Together We Keep Growing

EDM HK

Endocrinology, Diabetes & Metabolism Hong Kong

3rd Annual Meeting

17-18th Oct 2020

VIRTUAL MEETING
In the treatment of patients with type 2 diabetes and established CV disease receiving standard of care, CV death can strike at any time.

BATTLE CV DEATH NOW
MORE THAN EVER

JARDIANCE demonstrated 38% RRR in CV death

Established HbA1c efficacy

Demonstrated safety profile

Convenient, once-daily oral dosing

ADA & EASD recognize JARDIANCE as the SGLT2 inhibitor with the strongest evidence of CV benefits

Jardiance® (empagliflozin)

The ONLY OAD WITH CV INDICATION

Jardiance is indicated in T2DM patients and established cardiovascular disease to reduce the risk of cardiovascular death.
ACHIEVE TARGET LDL-C LEVELS WITH POWERFUL DUAL ACTION OF ATOZET®

Abbreviations: LDL-C: low-density lipoprotein cholesterol
References: 1. Hong Kong Product Circular (Atozet, MSD)

Selected Safety Information on ATOZET

Reduction and Prevention of Cardiovascular Events: ATOZET is indicated as an add-on therapy to diet for adults with primary hypercholesterolaemia and non-familial hypercholesterolaemia to reduce the risk of developing cardiovascular events. ATOZET is indicated as an add-on therapy to diet for use in adults with HCPH. ATOZET may also be used in combination with other lipid-lowering therapies, especially if an adequate response is not achieved with diet alone.

Contraindications: • Hypersensitivity to the active substances or any of the excipients. • Therapy with ATOZET is contraindicated during pregnancy and breastfeeding, and in women of child-bearing potential not using appropriate contraceptive measures. ATOZET is contraindicated in patients with active liver disease or unexplained persistent elevation in serum transaminases exceeding 2 times the upper limit of normal (ULN) values. • Myopathy/Rhabdomyolysis: • In post-marketing experience with ezetimibe, cases of myopathy and rhabdomyolysis have been reported. Most patients who developed rhabdomyolysis were taking a statin concurrently with ezetimibe. Rhabdomyolysis has been reported very rarely with ezetimibe monotherapy. • Atoz, ATOZET contain alcohol, which is a P450 3A4 substrate. Alcohol may in rare occasions affect the desired muscle and cause myalgia, intolerance, and impotence that may progress to rhabdomyolysis. • A CK level should be measured before starting treatment. If CK levels are significantly elevated (>5 times ULN) at baseline, treatment should not be started. • Patients must be asked to promptly report muscle pain, cramps, or weakness especially if accompanied by malaise or fever or if muscle pain and symptoms develop after discontinuing ATOZET. • Liver Function Tests should be performed before the initiation of treatment and periodically thereafter. • In some cases, increases in transaminases of greater than 3 times the ULN persist, reduction of dose or withdrawal of ATOZET is recommended. • Hepatic Insufficiency: Due to the unknown effects of the increased exposure to ezetimibe in patients with mild or moderate hepatic insufficiency, ATOZET is not recommended. • Incretin-like secretions: If it is suspected that a patient has developed incretin-like changes, statin therapy should be discontinued. • Diabetic mellitus: • Patients at risk (diabetes, obesity, hypertension) should be monitored both clinically and biochemically according to national guidelines. • Exacerbation: • ATOZET contains lactose. Patients with severe hereditary defects of galactose intolerance, the lactase deficiency, or glucose-galactose malabsorption should not take this medicine. • Inflammation: • Common adverse reactions (≥1/100, ≥1/10) include diarrhoea, myalgia. • In randomized clinical trials, the incidence of clinically important elevations in serum transaminases (AST and/or ALT) > 3 ULN, consecutive) was 0.6% for patients treated with ATOZET. These elevations were generally asymptomatic, not associated with cholestatics, and returned to baseline spontaneously or after discontinuation of therapy. Please consult the full prescribing information for detailed adverse events.

