anatomy. Due to the underlying prothrombotic predisposition, antithrombotic choices postprocedure are less well defined than in post-arterial intervention.

ENDOVENOUS INTERVENTION FOR ACUTE VENOUS THROMBOSIS

VTE is the commonest type of venous disease, with an annual incidence of 55 to 117 per 100,000 person years. The cornerstone of VTE treatment is anticoagulation, which can retard thrombus propagation and prevent thrombus embolisation. Conservative measures such as leg elevation and compression stockings can help resolve symptoms. However, in a selected group of patients, especially if the obstruction is more proximal at the ilio-femoral region, and if the symptoms are severe, such as those presenting with venous gangrene or plegmasia cerulae dolen, additional endovenous therapy should be considered to provide more rapid symptom relief, and to prevent the development of acute limb ischemia and post-thrombotic syndrome[2,5].

There are two endovenous treatment modalities for acute DVT: pharmacological using catheter-directed thrombolysis (CDT), or mechanical thrombectomy using various aspiration technologies. During CDT,

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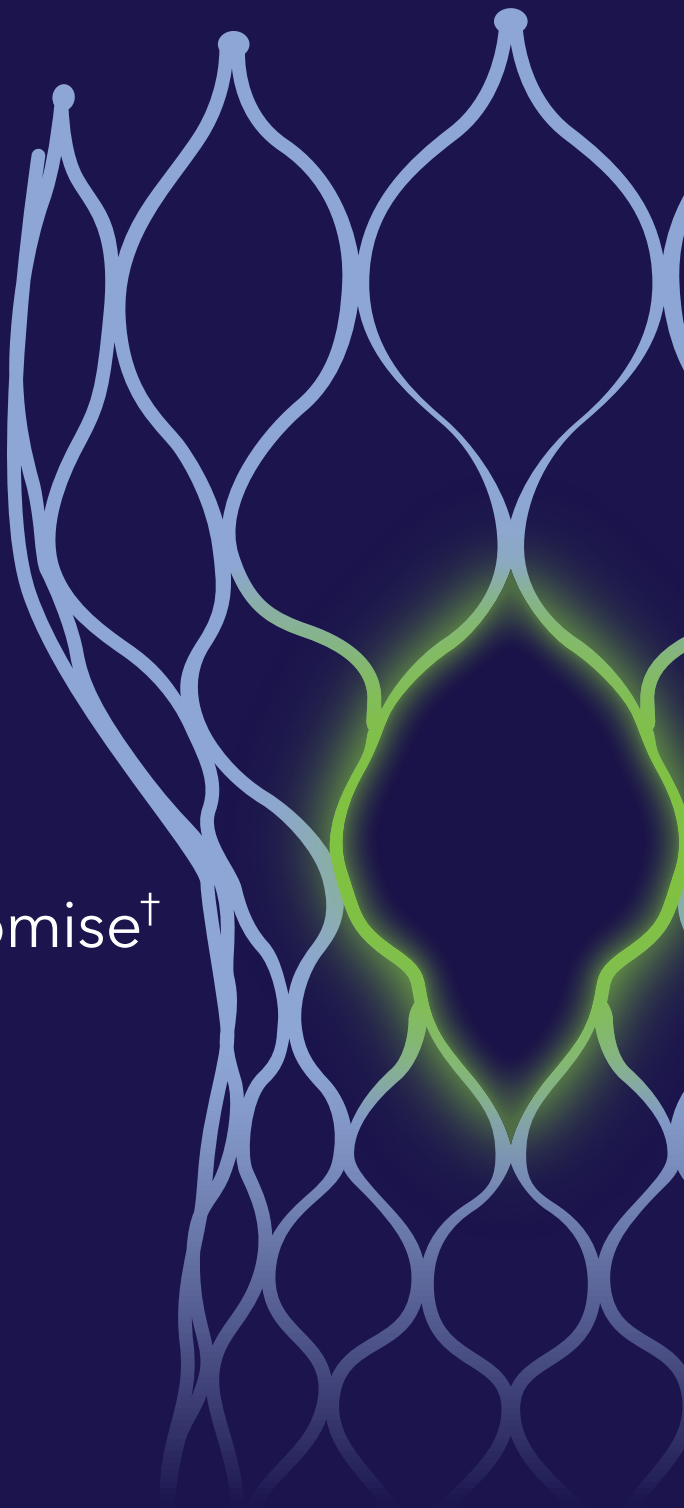

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thrombolytic agents are infused directly into the thrombus using either a multi side-holes infusion catheter, or via the EKOS system, which is equipped with ultrasonic probes to facilitate the delivery of thrombolytics. While CDT is effective in lysing most of the acute thrombus, infusion of thrombolytics is time-consuming, and is less effective against thrombus that is organised such as that found in subacute presentation. In mechanical thrombectomy, thrombus is removed directly by aspiration using either a vacuum suction, such as in the Penumbra system, or by the Venturi effect generated from a high velocity saline jet in the Angiojet system[6,7].

Additionally, thrombus can be broken down mechanically before aspiration by either a separator or water-jet, such as in the Jeti system. The Angiojet system has an additional advantage of being able to infuse thrombolytics using the Power-spray function and thus combining both treatment modalities to achieve a single-stage pharmaco-mechanical thrombectomy procedure. This has been shown to be safe and effective as a single-stage procedure in the treatment of phlegmasia[8].

ENDOVENOUS INTERVENTION FOR CHRONIC LIMB VENOUS INSUFFICIENCY

Post-thrombotic syndrome (PTS) is the most common long-term complication of acute DVT, affecting an estimated 20-50% of DVT patients. The symptoms and clinical signs of PTS include chronic pain, intractable limb oedema, varicose veins, venous claudication, hyperpigmentation, and venous ulcers. PTS significantly affects patient's QoL and imposes a hefty burden on the healthcare system. While conservative management, such as compression therapy, can be considered in patients with mild PTS, invasive interventions, especially endovenous venoplasty, can be offered to appropriately selected patients to reduce the symptoms of moderate to severe PTS[1].

Unlike the thrombotic occlusion in acute DVT, venous obstruction in PTS is a result of chronic organised thrombus and fibrotic remodelling of the venous structures. Therefore, endovenous treatments, such as CDT or mechanical thrombectomy, are less effective in recanalisation and restoring venous flow. Instead, venoplasty with POBA to break-up the organised thrombus, and metallic stents for scaffolding are usually required to restore the luminal patency. Moreover, extrinsic compressions at the iliac vein can be demonstrated in a significant proportion of patients suffering from severe PTS, making metallic scaffolding an important treatment modality in maintaining vascular patency in these patients[1].

ENDOVENOUS INTERVENTION FOR CENTRAL VENOUS STENOSSIS

Central veins refer to both the thoracic central veins (the superior vena cava) and the abdominal central veins (the inferior vena cava). Unlike the peripheral veins, the most common aetiology of central venous occlusion is extrinsic compression by malignancy, followed by chronic fibrotic stenosis from indwelling catheters or devices. Surgical reconstruction or bypass used to be the gold standard for the treatment of central venous stenosis. However, surgery is associated with major perioperative mortality and complications. Endovenous venoplasty with or without stenting has been proposed as an alternative to surgical repair with comparable mid-term and long-term patency rates[9]. Due to the large luminal size of central veins, usually special metallic stents with strong radial force are required to provide adequate scaffolding support.

ENDOOVENOUS INTERVENTION FOR ACUTE PULMONARY EMBOLISM

Acute pulmonary embolism is the third leading cause of cardiovascular mortality, with an annual incidence of 62 to 112 per 100,000 persons year. While the majority of acute PE are low risk and can be treated conservatively, some patients, especially those with intermediate to high-risk PE with evidence of right ventricular dilatation, will benefit from additional endovenous intervention to expedite patient recovery and prevent haemodynamic decompensation[3,4]. The goal of endovenous intervention is to remove or reduce the thrombus burden in the pulmonary arteries and relieve the pressure on the RV, speeding up patient recovery and potentially reducing the incidence of subsequent CTEPH. This goal can be achieved by either infusing low-dose thrombolytics via a specialised catheter empowered with ultrasonic emission (the EKOS system)[10], or by mechanically removing the thrombusby aspiration (the Penumbra system and the Flowtriever system)[11,12].

All of these procedures involve endovenous access, usually from the femoral veins. During the EKOS procedure, the ultrasonic catheters are placed at the targeted pulmonary arteries for direct infusion of thrombolytics to the targeted pulmonary artery with the assistance of ultrasound, which enhances the penetration of thrombolytics into the thrombus and facilitates the fibrinolytic actions. With the ultrasonic assistance, the dosage and the duration of thrombolytics infused are typically less than conventional CDT. The safety and clinical efficacy of EKOS in terms of RVD resolution have been reported in various registries and randomised controlled trials[10,13]. The penumbra system comprises of aspiration catheters of varied sizes (CAT 6, CAT 8, CAT 12), with the bigger size providing greater aspiration, a separator, and a vacuum pump generating a power suction. Accelerated thrombus clearance and resolution of RVD were reported in the EXRTACT-PE and STORM-PE study[11,14]. While both treatment modalities convey a similar safety profile and clinical efficacy, several factors need to be considered in choosing the appropriate device for the patients. Firstly, operator expertise and familiarity with the device are important factors in selecting the device. Secondly, the haemodynamic status will determine the urgency of thrombus removal and pulmonary artery recannalisation. The clinical improvement from CDT is not immediate as it requires time for the thrombolytics to act. In contrast, mechanical aspiration can achieve rapid thrombus removal and haemodynamic improvement during the procedure. Last but not least, mechanical aspiration

might be more suitable for patient with high bleeding risk and contraindications to thrombolytics, as it can be performed heparin and lytics-free.