Before prescribing, please consult the full prescribing information.
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Welcome Message

It is a great pleasure for us to welcome you to the 3rd Annual Meeting of Endocrinology Diabetes & Metabolism Hong Kong (EDM HK). Since its inauguration meeting in 2018, EDM HK has become a unique and important local conference of the year for all healthcare professionals who manage patients with diabetes and endocrine disorders.

2020 has been a very challenging year for us all. To ensure the safety and well-being of our participants, we have made a tough decision to convert EDM HK 2020 from a face-to-face meeting to a fully virtual conference. Nonetheless, amidst the pandemic, EDM HK manages to create this exciting programme, bringing together a multidisciplinary group of local and overseas speakers to share with us the most updated clinical knowledge and research findings.

We look forward to meeting you on our virtual platform. Stay safe and healthy! We are sure we can go through this and meet you all again in person in 2021!

Dr. Paul CH Lee
Co-chairman
EDM HK 2020

Dr. Alan CH Lee
Co-chairman
EDM HK 2020

[Signature]

[Photo of Dr. Paul CH Lee and Dr. Alan CH Lee]
Public Lecture

The program is at Hong Kong Time (GMT+8)
(17 October 2020, Saturday)

<table>
<thead>
<tr>
<th>Time</th>
<th>Public Lecture (Broadcasted via YouTube)</th>
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<tbody>
<tr>
<td>10:00 – 10:30</td>
<td>Public Lecture 1: Hyperthyroidism in Different Faces/甲狀腺功能亢進 (甲亢) 多面睇&lt;br&gt;Chairperson: Dr. Jo Jo Kwan&lt;br&gt;Speakers: Dr. Alan Lee &amp; Ms. Connie Loong</td>
</tr>
<tr>
<td>10:30 – 11:00</td>
<td>Public Lecture 2: Iatrogenic Endocrine Disorders/藥物與內分泌失調的關係&lt;br&gt;Chairperson: Ms. KM Loo&lt;br&gt;Speakers: Dr. Doris Chan &amp; Ms. Veronica Hung</td>
</tr>
<tr>
<td>11:00 – 11:30</td>
<td>Public Lecture 3: Diabetes Medications / 糖尿病藥物&lt;br&gt;Chairperson: Ms. Shimen Au&lt;br&gt;Speakers: Dr. Raymond Hue &amp; Ms. Amy Yee</td>
</tr>
<tr>
<td>11:30 – 12:30</td>
<td>Public Lecture 4: Diabetes and Exercise Guidelines - Introduction to High Intensity Interval Training (HIIT) / 糖尿病與運動工作坊*（高強度間歇訓練）&lt;br&gt;Chairperson: Dr. Nicole Chau&lt;br&gt;Speaker: Mr. Derek Yeung</td>
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Scientific Program

(17 October 2020, Saturday)

<table>
<thead>
<tr>
<th>Time</th>
<th>Session</th>
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<tbody>
<tr>
<td>13:20 – 13:55</td>
<td>The Role of SGLT2 Inhibitors in Prevention and Treatment of Cardio-Renal Disease&lt;br&gt;Speaker: Prof. Itamar Raz (Israel)</td>
</tr>
<tr>
<td>13:55 – 14:00</td>
<td>Q &amp; A</td>
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<tr>
<td>14:00 – 14:10</td>
<td>Opening Ceremony</td>
</tr>
<tr>
<td>14:20 – 14:55</td>
<td>Envisioning the Breakthrough of SGLT2 Inhibitors: Treating Diabetes Saves the Heart&lt;br&gt;Speaker: Prof. Guntram Scherthaner (Austria)</td>
</tr>
<tr>
<td>14:55 – 15:00</td>
<td>Q &amp; A</td>
</tr>
<tr>
<td>15:00 – 15:25</td>
<td>Acromegaly: Any Update?&lt;br&gt;Speaker: Dr. PY Wu (Hong Kong)</td>
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<tr>
<td>15:25 – 15:50</td>
<td>Update on Primary Hyperparathyroidism&lt;br&gt;Speaker: Dr. Joanne Lam (Hong Kong)</td>
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<tr>
<td>15:50 – 16:00</td>
<td>Q &amp; A</td>
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<tr>
<td>16:00 – 16:20</td>
<td>Break Time for Virtual Booth Viewing</td>
</tr>
<tr>
<td>16:20 – 16:55</td>
<td>The Lower the Better? Evolving Concepts of Dyslipidemia, Diabetes and Cardiovascular Disease&lt;br&gt;Speaker: Dr. Michele Yuen (Hong Kong)</td>
</tr>
<tr>
<td>16:55 – 17:00</td>
<td>Q &amp; A</td>
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<tr>
<td>17:00 – 17:25</td>
<td>Paediatric Osteoporosis&lt;br&gt;Speaker: Dr. Joanna Tung (Hong Kong)</td>
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<tr>
<td>17:25 – 17:50</td>
<td>Big Children are NOT Small Adults&lt;br&gt;Speaker: Dr. Elaine Kwan (Hong Kong)</td>
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<td>17:50 – 17:55</td>
<td>Q &amp; A</td>
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<tr>
<td>17:55 – 18:00</td>
<td>Q &amp; A</td>
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<thead>
<tr>
<th>Time</th>
<th>Session A: Atypical Diabetes</th>
<th>Session B: Thyroid Disorder</th>
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</thead>
<tbody>
<tr>
<td>09:00 – 09:25</td>
<td>MODY: From Molecular Basis to Clinical Management (Speaker: Dr. Shirley Cheng)</td>
<td>Managing Thyroid Cancers – Updates and Future Advances (Speaker: Prof. Brian Lang)</td>
</tr>
<tr>
<td>09:25 – 09:50</td>
<td>Latent Autoimmune Diabetes (Speaker: Dr. Andrea Luk)</td>
<td>Radioactive Iodine: Thyrotoxicosis and Thyroid Cancer (Speaker: Dr. Wendy Chan)</td>
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<tr>
<td>09:50 – 10:00</td>
<td>Q &amp; A</td>
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**WEBINAR 7 (Sponsored by Eli Lilly)**

Chairperson: Dr. WS Chow

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<tr>
<td>10:10 – 10:45</td>
<td>Intensify Treatment in Type 2 Diabetes Mellitus with GLP-1 Receptor Agonists (Speaker: Dr. Andrew Ho)</td>
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<tr>
<td>10:45 – 10:50</td>
<td>Q &amp; A</td>
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<tr>
<td>10:50 – 11:00</td>
<td>Break Time for Virtual Booth Viewing</td>
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**WEBINAR 8A: Multidisciplinary Care in Endocrine Disorders**

Chairpersons: Prof. Terence Lao & Dr. MW Tsang

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<tr>
<td>11:00 – 11:25</td>
<td>Polycystic Ovary Syndrome (Speaker: Dr. Sofie Yung)</td>
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<tr>
<td>11:25 – 11:50</td>
<td>Dermatosis in Endocrinology (Speaker: Dr. Mandy Chan)</td>
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<tr>
<td>11:50 – 12:00</td>
<td>Q &amp; A</td>
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<tr>
<td>12:00 – 12:30</td>
<td>Break Time for Virtual Booth Viewing</td>
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**WEBINAR 9 (Sponsored by Sanofi)**

Chairperson: Prof. Ronald Ma

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<tr>
<td>12:40 – 13:15</td>
<td>Role of Fixed-Ratio Combination of Basal Insulin and GLP-1 RA in Type 2 Diabetes Management (Speaker: Dr. WS Chow)</td>
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<tr>
<td>13:15 – 13:20</td>
<td>Q &amp; A</td>
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**WEBINAR 10**

Chairperson: Prof. Kathryn Tan

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<tr>
<td>13:20 – 13:55</td>
<td>Graves' Orbitopathy (Speaker: Prof. George Kahaly)</td>
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<tr>
<td>13:55 – 14:00</td>
<td>Q &amp; A</td>
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**WEBINAR 11 (Sponsored by Novo Nordisk)**