ENDOVENOUS INTERVENTION FOR CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION

CTEPH is one of the most serious chronic complications of acute PE. The incidence of CTEPH was reported to affect 5 - 7 % of patients with acute PE. However, with enhanced awareness and screening, the incidence of CTEPH has been shown to increase in recent years. Similar to PTS in DVT, the pathophysiology of CTEPH is the occlusion of the pulmonary artery from the organised thrombus, with additional involvement from pulmonary vascular and right ventricular remodelling. Therefore, besides endovenous intervention, comprehensive background medical therapy for pulmonary hypertension and management by a multi-disciplinary pulmonary hypertension unit are essential for the care of patients with CTEPH[15,16].

The gold standard treatment of CTEPH was surgical bilateral pulmonary endarterectomy, which, although it offers the best chance to improve long-term clinical outcomes, is limited by surgical expertise and is associated with significant peri-operative mortality. Endovascular Balloon pulmonary angioplasty (BPA) has been introduced recently as an alternative to surgical endarterectomy, especially for patients with involvement of more distal pulmonary artery and for whom surgical risk is estimated to be high. BPA involves standard angioplasty procedures using conventional coronary equipment, such as guiding catheters, guidewires and monorail balloon. BPA aims to disrupt organised, flow-limiting obstructions and improve pulmonary blood flow. Stenting is generally not encouraged in the exception of stent graft for the treatment of vascular perforation of a large vessel. Typically, several BPA sessions are required to produce any meaningful clinical improvement, as only 1-2 pulmonary lobes can be treated each time. The commonest periprocedural complications are pulmonary vascular injury and reperfusion pulmonary oedema, which occur more frequently in patients with more disease burden and higher baseline pulmonary arterial pressure. Several risk scoring systems, such as the PEPSI score[17], have been developed to predict which patients are at a higher risk of developing complications. Detailed pre-procedural planning and careful patient selection, therefore, are important to avoid these serious complications. Comparable clinical outcomes and haemodynamic improvements to those seen with surgical endarterectomy can be expected from successful BPA.

CONCLUSION

Venous disease can impart significant morbidity and mortality to patients if left untreated. Significant advancements in endovenous technology have enabled effective treatments of various venous diseases with minimal complications. In selected patients, endovenous intervention can significantly improve clinical outcomes and reduce disease burdens.

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Updates in the Management of Valvular Heart Disease: A Summary of the latest European Society of Cardiology Guidelines

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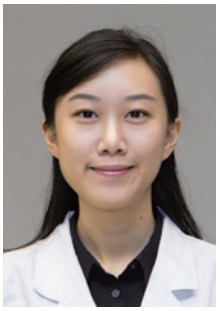

Dr Vyanne HT CHAN Dr Kent CY SO

Valvular heart disease (VHD) is a public health challenge worldwide. In Hong Kong, general practitioners (GPs) play a pivotal role in early identification, initial management, and timely referral of patients with VHD. The 2025 ESC/EACTS Guidelines for the Management of Valvular Heart Disease[1] emphasise a patient-centred, multidisciplinary approach. This review summarises key updates in the diagnosis and management strategies of VHDs.

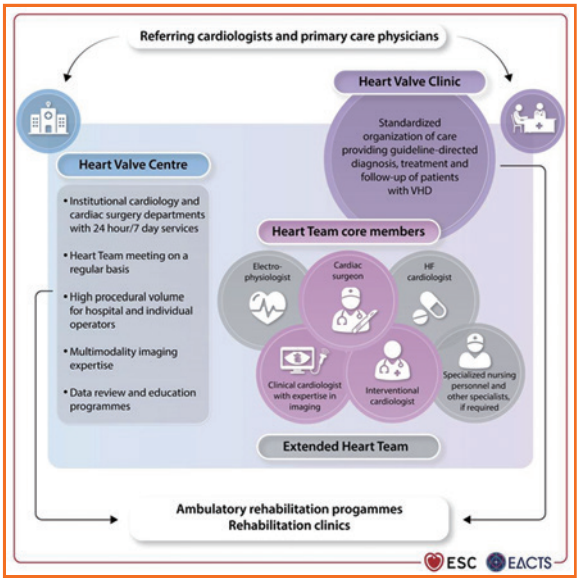

Fig. 1: The 2025 ESC/EACTS Guidelines for the Management of Valvular Heart Disease emphasise a patient-centred, multidisciplinary approach (Excerpted from the 2025 ESC/EACTS Guidelines for the management of valvular heart disease)

GENERAL PRINCIPLES

Early Recognition and Referral are crucial. Routine auscultation and vigilance for heart failure symptoms, including dyspnoea, reduced exercise tolerance, and syncope, are essential.

Imaging with transthoracic echocardiography (TTE) is the first-line investigation for suspected VHD. Transoesophageal echocardiography (TEE), cardiac computed tomography (CCT) and cardiac magnetic resonance can provide complementary assessment of VHDs.

Heart Valve Centres referral should be initiated if VHD is suspected or diagnosed. The updated guidelines advocate a Heart Team approach. This multidisciplinary team involves cardiac imaging specialists, interventional cardiologists, cardiac surgeons, and heart failure specialists. Timely referral to a Heart Valve Centre allows patient to receive comprehensive care.

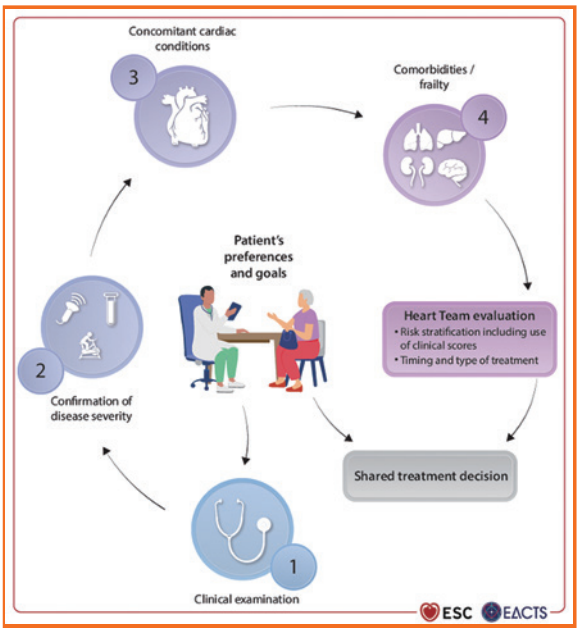

Fig. 2: ESC/EACTS Guidelines for the management of valvular heart disease (Excerpted from the 2025 ESC/EACTS Guidelines for the management of valvular heart disease)

SPECIFIC VHD

1.Aortic Stenosis (AS)

Clinical presentation of AS

AS is highly treatable but carries a poor prognosis if the diagnosis is delayed. Typical symptoms include angina, heart failure or syncope.

Diagnosis of AS

AS can be diagnosed with TTE. Severe AS is defined by: peak aortic jet velocity ≥ 4.0 m/s, mean gradient ≥ 40 mmHg, and aortic valve area ≤ 1.0 cm². Low-flow, low-gradient AS is a entity with impaired left ventricular ejection fraction (LVEF), and gradients are usually insufficient for diagnosis. This group requires investigations, including dobutamine stress echocardiography and CCT.

MANAGEMENT OF SEVERE

- **When to Intervene:** Intervention is strongly recommended in patients with severe symptomatic AS. In asymptomatic patients, early intervention can reduce heart failure hospitalisation and death risks[2]. Early intervention is indicated if LVEF ≤ 50 % and should be considered in patients with low procedural risk, or in very severe AS (mean gradient ≥ 60 mmHg), or severe aortic valve calcification. Careful assessment of symptom status is essential and includes exercise testing and measurement of natriuretic peptides (BNP and NTproBNP).

- **How to intervene:** Definitive treatment includes surgical aortic valve replacement (SAVR) and transcatheter aortic valve implantation (TAVI). Treatment modality should be individualised and guided by Heart Team, considering patient's age, surgical risk, comorbidities, anatomy and patient preference.

  - **Age and Risk:** TAVI is favoured in patients aged ≥ 70 years. In younger patients with low surgical risk, SAVR remains the preferred option.

  - **Anatomical Suitability:** SAVR is favoured in patients with bicuspid aortic valve disease with associated aortopathy, very large annular dimensions, or extensive valve calcification. Conversely, certain features, e.g., porcelain aorta, increase surgical risks and favour a transcatheter approach.

- **Lifetime management of AS:** With the expansion of transcatheter therapies to younger and lower-risk patients, the concept of lifetime management of AS is increasingly important[3]. Treatment decisions should focus on long-term strategy over a patient's remaining lifespan. Key considerations include valve durability and the anticipated need and feasibility for future re-intervention.

Factor	SAVR Preferred	TAVI Preferred
Age/Risk	< 70 y, low risk (durability priority)	≥ 70 y, high risk
Anatomy	Bicuspid + aortopathy, large annulus, high coronary obstruction risk	Porcelain aorta

2. Aortic Regurgitation (AR)

Clinical presentation of AR

Chronic AR presents with exertional dyspnoea, reduced exercise tolerance and fatigue. In younger patients, it can be associated with underlying connective tissue disease such as Marfan syndrome. Acute AR is a distinct entity and is commonly related to medical emergencies such as infective endocarditis or aortic dissection.

Diagnosis of AR

Severe AR is defined by quantitative echocardiographic parameters including an effective regurgitant orifice area (EROA) ≥ 30 mm², a regurgitant volume (RV) ≥ 60 mL/beat, or a regurgitant fraction (RF) ≥ 50 %. Assessment should include evaluation of LVEF and left ventricular (LV) dimensions. As AR is frequently associated with aortopathy, CCT imaging allows accurate assessment of aortic dimensions and anatomy for intervention planning.

Management of AR

- **When to Intervene:** Patients with **acute severe AR** usually require urgent surgery. In **chronic severe AR**, surgical intervention is recommended for all **symptomatic** patients. Surgery is also indicated in **asymptomatic** patients with LVEF ≤ 50 % or significant LV dilatation, defined as an LV end-systolic diameter (LVESD) > 50mm. In patients with low surgical risk, earlier intervention may be considered. Concomitant aortic surgery may be required in cases with aortic root or ascending aortic dilatation.

- **How to Intervene:** Definitive treatment is aortic valve surgery, including SAVR and surgical aortic valve repair. TAVI may be considered for high-risk symptomatic patients with suitable anatomy for transcatheter intervention.

3. Mitral Stenosis (MS)

Clinical presentation of MS

MS is caused by rheumatic heart disease or degeneration related to mitral annular calcification (MAC). It is related to atrial fibrillation (AF) and thromboembolic events. It can cause secondary tricuspid regurgitation (TR) in advanced stages.

Diagnosis of MS

Severe MS is defined by a mitral valve area ≤ 1.5 cm², accompanied by an elevated mean transmitral gradient. Exercise echocardiography can unmask symptoms and haemodynamic changes during exertion.

Management of MS

- **Medical management:** Heart rate control and diuresis are the mainstay of management. For patients with AF, warfarin is the anticoagulant of choice.

- **When to Intervene: In symptomatic** patients with severe MS, Percutaneous Mitral Commissurotomy (PMC) is the first-line treatment if anatomy is favourable. Mitral valve (MV) surgery (repair or replacement) is recommended for patients unsuitable for PMC. In **asymptomatic** patients, intervention should be considered for patients at high thromboembolic risk or haemodynamic decompensation.

- **Approach to Intervene:** PMC is preferred for rheumatic MS if anatomically suitable. Anatomical favourability can be assessed by the Wilkins score, taking into account leaflet mobility, calcification, valve thickness and subvalvular thickening[4]. Concomitant significant mitral regurgitation (MR) is a contraindication for PMC. MV surgery can be pursued if PMC is not feasible. For patients with degenerative MS related to extensive MAC, transcatheter mitral valve implantation (TMVI) may be considered in experienced centres.

4. Mitral Regurgitation (MR)

Diagnosis of MR

MR can be classified into primary MR (PMR), caused by structural abnormalities of the MV leaflets, chordae, or papillary muscles, and secondary MR (SMR), resulting from LV or atrial remodelling. Severe MR is defined by EROA ≥ 40 mm^2 (or ≥ 30 mm^2 if elliptical regurgitant orifice area), RV ≥ 60 mL, and RF ≥ 50 %. TEE provides additional anatomical evaluation and delineation of the cause of MR, and plays a key role in pre-procedural planning before interventions.

Management of MR

- **When to Intervene:**
  - **PMR:** In **symptomatic** patients, MV surgery, preferably MV repair, is indicated. If patients are **asymptomatic**, MV surgery is recommended when LVEF ≤ 60 %, LVESD ≥ 40 mm or indexed LVESD ≥ 20 mm/m^2. It should be considered in low-risk patients with specific criteria (AF, SPAP > 50 mmHg, left atrial dilatation, concomitant moderate TR).
  - **SMR (Ventricular):** Optimisation of guideline-directed medical therapy (GDMT), including cardiac resynchronisation therapy (CRT), is indicated. If patients remain **symptomatic**, Transcatheter Edge-to-Edge Repair (TEER) is recommended if criteria are met (e.g., LVEF 20 - 50 %, LVESD ≤70 mm, SPAP ≤ 70 mmHg)[5]. MV surgery may be considered if the patient is unsuitable for TEER.
  - **SMR (Atrial):** In atrial SMR, patients should receive medical therapy for heart failure with preserved ejection fraction and AF. If they remain symptomatic, MV surgery with surgical AF ablation and left atrial appendage occlusion should be considered. TEER may be considered if patients are ineligible for surgery.

- **How to Intervene:** For PMR, MV surgery (MV repair or replacement) is preferred due to better durability. TEER is a less invasive option for high-risk PMR or SMR patients who are anatomically suitable.

Type	Symptomatic	Asymptomatic
PMR	Surgery (repair > replacement)	LVEF ≤ 60 %, LVESD ≥ 40 mm (LVESDi ≥ 20 mm/m^2); consider low-risk + AF/PH (> 50 mmHg)/LA dilatation/moderate TR
SMR (Ventricular)	GDMT/CRT; TEER if persistent (LVEF 20 - 50 %, LVESD ≤ 70 mm, SPAP ≤ 70 mmHg)	
SMR (Atrial)	HFpEF/AF therapy; surgery (AF ablation/ LAAO) or TEER if ineligible	

5. Tricuspid Regurgitation (TR)

Clinical presentation of TR

TR patients present with right-sided heart failure, including peripheral oedema and abdominal distension. As the disease progresses, hepatic congestion and symptoms of cardiorenal syndrome may develop due to right ventricular (RV) dysfunction. TR frequently co-exists with left-sided VHD or chronic pulmonary diseases.

Diagnosis of TR

Severe TR is defined by EROA ≥ 40mm^2, RV ≥ 45 mL/ beat, RF ≥ 50 %, or 3-dimensional vena contracta area ≥ 75mm^2. Aetiology of TR includes: primary TR with structural abnormalities, secondary TR caused by annular dilatation or leaflet tethering, or cardiac implantable electronic device-related TR.

Management of TR

- **When to Intervene:** In the presence of **left-sided VHD** requiring surgery, concomitant tricuspid valve surgery (repair preferred) is recommended in severe TR, and should be considered for moderate TR to avoid progression. In Isolated **symptomatic** TR, TV surgery is recommended in patients without severe RV dysfunction or pulmonary hypertension. In **asymptomatic** patients, TV surgery should be considered if there is RV dilatation or RV function deterioration.

- **How to Intervene:** TV surgery (repair preferred) is the treatment of choice. In patients with high risks for surgery, transcatheter TV treatment (repair or replacement) should be considered.

CONCLUSION

The 2025 ESC guidelines provide a framework for contemporary VHD management. Timely recognition and early referral to Heart Valve Centres are essential, and GPs play a crucial role in the initial care for patients with VHDs.

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SIADH: syndrome of inappropriate antidiuretic hormone secretion.

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Sotatercept for Pulmonary Arterial Hypertension

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CASE PRESENTATION

A 77-year-old male chronic smoker with a history of diabetes mellitus, hypertension, coronary artery disease treated with stenting to left circumflex artery and ramus intermediate artery in 2015, chronic obstructive pulmonary disease (COPD), and interstitial lung disease (ILD) presented on 19 January 2024 with shortness of breath (SOB) on exertion, which began following an episode of COVID-19 in May 2023 and worsened after a subsequent COVID-19 episode in April 2024. Before the two COVID-19 episodes, the patient had good exercise tolerance and could manage a 1-hour hike.

At presentation in January 2024, his blood pressure was 138/79 mmHg, with a pulse rate of 101 bpm, and his peripheral oxygen saturation (SpO^2) was 85 % while receiving supplemental oxygen at 3 L/min via a portable O_2 concentrator. The patient required an oxygen concentrator for both home use and outings. After minor activities such as bathing, his oxygen saturation would drop to 60 %, and it would take 20 minutes of rest for it to return to 90 %, placing him in World Health Organization (WHO) functional class (FC) III. His self-assessed grade of SOB was 8 out of 10, with 10 being the worst (Table 1).

Initially, the patient was treated for COPD with decompensation following the second episode of COVID-19 infection. However, his symptoms progressively worsened. In late July 2024, electrocardiography revealed sinus rhythm in V1 and right ventricular (RV) strain with T-wave inversion in the right precordial leads V1–4, suggesting increased right heart pressure. Echocardiography showed indicators of pulmonary hypertension (PH), including a D-shaped interventricular septum during diastole, an enlarged RV and right atrium, and moderate tricuspid regurgitation, with an elevated RV systolic pressure (RVSP) of 106 mmHg. His left ventricular ejection fraction was normal at 60 %. N-terminal pro-B-type natriuretic peptide (NT-proBNP) level was elevated at 2,958 pg/mL (Table 1).

Full lung function tests showed no obstructive or restrictive pattern. Still, markedly reduced diffusion capacity was present, excluding COPD and fibrosis as causes of dyspnoea. Computed tomography (CT) pulmonary angiography showed no evidence of pulmonary embolism, with the pulmonary arteries well opacified by contrast and no abnormal filling defects indicative of thrombus. Chest CT showed extensive emphysematous changes in both lungs, septal thickening intermixed with ill-defined patchy ground-glass opacities, and multiple tiny, calcified foci in the bilateral lung bases, the right middle lobe, and the lingula, indicating postinflammatory change in ILD.