Chairperson: Prof. Rosie Young

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<th>Time</th>
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<td>14:10 – 14:45</td>
<td>Realise the Potential of the New GLP-1 RA in DM Treatment (Speaker: Prof. Thomas Pieber)</td>
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<tr>
<td>14:45 – 14:50</td>
<td>Q &amp; A</td>
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<tr>
<td>14:50 – 15:00</td>
<td>Break Time for Virtual Booth Viewing</td>
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**WEBINAR 12 (Sponsored by Bayer)**

Chairperson: Dr. Peter Tong

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<th>Time</th>
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<tr>
<td>15:00 – 15:35</td>
<td>Addressing the Unmet Need in Diabetes: Role of Antithrombotic Therapy (Speaker: Dr. Godwin Leung)</td>
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<tr>
<td>15:35 – 15:40</td>
<td>Q &amp; A</td>
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**WEBINAR 13A: Endocrine Workshop**

Chairpersons: Dr. KW Chan & Dr. Anthony Shek

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<tr>
<td>15:40 – 16:05</td>
<td>Bone Health Assessment (Speaker: Dr. Eunice Leung)</td>
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<tr>
<td>16:05 – 16:30</td>
<td>Dynamic Tests in Endocrinology (Speaker: Dr. CH Choi)</td>
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<tr>
<td>16:30 – 16:40</td>
<td>Q &amp; A</td>
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**WEBINAR 13B: Inherited vs. Acquired Disorders**

Chairpersons: Prof. Sidney Tam & Dr. Annette Tso

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<tr>
<td>15:40 – 16:05</td>
<td>When are Your Patients Abnormal Lipid Levels Not Their Fault? (Speaker: Prof. Kathryn Tan)</td>
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<tr>
<td>16:05 – 16:30</td>
<td>Effects of Perfluorinated Compounds on Animal Metabolic Functions (Speaker: Prof. Chris Wong)</td>
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<tr>
<td>16:30 – 16:40</td>
<td>Q &amp; A</td>
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**WEBINAR 14: Endocrine & Cancer Case Discussions**

Panelists: Dr. Joanne Chiu, Dr. Ingrid Mak & Dr. Michele Yuen

Moderators: Dr. Johnny Chang & Dr. Chariene Woo
Conference Information & Accreditations

Organizing Committee

Co-chairmen
Dr. Paul Lee
Dr. Alan Lee

Members
Prof. Karen Lam
Dr. Johnny Chang
Prof. Kathryn Tan
Dr. Chariene Wool
Dr. WS Chow
Ms. Karen Wong
Dr. YC Woo
Ms. Amy Yee
Dr. TP Ip
Ms. SK Leung
Dr. David Lui
Ms. Connie Loong
Dr. Eunice Leung

Academic Accreditations

CME/ CNE points have been accredited by the following colleges and institution.

<table>
<thead>
<tr>
<th>College</th>
<th>Max for whole function</th>
<th>17 October 2020</th>
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<td>Hong Kong College of Community Medicine</td>
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<td>6</td>
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<tr>
<td>The Hong Kong College of Pathologists</td>
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<td>7</td>
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CNE Accreditation

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<th>17 - 18 October 2020</th>
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<tr>
<td>Hong Kong West Cluster</td>
<td>16</td>
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</table>

Participants are required to attend whole meeting on 17-18 October in order to receive the CNE points.
List of Overseas Speakers

Prof. George Kahaly
Professor
Department of Medicine I
Johannes Gutenberg University Medical Centre, Germany

Prof. Thomas Pieber
Chair Professor
Department of Internal Medicine
Medical University of Graz, Austria

Prof. Itamar Raz
Medical Director
Diabetes Medical Center in Tel Aviv, Israel

Prof. Guntram Schernthaner
Professor
Department of Medicine
University of Vienna, Austria
<table>
<thead>
<tr>
<th>Name</th>
<th>Title</th>
<th>Department/Division</th>
<th>Hospital/Institution</th>
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<tbody>
<tr>
<td>Ms. Shimen Au</td>
<td>Nurse Consultant</td>
<td>Department of Medicine</td>
<td>Pamela Youde Nethersole Eastern Hospital</td>
</tr>
<tr>
<td>Dr. CH Choi</td>
<td>Consultant</td>
<td>Department of Medicine</td>
<td>Queen Elizabeth Hospital</td>
</tr>
<tr>
<td>Dr. Joanne Lam</td>
<td>Honorary Clinical Assistant Professor</td>
<td>Department of Medicine</td>
<td>The University of Hong Kong</td>
</tr>
<tr>
<td>Dr. Doris Chan</td>
<td>Associate Consultant</td>
<td>Department of Medicine &amp; Geriatrics</td>
<td>Pok Oi Hospital</td>
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<td>Queen Mary Hospital</td>
</tr>
<tr>
<td>Prof. Karen Lam</td>
<td>Chair Professor</td>
<td>Department of Medicine</td>
<td>The University of Hong Kong</td>
</tr>
<tr>
<td>Dr. KW Chan</td>
<td>Senior Medical Officer</td>
<td>Department of Medicine and Geriatrics</td>
<td>Princess Margaret Hospital</td>
</tr>
<tr>
<td>Dr. Andrew Ho</td>
<td>Consultant</td>
<td>Department of Medicine &amp; Geriatrics</td>
<td>Tuen Mun Hospital</td>
</tr>
<tr>
<td>Prof. Brian Lang</td>
<td>Clinical Professor</td>
<td>Department of Surgery</td>
<td>The University of Hong Kong</td>
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<tr>
<td>Dr. Mandy Chan</td>
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<tr>
<td>Prof. Terence TH Lao</td>
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<td>Department of Obstetrics &amp; Gynaecology</td>
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<tr>
<td>Dr. Wendy Chan</td>
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<td>Dr. Emmy Lau</td>
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<td>Pamela Youde Nethersole Eastern Hospital</td>
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<tr>
<td>Dr. Johnny Chang</td>
<td>Resident</td>
<td>Department of Medicine</td>
<td>Queen Mary Hospital</td>
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<td>Dr. Victor Hung</td>
<td>Associate Consultant</td>
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<td>Dr. KP Lau</td>
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<td>North District Hospital</td>
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<tr>
<td>Dr. Nicole Chau</td>
<td>Associate Consultant</td>
<td>Department of Medicine and Geriatrics</td>
<td>Princess Margaret Hospital</td>
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<td>Department of Medicine</td>
<td>Queen Mary Hospital</td>
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<tr>
<td>Dr. Shirley Cheng</td>
<td>Medical and Health Officer</td>
<td>Clinical Genetic Service</td>
<td>Department of Health</td>
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<td>Dr. TP Ip</td>
<td>Consultant</td>
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<td>Tung Wah Hospital</td>
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<tr>
<td>Prof. Alice Kong</td>
<td>Professor</td>
<td>Department of Medicine &amp; Therapeutics</td>
<td>The Chinese University of Hong Kong</td>
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<tr>
<td>Dr. K K Lee</td>
<td>Honorary Clinical Associate Professor</td>
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<td>The University of Hong Kong</td>
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<tr>
<td>Dr. PT Cheung</td>
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<td>Department of Paediatrics &amp; Adolescent Medicine</td>
<td>Pamela Youde Nethersole Eastern Hospital</td>
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<td>Prof. Gilberto Leung</td>
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<td>The University of Hong Kong</td>
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<td>Dr. Joanne Chu</td>
<td>Clinical Assistant Professor</td>
<td>Department of Medicine</td>
<td>The University of Hong Kong</td>
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</table>
Dr. Eunice Leung  
Resident  
Department of Medicine  
Queen Mary Hospital

Dr. Godwin Leung  
Specialist in Cardiology  
Private Practice

Ms. KM Loo  
Nurse Consultant  
Department of Medicine & Therapeutics  
Prince of Wales Hospital

Ms. Connie Loong  
Advanced Practice Nurse  
Department of Medicine  
Queen Mary Hospital

Dr. David Lui  
Resident  
Department of Medicine  
Queen Mary Hospital

Dr. Andrea Luk  
Associate Professor  
Department of Medicine and Therapeutics  
The Chinese University of Hong Kong

Dr. John Ma  
Specialist in Endocrinology, Diabetes & Metabolism  
Private Practice