Right heart catheterisation (RHC) was performed on 3 January 2025, which revealed a mean pulmonary arterial pressure (mPAP) of 34 mmHg, increased pulmonary vascular resistance (PVR) of 7.94 WU, pulmonary artery wedge pressure of 9 mmHg, mean right atrium pressure of 8 mm Hg, pulmonary artery oxygen saturation of 58 %, reduced cardiac output (Fick cardiac output, 2.3 L/min; Fick cardiac index, 1.38 L/min/m), and normal left ventricular end-diastolic pressure. PVR and PAP did not improve with 100 % oxygen therapy, and PVR did not decrease with vasoreactivity testing using adenosine infusion. Intracardiac shunt was excluded because there was no step-up in oxygen saturation.

The findings led to a diagnosis of idiopathic pulmonary arterial hypertension (PAH) with a high-risk stratification based on the three-strata model.

Table 1: The patient's improvements before and after the addition of sotatercept (Developed by authors)

	Before sotatercept	After sotatercept
Self-assessed grade of SOB	8/10	3/10
Time required for SpO^2 to return to 90 % after minor activities	20 min	10 min
6-minute hall walk	40 m (2 min only)	80 m (4 min only)
NT-proBNP	2,958 pg/mL	259 pg/mL
WHO FC	III	II–III
Risk status	High	Intermediate-high
Echocardiography		
RVSP	106 mm Hg	83 mm Hg
RA area	27 cm^2	18 cm^2
Pericardial effusion	Absent	Absent

FC = functional class; NT-proBNP = N-terminal pro-B-type natriuretic peptide; RA = right atrium; RVSP = right ventricular systolic pressure; SOB = shortness of breath; SpO^2 = peripheral oxygen saturation; WHO = World Health Organization

TREATMENT AND RESPONSE

Triple therapy consisting of sildenafil 80 mg thrice daily, macitentan 10 mg daily, and selexipag 1.6 mg twice daily was initiated from 31 July 2024 onward. Still, there was no significant clinical or echocardiographic response.

During a 6-minute hall walk test in March 2025, the patient had to stop after walking for only 2 minutes (covering 40 m) due to SOB, despite receiving supplemental oxygen at 3 L/min, and his SpO^2 dropped to 72 % (Table 1).

The first subcutaneous dose of sotatercept 16.2 mg was added to the current treatment on 17 April 2025. The dose was uptitrated to 38.84 mg on 8 May 2024 (second dose) and to 38.85 mg on 29 May 2025 (third dose).

After four 3-weekly injections, the patient showed definite clinical improvement, with a self-assessed grade of SOB improving from 8 to 3 out of 10. His SpO^2 returned to 90 % in a shorter time (10 minutes) after bathing. His 6-minute hall walk distance doubled to 80 m in 4 minutes, NT-proBNP level reduced to 259 pg/mL, and WHO FC improved to II–III (Table 1). Follow-up risk reassessment revealed intermediate- to high-risk status based on the four-strata model.

The patient tolerated sotatercept well, without experiencing any evident adverse events (AEs). His haemoglobin and platelet levels were within normal ranges throughout the treatment. Last seen on 19 June 2025, after 3 months of sotatercept treatment, his RVSP reduced to 83 mmHg (Table 1).

DISCUSSION

Faces of PH

PAH (WHO Group 1 PH) is a rapidly progressive and life-limiting disease characterised by proliferative remodelling of small pulmonary arteries and progressive luminal narrowing, ultimately leading to right heart failure and death[1-4]. WHO classifies PH into five groups based on underlying causes[3,4]:

- Group 1 (PAH)
- Group 2 (related to left heart disease)
- Group 3 (due to lung diseases or hypoxia)
- Group 4 (due to chronic pulmonary artery obstructions)
- Group 5 (unclear or multifactorial mechanisms)

PAH: Rare and Challenging to Diagnose

PAH is a rare type of PH that can be idiopathic, hereditary, induced by toxins or drugs, or associated with connective tissue diseases, HIV infection, portal hypertension, congenital heart disease, or schistosomiasis[4]. Based on a systematic analysis of the Global Burden of Disease Study 2021, the age-standardised prevalence of PAH was 2.28 cases per 100,000 population, translating to approximately 170 cases in Hong Kong[5].

On average, it takes 1–4 years from the onset of the first symptoms to confirm the diagnosis of PAH. As seen in our case, patients with PAH generally present with nonspecific symptoms, such as dyspnoea on exertion and rapid exhaustion. These symptoms may be attributed to general deconditioning or other comorbid cardiopulmonary conditions, making early diagnosis of PAH challenging[6].

Possible Clues for Correct Diagnosis

In our patient with multiple cardiopulmonary comorbidities, the diagnostic process was particularly challenging. However, worsening of symptoms following COVID-19 provided clues, as did comprehensive lung and heart assessments to exclude other potential causes of exertional dyspnoea, including lung and cardiac diseases (e.g., COPD, ILD, coronary artery disease)[3,7].

RHC is the gold standard for diagnosing and classifying PH. Based on the updated haemodynamic definition, PAH may be diagnosed in patients with a mean PAP >20 mmHg and PVR >2 WU[3].

Conventional Therapies Help, but Are They Enough?

The prognosis of PAH is typically poor, with a median overall survival (OS) of 2.8 years in untreated patients[8]. Conventional PAH therapies target the endothelin-1, nitric oxide, and prostacyclin pathways, which mainly promote vasodilation (Fig. 1)[9].

When given alone or in combination, these conventional treatments may enhance pulmonary haemodynamics, increase exercise capacity, and prolong progression-free survival in patients with PAH[2]. For example, the SERAPHIN and GRIPHON trials showed that macitentan and selexipag significantly reduced the risk of morbidity and mortality in PAH patients, with hazard ratios (HRs) of 0.70 (p=0.01) and 0.60 (p<0.001), respectively, vs placebo[10,11].

However, overall mortality remains high, with a mean OS of 5–7 years with conventional treatments. No significant improvements in survival were observed during the past decade, and patients often experienced severe disability in the final months of life. This underscores the need for additional treatment options that target new pathways involved in pulmonary vascular remodelling[2,5].

Is Sotatercept a Disease-modifying Treatment?

Sotatercept, a first-in-class activin signalling inhibitor, received US Food and Drug Administration approval in March 2024. It works by restoring balance between the antiproliferative and proproliferative signalling pathways. Unlike current vasodilatory therapies, sotatercept holds promise as a disease-

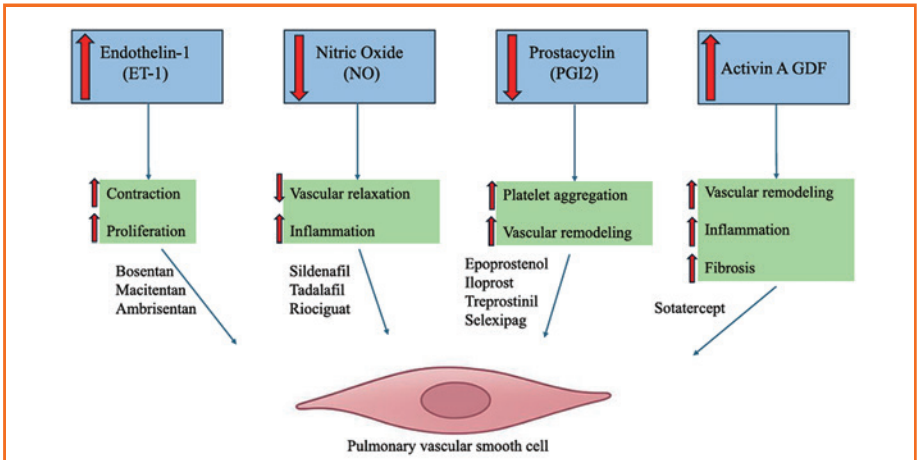

Fig. 1: Conventional pulmonary arterial hypertension treatment pathways and the new activin signalling pathway. Source: Mouratoglou SA, et al. Int J Cardiol Congenit Heart Dis 2025[9], reused under the terms of the CC BY-NC 4.0 license (https://creativecommons.org/licenses/by-nc/4.0/).

Table 2: STELLAR: Summary of secondary endpoints (ranked in hierarchical order) (Adapted from Hoeper et al., presented at ACC/WCC 2023[14])

	Secondary endpoint	Placebo (N=160)	Sotatercept (N=163)	Sotatercept vs placebo	
				HL location shift (95% CI)	p-value*
1	Multicomponent improvement,† n/N (%)	16 (10.1)‡	63 (38.9)‡	–	< 0.001
2	PVR – dyn·sec·cm^{-5}			-234.6 (-288.4 to -180.8)	< 0.001
3	NT-proBNP – pg/mL			-441.6 (-573.5 to -309.6)	< 0.001
4	WHO FC, n/N (%)	22 (13.8)‡	48 (29.4)‡	–	< 0.001
5	TTCW or all-cause death			0.16 (0.08 to 0.35)§	< 0.001§
6	French low-risk score, n/N (%)	29 (18.2)‡	64 (39.5)‡	–	< 0.001
7	PAH-SYMPACT Physical Impacts			-0.26 (-0.49 to -0.04)	0.010
8	PAH-SYMPACT Cardiopulmonary			-0.13 (-0.26 to -0.01)	0.028
9	PAH-SYMPACT Cognitive/Emotional			-0.16 (-0.40 to 0.08)	0.156

**Based on aligned rank stratified Wilcoxon test for continuous parameters or Cochran-Mantel-Haenszel method for dichotomous variables, both stratified by randomisation factors. †Defined as meeting all three of the following criteria at week 24: improvement in 6MWD [increase of ≥ 30 meters]; improvement in NT-proBNP level [decrease of ≥30%] or maintenance/achievement of NT-proBNP level < 300 pg/mL; and improvement in WHO FC [shift from class III to II or I, or class II to I] or maintenance of class II. ‡For multicomponent improvement and French risk score, N=159 (placebo) and N=162 (sotatercept) due to one patient in each treatment group with missing data due to COVID-19 and excluded from analysis. For WHO FC, N= 159 (placebo) and N= 163 (sotatercept) due to a placebo-treated patient with missing data due to COVID-19 and excluded from analysis. §Expressed as hazard ratio (95 % CI) and log-rank test p-value. 6MWD = 6-minute walk distance; CI = confidence interval; FC = functional class; HL = Hodges-Lehmann; NT-proBNP = N-terminal pro–B-type natriuretic peptide; PAH SYMPACT = pulmonary arterial hypertension symptoms and impact patient-reported questionnaire; PVR = pulmonary vascular resistance; TTCW = time to clinical worsening; WHO = World Health Organization; WHO FCII = slight limitation of activity (ordinary activities cause some symptoms); WHO FCIII = marked limitation of activity (less than ordinary activity causes symptoms)*

modifying treatment because it directly targets the pathophysiological mechanism in PAH (Fig. 1)[2,9,12].

Our patient sequentially received sildenafil, macitentan, and selexipag, targeting all three treatment pathways. However, he was refractory to all conventional treatments (Fig. 1). In PAH, non-responders have worse survival than long-term responders[13]. At this point, sotatercept was the only remaining hope for the patient, aside from a lung transplant or heart–lung transplant.

STELLAR: Efficacy and Safety Findings

The decision to add sotatercept to triple therapy was based on the multicentre, double-blind, phase 3 STELLAR trial in 323 adult patients (median age, 47.9 years; female, 79.3 %) with PAH (WHO Group 1, FC II [48.6 %] or III [51.4 %]) who were receiving stable background therapy, including triple therapy (61.3 %), prostacyclin infusion therapy (39.9 %), double therapy (34.7%), and monotherapy (4.0 %). Patients were randomised 1:1 to receive subcutaneous sotatercept (starting dose, 0.3 mg/kg; target dose, 0.7 mg/kg) or placebo, administered thrice weekly[2].

STELLAR met its primary endpoint by demonstrating an improvement from baseline in 6-minute walk distance (6MWD) at week 24. Sotatercept significantly improved 6MWD by 40.8 m vs placebo (p<0.001), indicating enhanced exercise capacity[2]. Consistently, our patient's 6-minute walk distance improved from 40 to 80 m.

In addition, sotatercept demonstrated statistically significant improvements in eight out of nine prespecified secondary endpoints, including multicomponent improvement, PVR, NT-proBNP,

WHO FC, time to death or clinical worsening, French low-risk score, as well as changes in PAH–Symptoms and Impact (PAH-SYMPACT) physical impacts and cardiopulmonary symptom domains ($p<0.05$ for all). The PAH-SYMPACT cognitive/emotional impacts domain, which was assessed as the final secondary outcome measure, did not achieve statistical significance ($p=0.156$; Table 2)[2,14]. The subjective improvements in quality of life and objective improvements in WHO FC and NT-proBNP are similarly observed in our patient.

Of note, treatment with sotatercept resulted in an 84 % lower risk of a composite of death from any cause or non-fatal clinical worsening event (5.5 % vs 26.2 %; HR, 0.16; 95 % CI, 0.08 – 0.35; $p < 0.001$) vs placebo (Table 2)[2].

Sotatercept was associated with lower rates of AEs (84.7 % vs 87.5 %) and serious AEs (14.1 % vs 22.5 %) compared with placebo. AEs that occurred more frequently with sotatercept than with placebo included epistaxis (12.3 % vs 1.9 %), dizziness (10.4 % vs 1.9 %), telangiectasia (10.4 % vs 3.1 %), thrombocytopenia (6.1 % vs 2.5 %), increased haemoglobin levels (5.5 % vs 0 %), and increased blood pressure (3.7 % vs 0.6 %)[2].

Close Monitoring for Early Intervention

Although at a later PAH stage (WHO FC III), our patient, who was refractory to all conventional medications, still responded to sotatercept. However, early intervention is important, as early use of sotatercept may help prevent or delay the onset of irreversible symptoms and clinical worsening events (e.g., deterioration requiring lung or heart–lung transplantation) by targeting the underlying mechanisms of PAH.

Severe SARS-CoV-2 infection may cause pulmonary vascular injury, potentially aggravating PAH. Given the persistence of COVID-19, clinicians should closely monitor PAH patients, as they may be at increased risk of complications and poorer outcomes following COVID-19[15,16].

CONCLUSION

Due to its rapidly progressive nature, the treatment strategy for PAH has shifted from targeting individual pathways to earlier and more aggressive use of combination therapy. Considering its potential disease-modifying properties, tolerability, and improvements in 6MWD, haemodynamic parameters and quality of life, as well as the 84 % reduction in risks of all-cause mortality and non-fatal clinical worsening of PAH, the addition of sotatercept to background therapy should be considered in patients at earlier stages (e.g., WHO FC II) to optimise outcomes.

Acknowledgement
Medical writing and editorial assistance were provided by MSD. The authors directed the content and development of the manuscript, had full control over all aspects, and approved the final draft for submission.

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HKSH AIR
(Allergy-Immunology-Rheumatology)
SYMPOSIUM 2026

DATE: Sunday, 26 April 2026
VENUE: 9/F, HKSH Eastern Building, 3 Tung Wong Road, Shau Kei Wan, Hong Kong

FORMAT: In-person
LANGUAGE: English

PROGRAMME

The symposium will convene leading experts in allergy, immunology, and rheumatology to share cutting-edge insights and clinical advancements.

09:30 – 09:45	REGISTRATION		
09:45 – 10:00	WELCOME		Dr. LIANG Hin Suen, Raymond
10:00 – 11:00	**Plenary 1- JAKi from Inflammation to Allergy**	Chairperson	Dr. YU Ka Lung, Carrel Dr. ZHANG Lijun
	JAKi in the Treatment of Rheumatic Diseases: What's New (Rheumatology Perspective)		Dr. LEE Ka Wing, Gavin
	JAKi in the Management of Skin Diseases (Dermatologist Perspective)		Dr. CHAN Chun Yin, Johnny
11:00 – 11:30	BREAK		
11:30 – 12:30	**Scientific Session 1**	Chairperson	Dr. WONG Pui Yan, Stella
	1: Allergy Testing Makes Simple – Which and How		Dr. AU Yuen Ling, Elaine
	2: The Pathogenesis and Therapeutic Implications of Eczema		Dr. CHUNG Man Ho, Martin
12:30 – 13:30	**Lunch Symposium (Novartis)**	Chairperson	Dr. CHEUNG Tsang, Tommy
	BTK Inhibitor – Novel Targets for Urticaria		Dr. LI Philip Hei
13:30 – 14:30	**Scientific Session 2**	Chairperson	Dr. YEUNG Wan Yin, Winnie
	1: Omalizumab Assisted Multiple OIT – Multidisciplinary Evidenced Practice		Dr. HO Hok Kung, Marco
	2: Anti-Cytokine Syndromes in Hong Kong: Autoimmunity Meet Immunodeficiency		Dr. Valerie CHIANG
14:30 – 15:00	BREAK		
15:00 – 15:30	**Plenary 2 – Interleukins & Others**	Chairperson	Dr. IONG Iok In Dr. LEE Ka Wing, Gavin
	Benralizumab (IL-5 Inhibitor) in Asthma and Very Early EGPA		Dr. YEUNG Wan Yin, Winnie
15:30 – 16:00	**Sponsored Symposium (Sanofi)**	Chairperson	Dr. CHAN Pui Shan, Julia
	Multidisciplinary Approach to Type II Diseases		Dr. LI Philip Hei
16:00	CLOSING		

** Content is subject to change without prior notice*

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香港大學內科學系

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The Hong Kong Society of Rheumatology 香港風濕病學會

Lifestyle of Cardiologists

Dr Victor WT YAN
MBBS (HK), MRACP, FRACP
Cardiologist

Dr Victor WT YAN

CARDIOLOGIST IN PRIVATE PRACTICE

Cardiologists usually have a very tight schedule, requiring prompt attention to the problem, quick decision-making, and timely appropriate treatment. Very often, several problems turn up at the same time. New technologies and new drugs are constantly emerging. Cardiologists need to stay up to date to keep pace with rapid developments. Our academic colleagues face an additional burden in teaching, training, and research. Administrative work and funding responsibilities are added as they move up the ladder. This stressful life is somehow adapted by most cardiologists to strike the balance between work and life. They all have different lifestyles according to their individual interests, abilities, and commitments. Lifestyle is the taste of life. It is highly personal.

Without mentioning names, I would like to share with you how the cardiologists I know manage to achieve this.