Prof. Ronald Ma  
Professor  
Department of Medicine and Therapeutics  
The Chinese University of Hong Kong

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Associate Consultant  
Department of Medicine  
Queen Elizabeth Hospital

Dr. Jason Ng  
Associate Consultant  
Department of Medicine  
Queen Elizabeth Hospital

Dr. Risa Ozaki  
Consultant  
Department of Medicine and Therapeutics  
Prince of Wales Hospital

Dr. Anthony Shek  
Consultant  
Department of Pathology  
Queen Elizabeth Hospital

Prof. Sidney Tam  
Honorary Clinical Professor  
Department of Pathology  
The University of Hong Kong

Prof. Kathryn Tan  
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The Role of SGLT2 inhibitors in Prevention and Treatment of Cardio-Renal Disease

Heart failure and renal insufficiency are two of the main reasons for morbidity and mortality in diabetic patients. Treatment with ACE inhibitors provides some benefit for the heart and kidney but the risk for HF, renal failure and cardiovascular death remains high in patients with and without HF and renal failure. Recently several large cardio renal outcome studies with SGLT2 inhibitors demonstrated a robust beneficial effect on prevention of heart and renal failure as well as CV death.

The DECLARE TIMI 58 was the largest study on SGLT in inhibitors trail with the longest follow-up which recruited a broad population of T2D patients that demonstrated for the first time that the effect of the drug on prevention of heart and kidney failure was similar even in patients without heart or kidney disease.

The beneficial effects on the heart were demonstrated in patients with HF and with both reduced and preserved ejection fractions as well as in patients without a history of heart disease or kidney disease. The beneficial effect on mortality however was only shown in T2D patients with reduced ejection fractions. The cardio renal effect of SGLT2s in T2D patients was only slightly related to its effect on glucose control, leading to new studies on the cardio renal effect of the drug in broader populations with or without diabetes with HF (DAPA-HF) or renal disease (DAPA-CKD).

The DAPA-HF study recruited 4,744 patients with HF and ejection fractions equal or lower than 40% most of them with pro BNP above 600 and randomized them to Dapagliflozin 10 mg or placebo. The primary end points were the combination of worsening of HF event or CV death or urgent HF visit requiring intravenous therapy. Dapagliflozin reduced the primary EP by 26%, HF by 30%, CV death by 18% and total mortality by 17%. The beneficial effects on the primary were similar in both T2D (redaction 25%) and non-diabetic (redaction 27%) subjects and in various subgroups - all highly significant.

DAPA-CKD recruited 4,304 patients with EGFR of 25-75% and UACR of 200-5000 mg/gr creatinine, mean EGFR was 43%and mean UACR was 965 mg/gr creatinine. The primary EP was sustained equal or more than 50% reduction in EGFR or end stage renal disease or renal or CV death. 67% of the patients had T2D - most of them with diabetic nephropathy. The primary end points were reduced by 39%, the renal specific end point by 44%, HF or CV death by 29% and all-cause mortality by 31% - all highly significant. The primary end points were equal between different subgroups.

These studies demonstrate that Dapagliflozin has similar beneficial effects in diabetic and non-diabetic patients with HF and Kidney disease. This calls for further studies to learn more about the effect of this family of drugs on non-diabetic patients with atherosclerotic heart disease, post MI atrial fibrillation and other high cardiovascular risk factors. The mechanism by which SGLT2s improve cardio-renal outcome is now ongoing in various studies which will hopefully open new windows to other clinical studies in this family of drugs.
Envisioning the Breakthrough of SGLT2 Inhibitors: Treating Diabetes Saves the Heart

My lecture will summarize highlights of SGLT2 inhibitor data presented at the ADA 2020.

Treatments that reduce risk of CV and HF outcomes expeditiously are important in the management of patients with T2D and atherosclerotic CV disease. In a post-hoc analysis, it was demonstrated that empagliflozin exerts clinically and statistically significant CV benefits within weeks of treatment initiation. The earliest effect appears to be on HHF at day 17. These findings demonstrate the rapidity of benefit of empagliflozin in reducing the risk of CV and HF outcomes.