Some are religious. At the time of crisis, they can stay calm and controlled, as they have faith in God. Their commitment to the church and to their daily work creates a well balanced lifestyle , helping people spiritually and medically, attaining a great sense of fulfilment.

Voluntary works such as St John Ambulance Bridgate and Auxiliary Medical Service are commonly joined by cardiologists. They provide their professional supervision. This helps diversify their responsibilities across different sectors of medical service. Some prefer to provide free medical care to the underprivileged people, particularly in remote areas of the world. On return from each trip, they gain new emergy to continue the journey of their regular job.

After half a century of private cardiology practice, I appreciate the value of philosophy more and more. Many of my colleagues see the same. Philosophy seems empty, but the function of a cup is the space in it, rather than the material that makes the cup. Philosophy is useful in counselling patients and sometimes even in counselling ourselves. Taoism is what I favour. It helps us understand the less interference is better.

Cardiologists are good readers. Apart from medical journals, they read novels and Chinese literature. The advantage of reading is that one can do it alone, anytime and anywhere. It is more than a lifelong self-entertainment. Sharing with people with common interests often generates unlimited interactive resonance. I started reading Chinese Literature in my forties and continue doing it in my spare time. The old teaching may not be directly applicable to present-day life. But the wisdom and inspiration have helped shape my career, and the offers it brings have brought a sense of taste to my life over the years. We all agree that medicine is science, but the practice of medicine is an art.

Some like travelling. Taking holidays is the best way to replenish the energy for a forward drive. To see more of the world broadens one's mind. Travelling with the family is even more rewarding, especially when the children are still young. When they are older, they may prefer to travel by themself and with friends. After all, a balance between work and family is what most cardiologists work hard to maintain.

Many cardiologists are collectors. They collect old furniture, paintings, ceramics, watches, jade, and all sorts of antiques. They have to do lots of homework. Some of them paid high school fees for buying the wrong thing. Anyway, all these are mainly for their own interest and satisfaction. The collected items will usually appreciate over the course of time.

All cardiologists are aware of the importance of exercise. Regular walking, running, hiking, swimming and Taichi are the common choices. Some are more sportive. Many of them play tennis and golf. Sailing and skiing are less common. ally, I prefer golf, which I started in my fifties and am still enjoying now in my eighties. It is a social game providing mild exercise and a sense of achievement.

Many cardiologists are music lovers. Apart from going to concerts regularly, some play different types of instruments. Many of their children also play, forming small music groups.

Photography and painting have something in common. They both are compositions of colour and light. We all look for colour and light in our lives. There are many good photographers among ourselves. It does not need expensive equipment and complicated cameras; a smart cameras or a smartphone will do. It is handy and convenient. Good shots can be captured at anytime. Painting is different. It takes much longer to complete one picture ,but with a brush in hand, one can modify the composition into a piece of art. I

picked up oil painting when I was 75, and it remains a challenge for me.

The next group is wine and dine. They love fine wine and good food. Some even have their own collections of wine and whiskey. When they go out with friends and doctors, they contribute to the drinks while others pay for the food. Very often, the drink is more expensive than the food.

To complete my list, I have to include those who love pets and those who chase after AI and technology. I hope that I have not missed out anybody. Among the list, you may find yourself or some of your colleagues. Remember, lifestyle may change as one ages due to multiple factors such as health and physical capacity. Whatever happens we all aim for the equilibrium of a triangle : work, family, and livelihood.

Federation Sports Day 2026

Dr Alson Wai-ming CHAN

FHKAM(Paed), FHKCPaed, FRCPCH, DCH (Ireland), Dip Ger Med RCPS (Glasg), PDipCmmunityGeriatrics(HK), MBChB

Specialist in Paediatric Immunology, Allergy and Infectious Diseases
Hon Secretary of FMSHK

Dr Alson Wai-ming CHAN

The Federation President's Cup Soccer 5, Basketball and Table Tennis Tournament for 2026 was held at Ying Wah College on 15th March 2026. There were six soccer, four basketball and four table tennis teams that participated in the tournament this year, including Hong Kong Academy of Medicine, the Hong Kong Medical Association, Hong Kong Dental Association, Hong Kong Doctors Union, AstraZeneca Hong Kong Ltd, Jacobson Pharma Corporation Ltd, Pfizer Corporation Hong Kong Limited, Roche Hong Kong Limited and Viatris Healthcare Hong Kong Limited.

This year, we were honoured to have again the participation of the Sun Hei All Stars Football team (晨曦明星足球隊) on the final day. Our Federation United Team, comprising members from various tournament teams, played a friendly exhibition match with the Sun Hei All-Star Football team.

We congratulate the winning teams and express our sincere gratitude for the support from all the participating teams and guests. We look forward to seeing you again at the Federation President Cup Soccer Five & Table Tennis Tournament in 2027!

The final results were as follows:

Table Tennis Tournament	
Champion :	Hong Kong Dental Association
1st Runner Up :	The Hong Kong Medical Association
2nd Runner Up :	Hong Kong Doctors Union

Basketball Tournament	
Champion :	Hong Kong Academy of Medicine
1st Runner Up :	Jacobson Pharma Corporation Ltd
2nd Runner Up :	Viatris Healthcare Hong Kong Limited

Soccer Five Tournament	
Champion :	The Federation of Medical Societies of Hong Kong
1st Runner Up :	The Hong Kong Medical Association
2nd Runner Up :	Roche Hong Kong Limited


FEDERATION SPORTS DAY
Pfizer
Roche


香港牙醫學會
Hong Kong Dental Association
FEDERATION SPORTS DAY2026

Medical Diary of April

Sunday	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
			★ Certificate Course on Urological Disorders 2026 (Video Lectures) **1**	**2**	**3**	**4**
5	**6**	**7**	★ The Hong Kong Neurosurgical Society Monthly Academic Meeting – To be confirmed ★ Certificate Course on Urological Disorders 2026 (Video Lectures) **8**	★ HKMA CME Portal (Online) The HKMA CME Online Lecture Topic: Holistic and Practical Skincare Guidance for Sensitive and Atopic Dermatitis Skin ★ Certificate Course on Clinical Ophthalmology 2026 - Contemporary Practice and Emerging Trends (Video Lectures) **9**	**10**	**11**
12	★ HKMA CME Portal (Online) HKMA-KWC CME Online Lecture Series on Common Medical Conditions for GP in 2026 Topic: Cognitive Impairment, Frequent Fall **13**	★ In-person / HKMA CME Portal (Online) HKMA-HKSH CME Programme 2026 Topic: Update Management for Patient with Proteinuria **14**	★ HKMA CME Portal (Online) The HKMA CME Online Lecture Topic: Updates on Long-term Antiplatelet Strategies for Chronic Coronary Syndromes ★ Certificate Course on Urological Disorders 2026 (Video Lectures) **15**	★ HKMA CME Portal (Online) The HKMA CME Online Lecture Topic: Beyond Mood: The Gut–Brain–Body Axis and Its Role in Stress Resilience and Whole Person Mental Health ★ Certificate Course on Clinical Ophthalmology 2026 - Contemporary Practice and Emerging Trends (Video Lectures) **16**	★ In-person The HKMA CME Lecture in Physical Attendance Mode Topic: Strengthening the Management of Mixed Dyslipidemia **17**	**18**
19	**20**	**21**	★ In-person The HKMA CME Lecture in Physical Attendance Mode Topic: Impact of Influenza and the Need for Better Protection against Influenza ★ Certificate Course on Urological Disorders 2026 (Video Lectures) **22**	★ In-person The HKMA CME Lecture in Physical Attendance Mode Topic: Advances in Osteoporosis Management: Safety, Real-World Evidence, and the Efficacy of Anabolic Therapies ★ Certificate Course on Clinical Ophthalmology 2026 - Contemporary Practice and Emerging Trends (Video Lectures) ★ FMSHK Foundation Meeting ★ FMSHK Executive Committee Meeting **23**	**24**	**25**
26	★ In-person / HKMA CME Portal (Online) HKMA-HKWC CME Hybrid Lecture Topic: Dementia in Primary Care: From Screening to Shared Care in Hong Kong's Aging Society **27**	**28**	**29**	★ Certificate Course on Clinical Ophthalmology 2026 - Contemporary Practice and Emerging Trends (Video Lectures) **30**		

Date / Time	Function	Enquiry / Remarks
1 WED 7:00 PM	**Certificate Course on Urological Disorders 2026 (Video Lectures)** Organisers: The Federation of Medical Societies of Hong Kong, Hong Kong Society of Practising Urologists & The Hong Kong Society of Endourology Speakers: Dr Clarence LEUNG & Dr James TSU	Ms ToTo CHAN Tel: 2821 3512
8 WED 7:00 AM	**The Hong Kong Neurosurgical Society Monthly Academic Meeting – To be confirmed** Organiser: Hong Kong Neurosurgical Society Speaker(s): Dr Andy Lok-heng CHENG Chairman: Dr Su-to WONG Venue: Seminar Room, G/F, Block A, Queen Elizabeth Hospital; or via Zoom meeting	CME Accreditation College: 1.5 points College of Surgeons of Hong Kong Dr Jason CHOW Tel: 2595 6456 Fax. No.: 2965 4061
7:00 PM	**Certificate Course on Urological Disorders 2026 (Video Lectures)** Organisers: The Federation of Medical Societies of Hong Kong, Hong Kong Society of Practising Urologists & The Hong Kong Society of Endourology Speakers: Dr Thomas WONG & Dr Wilson LAM	Ms ToTo CHAN Tel: 2821 3512
9 THU 2:00 PM	**HKMA CME Portal (Online)** **The HKMA CME Online Lecture** **Topic: Holistic and Practical Skincare Guidance for Sensitive and Atopic Dermatitis Skin** Organiser: The Hong Kong Medical Association Speaker: Dr Dominic Yik-kiu LAI	HKMA CME Dept. Tel: 2527 8452 1 CME Point
7:00 PM	**Certificate Course on Clinical Ophthalmology 2026 - Contemporary Practice and Emerging Trends (Video Lectures)** Organisers: The Federation of Medical Societies of Hong Kong & The Hong Kong Ophthalmological Society Speaker: Dr Tommy CHAN	Ms ToTo CHAN Tel: 2821 3512
13 MON 2:00 PM	**HKMA CME Portal (Online)** **HKMA-KWC CME Online Lecture Series on Common Medical Conditions for GP in 2026** **Topic: Cognitive Impairment, Frequent Fall** Organisers: The Hong Kong Medical Association and Kowloon West Cluster of HA Speaker: Dr Sze-chiu HUNG	HKMA CME Dept. Tel: 2527 8452 1 CME Point
14 TUE 2:00 PM	**In-person / HKMA CME Portal (Online)** **HKMA-HKSH CME Programme 2026** **Topic: Update Management for Patient with Proteinuria** Organisers: The Hong Kong Medical Association and Hong Kong Sanatorium & Hospital Speaker: Dr Man-fai LAM Venue: The HKMA Wanchai Premises, 5/F, Duke of Windsor Social Service Building, 15 Hennessy Road, Wanchai, Hong Kong	HKMA CME Dept. Tel: 2527 8452 1 CME Point
15 WED 2:00 PM	**HKMA CME Portal (Online)** **The HKMA CME Online Lecture** **Topic: Updates on Long-term Antiplatelet Strategies for Chronic Coronary Syndromes** Organiser: The Hong Kong Medical Association Speaker: Dr Shek-yin AU	HKMA CME Dept. Tel: 2527 8452 1 CME Point
7:00 PM	**Certificate Course on Urological Disorders 2026 (Video Lectures)** Organisers: The Federation of Medical Societies of Hong Kong, Hong Kong Society of Practising Urologists & The Hong Kong Society of Endourology Speakers: Dr Wayne LAM & Dr Brian HO	Ms ToTo CHAN Tel: 2821 3512
16 THU 2:00 PM	**HKMA CME Portal (Online)** **The HKMA CME Online Lecture** **Topic: Beyond Mood: The Gut–Brain–Body Axis and Its Role in Stress Resilience and Whole Person Mental Health** Organiser: The Hong Kong Medical Association Speaker: Dr Lisa Shan CHENG	HKMA CME Dept. Tel: 2527 8452 1 CME Point
7:00 PM	**Certificate Course on Clinical Ophthalmology 2026 - Contemporary Practice and Emerging Trends (Video Lectures)** Organisers: The Federation of Medical Societies of Hong Kong & The Hong Kong Ophthalmological Society Speaker: Dr Julia CHAN	Ms ToTo CHAN Tel: 2821 3512
17 FRI 2:00 PM	**In-person** **The HKMA CME Lecture in Physical Attendance Mode** **Topic: Strengthening the Management of Mixed Dyslipidemia** Organiser: The Hong Kong Medical Association Speaker: Dr Chak-kwan LAU Venue: Crystal Ballroom A&B, 2/F, The Cityview, 23 Waterloo Road, Kowloon	HKMA CME Dept. Tel: 2527 8452 1 CME Point
22 WED 2:00 PM	**In-person** **The HKMA CME Lecture in Physical Attendance Mode** **Topic: Impact of Influenza and the Need for Better Protection against Influenza** Organiser: The Hong Kong Medical Association Speaker: Dr Julie Kwan-ling WANG Venue: Crystal Ballroom A&B, 2/F, The Cityview, 23 Waterloo Road, Kowloon	HKMA CME Dept. Tel: 2527 8452 1 CME Point
7:00 PM	**Certificate Course on Urological Disorders 2026 (Video Lectures)** Organisers: The Federation of Medical Societies of Hong Kong, Hong Kong Society of Practising Urologists & The Hong Kong Society of Endourology Speakers: Dr Chi-man NG & Dr Wilson CHAN	Ms ToTo CHAN Tel: 2821 3512
23 THU 2:00 PM	**In-person** **The HKMA CME Lecture in Physical Attendance Mode** **Topic: Advances in Osteoporosis Management: Safety, Real-World Evidence, and the Efficacy of Anabolic Therapies** Organiser: The Hong Kong Medical Association Speaker: Dr Julian Lai-lok CHAN Venue: Crystal Ballroom A&B, 2/F, The Cityview, 23 Waterloo Road, Kowloon	HKMA CME Dept. Tel: 2527 8452 1 CME Point

Date / Time	Function	Enquiry / Remarks
23 THU 7:00 PM	**Certificate Course on Clinical Ophthalmology 2026 - Contemporary Practice and Emerging Trends (Video Lectures)** Organisers: The Federation of Medical Societies of Hong Kong & The Hong Kong Ophthalmological Society Speaker: Dr Nancy YUEN	Ms ToTo CHAN Tel: 2821 3512
7:00 PM	**FMSHK Foundation 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
8:00 PM	**FMSHK Executive Committee 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
27 MON 2:00 PM	**In-person / HKMA CME Portal (Online)** **HKMA-HKWC CME Hybrid Lecture** **Topic: Dementia in Primary Care: From Screening to Shared Care in Hong Kong's Aging Society** Organisers: The Hong Kong Medical Association and Hong Kong West Cluster Speaker: Dr Ka-keung YAM Venue: Central & Western District Health Centre, 7/F & 8/F, 308 Central Des Voeux, No. 308 Des Voeux Road Central, Sheung Wan, Hong Kong	HKMA CME Dept. Tel: 2527 8452 1 CME Point
30 THU 7:00 PM	**Certificate Course on Clinical Ophthalmology 2026 - Contemporary Practice and Emerging Trends (Video Lectures)** Organisers: The Federation of Medical Societies of Hong Kong & The Hong Kong Ophthalmological Society Speaker: Dr Poemen CHAN	Ms ToTo CHAN Tel: 2821 3512

Certificate Course for Doctors, Nurses, Dietitians (and Students), and Allied Health Professionals • Course No. C435 • CME/CNE Course

Certificate Course on

Clinical Nutrition and Disease Management 2026

(Video Lectures)

Jointly organised by

The Federation of Medical Societies of Hong Kong

Hong Kong Dietitians Association

Date	Topics	Speakers
21 May 2026	Bone–Muscle Crosstalk – The Critical Role of Nutrition in Healthy Aging and Osteoporosis	Ms. Sally Shi Po POON Registered Dietitian (UK) Accredited Practising Dietitian (Australia)
28 May 2026	Nourishing Recovery – The Vital Role of Dietitians in Eating Disorders	Ms. Sylvia See Way LAM Honorary Consultant Hong Kong Dietitians Association Accredited Dietitian (HKAAD) Accredited Practising Dietitian (Australia)
4 June 2026	Medical Nutrition Therapy for Gut Disorders – From Irritable Bowel Syndrome (IBS) to Complex Crohn's Disease	Ms. Denise Wai Wah LUK Registered Dietitian (CDO) MSc in Endocrinology, Diabetes and Metabolism
11 June 2026	Dietary Strategies to Manage Liver Diseases	Ms. Sylvia See Way LAM Honorary Consultant Hong Kong Dietitians Association Accredited Dietitian (HKAAD) Accredited Practising Dietitian (Australia)
18 June 2026	Nutritional Strategies for the Elderly with Stroke Complications	Ms. Heidi Tze Man CHAN Registered Dietitian (USA) Board Certified Specialist in Gerontological Nutrition (USA)
25 June 2026	Evidence Based Approach to Weight Management – Lifestyle, GLP-1 Therapy and Metabolic Surgery	Ms. Ingrid Yuen Man KAN Accredited Practising Dietitian (Australia)

Date : 21, 28 May and 4, 11, 18, 25 June 2026 (THUR)
Duration of Session : 1.5 hours (6 sessions)
Time : 7:00 pm – 8:30 pm *(or within 4 days from each Lecture Date)*
Course Feature : Video lectures (with Q&A platform for participants to post the questions)
Language Media : Cantonese (Supplemented with English)
Quiz for CME : DOCTORS are required to complete a quiz after the completion of each lecture
Course Fee : HK$1,200
FMSHK Certificate : Awarded to participants with a minimum attendance of 70% (per Lecture and 4 out of 6 Lectures)
Deadline : **14 May 2025**
Enquiry : The Secretariat of The Federation of Medical Societies of Hong Kong
Tel.: 2821 3512 Fax : 2865 0345 Email : toto.chan@fmshk.org

CME / CNE / CPD_PT / CPD_OT (100% Attendance) Accreditation in application
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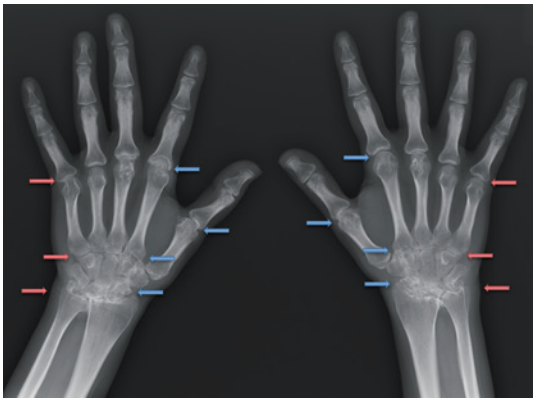
Answers to Radiology Quiz

Answers:



1. Bilateral symmetrical erosions and joint space narrowing at metacarpophalangeal, intercarpal and distal radiocarpal ulnar joints (blue arrows). Sparing of the distal interphalangeal joints. Periarticular osteopenia and mild soft tissue swelling (red arrows).

2. Rheumatoid arthritis.

3. Referral to rheumatologists for work-up of autoimmune markers, including rheumatoid factor, anti-CCP/ ACPA and CRP/ ESR markers.

Dr Janet Hin-man WONG
MBBS, FRCR

Certificate Course for General Practitioners, Family Physicians, Nurses and Allied Health Professionals

● Course No. C434 ● CME/CNE Course

Certificate Course on

Cardiovascular Medicine 2026

(Video Lectures)

Jointly organised by

The Federation of Medical Societies of Hong Kong

Hong Kong College of Cardiology

Objectives:

Designed for General Practitioners, Family Physicians, Nurses, and Allied Health Professionals, this course offers a comprehensive update in Cardiology. Through twelve focused lectures, participants will explore the management of common and important Cardiac Diseases, guided by current evidence and best practices.

Date	Topics	Speakers
5 May 2026	CT Coronary Angiography and Cardiac Stress Imaging in Stable Coronary Artery Disease	Dr. Ching Shing Associate Consultant United Christian Hospital
	Mitigating Cardiovascular Risk - A Clinical Guide to Dietary Modification	Dr. Ko Kwok Chun, Jason Specialist in Cardiology
12 May 2026	Diagnosis of Heart Failure and Management	Dr. Cheng Yue Hong Specialist in Cardiology
	What is Cardiovascular-Kidney-Metabolic Syndrome?	Dr. Lo Ka Yip, David Specialist in Cardiology
19 May 2026	Exercise Stress Testing - Principles, Protocols and Interpretation	Dr. Lam Hing Yin, Wilson Associate Consultant Yan Chai Hospital
	How Best to Evaluate and Manage Perioperative Cardiovascular Risk in Patients Undergoing Noncardiac Surgery	Dr. Leung Wang Hei, Ricky Specialist in Cardiology Queen Mary Hospital
26 May 2026	The Pan-Vascular Management Center - Integrated Pathophysiology-based Management of Vascular Disease	Dr. Cheong Yan Yue, Adrian Piers Specialist in cardiology
	Comprehensive Atrial Fibrillation Care Across the Continuum	Dr. Tam Tsz Kin Assistant Professor The Chinese University of Hong Kong
2 June 2026	Navigating the New Hypertension Guidelines and New Definition of Elevated Blood Pressure	Dr. Lai Tsun Kwong Associate Consultant Pamela Youde Nethersole Eastern Hospital
	Making the most of SGLT2i in Primary Care	Dr. Yau Kwok Ho Consultant Pamela Youde Nethersole Eastern Hospital
9 June 2026	Hyperlipidaemia Management - New Horizons and Evolving Strategies	Dr. Chim Ming Yam, Thomas Specialist in Cardiology Queen Elizabeth Hospital
	Coronary Artery Disease Management - Integrating Updates into Practice	Dr. Yiu Yuen Fung Specialist in Cardiology

Dates : 5, 12, 19, 26 May and 2, 9 June 2026 (TUE)
Duration of Session : 1.5 hours (6 sessions)
Time : 7:00 pm – 8:30 pm *(or within 4 days from each Lecture Date)*
Course Feature : Video lectures (with Q&A platform for participants to post the questions)
Language Media : Cantonese (Supplemented with English)
Quiz for Doctors : DOCTORS are required to complete a quiz after the completion of each lecture
Course Fee : HK$1,200
FMSHK Certificate : Awarded to participants with a minimum attendance of 70% (per lecture and 4 out of 6 sessions)
Deadline : **28 April 2026**
Enquiry : The Secretariat of The Federation of Medical Societies of Hong Kong
Tel.: 2821 3512 Fax : 2865 0345 Email : toto.chan@fmshk.org

CME / CNE / CDE / CPD_PT / CPD_OT (100% Attendance) Accreditation in application
Online Application from website: http://www.fmshk.org

ELIQUIS™

THE SAFER CHOICE[1,2^]

#1 OAC GLOBALLY[3,4#*]

Choose both efficacy and safety with ELIQUIS™

- The only NOAC to offer both **superior risk reduction in stroke/SE and major bleeding** over warfarin in NVAF[1,2^]
  - Real-world data demonstrated **a lower risk for GIB** with ELIQUIS™ vs other NOACs[†], with results **consistent in patients ≥80 years old and those with CKD**[5‡]
- **Continued efficacy, with favorable bleeding profile** regardless of bleeding endpoint, for the treatment of DVT/PE[6§]
- The **lowest renal clearance** among NOACs[7†]

^There are no head-to-head trials comparing NOACs

#Accounting for more patient treatment days prescribed around the world than any other OAC within NVAF & VTE indications***¶

*Geographies: (1) IQVIA MIDAS Sales Data: Algeria; Argentina; Austria; Australia; Belarus; Belgium; Bosnia; Brazil; Bulgaria; Canada; Central America; Chile; China; Colombia; Croatia; Czech Republic; Denmark; Ecuador; Egypt; Finland; France; French West Africa; Germany; Greece; Hong Kong; Hungary; India; Indonesia; Ireland; Italy; Japan; Korea; Luxembourg; Malaysia; Mexico; Morocco; Netherlands; New Zealand; Norway; Pakistan; Peru; Philippines; Poland; Portugal; Puerto Rico; Romania; Russia; Serbia; Singapore; Slovakia; Slovenia; South Africa; Spain; Sweden; Switzerland; Taiwan; Thailand; Tunisia; Turkey; UAE; UK; US. (2) IQVIA MIDAS Medical Detailed Data: - Argentina, Brazil, Canada, France, Germany, Japan, Mexico, Spain, UK & US. (3) IQVIA MIDAS Medical Summary Data: - Argentina, Australia, Austria, Belgium, Brazil, Canada, Colombia, Czech, Egypt, France, Germany, Greece, Indonesia, Italy, Japan, Mexico, Morocco, Netherlands, New Zealand, Pakistan, Peru, Philippines, Poland, Portugal, Spain, Switzerland, Thailand, Turkey, UK & US; for NOAC (Apixaban, Betrixaban, Dabigatran Etexilate, Edoxaban, Rivaroxaban) and VKA (Acenocoumarol, Coumarin, Daphnetin, Dicoumarol, Fluindione, Phenindione, Phenprocoumon, Warfarin) molecules, reflecting estimates of real-world activity

***Indications accounted for by factoring standard unit volume based on medical source data and relevant WHO ICD10 codes

¶Methodology - Patient treatment days prescribed estimated based on the latest six-month period Q4'23 Sell-In/Sell-Out data. Standard Units divided by recommended administration of each NOAC within 24 hours (apixaban BID, dabigatran BID, edoxaban QD, rivaroxaban QD). VKA drugs treatment days estimated based on standard units divided by medical average daily dose

†Other NOACs include dabigatran, rivaroxaban and edoxaban[1,7]

‡In patients ≥80 years old: compared with dabigatran, rivaroxaban and edoxaban; In patients with CKD (defined as having a diagnosis of chronic kidney disease or a dialysis procedure): compared with dabigatran and rivaroxaban[5]

§ELIQUIS™ provided significant risk reduction across all types of bleeding vs enoxaparin/warfarin in patients treated for DVT/PE[6]

BID, twice daily; CKD, chronic kidney disease; DVT, deep vein thrombosis; GIB, gastrointestinal bleeding; ICD, International Statistical Classification of Diseases and Related Health Problems; NOAC, non-vitamin K antagonist oral anticoagulant; NVAF, nonvalvular atrial fibrillation; OAC, oral anticoagulant; PE, pulmonary embolism; QD, once daily; SE, systemic embolism; UAE, United Arab Emirates; UK, United Kingdom; US, United States; VKA, vitamin K antagonist; VTE, venous thromboembolism; WHO, World Health Organization.

References: 1. Granger CB, et al. *N Engl J Med* 2011;365:981-992. **2.** Ruff CT, et al. *Lancet* 2014;383:955-962. **3.** IQVIA MIDAS® Quarterly Volume Sales Data Q4'23 Sell-In/Sell-Out data. **4.** IQVIA MIDAS Medical Summary and IQVIA MIDAS Medical Detailed Data Q4'23. **5.** Lau WCY, et al. *Ann Intern Med* 2022;175:1515-1524. **6.** Agnelli G, et al. *N Engl J Med* 2013;369:799-808. **7.** Heine GH, et al. *Dtsch Arztebl Int* 2018;115:287-294.

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Scan the QR codes or type the URLs in your browser to find the full Prescribing Information of apixaban:

Apixaban (2.5 mg) https://www.pfi.sr/JzI

Apixaban (5 mg) https://www.pfi.sr/JzT

The QR codes/URLs link to the latest Prescribing Information approved by the Department of Health in Hong Kong and may not be effective and the same as presented in the actual product package.

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