In EMPA-REG OUTCOME® participants with T2D and CVD, empagliflozin markedly and durably delayed the need for insulin initiation and reduced the need for large dose increases in patients already using insulin. Empagliflozin may facilitate clinically meaningful reductions in insulin requirements among high-risk patients with T2D. Long-term implications of these effects on improvement in patient satisfaction, quality of life, and costs of care require further study.

A subgroup analysis from EMPRISE focusing on T2D patients >65 years showed that empagliflozin was associated with a decreased risk of modified MACE compared to DPP4i and a decreased risk of HHF compared with DPP4i or GLP1-RA in routine clinical care. These findings, addressing the comparative effectiveness of EMPA in routine care patients with mean age of 72 years, complement available information from RCTs, and reinforce the notion of the major role that heart failure may play in deciding when to prescribe empagliflozin.
Dr. PY Wu
Associate Consultant
Department of Medicine & Geriatrics
Tuen Mun Hospital

Acromegaly, Any Update?

Acromegaly is a disease characterized by the presence of excessive growth hormone. Most of the time the excessive growth hormone originated from the pituitary gland, which then stimulates the production of insulin-like growth factor-1 by the liver. It causes many of the different clinical manifestation of acromegaly, and the disease associated morbidity and mortality.

In this talk, we will discuss about the latest diagnostic approach, treatment aim and management plan for acromegaly. Newer treatment modalities will be updated.
Sweet and Brittle: Bone Fragility in Diabetes

Type 2 diabetes is associated with an increased risk of fragility fractures. Among the fragility fractures, hip and vertebral fractures can lead to significant morbidity and mortality, which are further aggravated by the presence of type 2 diabetes. With an aging population, both type 2 diabetes and osteoporosis are becoming important global health issues. This calls for clinicians’ appropriate assessment and management of bone fragility in type 2 diabetes.

It remains challenging to identify the patients with type 2 diabetes who are at risk of fragility fractures. Current fracture risk assessment tools widely adopted for the general population, such as FRAX and bone mineral density measurement by dual-energy x-ray absorptiometry, tend to underestimate the fracture risk in patients with type 2 diabetes. This is further complicated by the influence from various diabetes-specific risk factors, for instance, the level of glycaemic control, the duration of diabetes, the presence of diabetic complications, and the anti-diabetic regimen.

To translate the available clinical evidence to our daily practice, the Bone and Diabetes Working Group of the International Osteoporosis Foundation has proposed an algorithm to guide clinicians' management of bone fragility in type 2 diabetes. In addition to discussing ways to optimize the fracture risk assessment in this at-risk population including the use of trabecular bone score, the impact of anti-diabetic agents on bone and the efficacy of currently available anti-osteoporosis medications will be reviewed in this webinar.
Dr. Joanne Lam  
Honorary Clinical Assistant Professor  
Department of Medicine  
The University of Hong Kong

Update on Primary Hyperparathyroidism

Primary hyperparathyroidism (PHPT) is a common disorder of dysregulated calcium homeostasis. It is often recognized during biochemical screening or as part of an evaluation for decreased bone mass. Most of the patients have serum calcium concentrations within 0.25 to 0.375 mmol/L above the upper limit of normal with an elevated or inappropriately normal PTH level. Among such patients, the majority are asymptomatic women over 50 years old. In this talk, the clinical manifestations and diagnosis of PHPT, diagnostic localization of abnormal parathyroid gland(s), and the risks and benefits of medical versus surgical management in asymptomatic patients will be discussed.
Dr. TP Ip  
Consultant  
Department of Medicine  
Tung Wah Hospital  

Upcoming Anti-Osteoporosis Treatment

Osteoporosis is the most common metabolic bone disease such that one osteoporotic fracture is occurring in some parts of the world every three seconds. Many international treatment guidelines have conventionally recommended the use of antiresorptive drugs as first-line therapy for postmenopausal osteoporosis despite considerable literature evidences demonstrating superiority in therapeutic efficacy of bone-forming drugs over antiresorptive drugs. The major reason may probably be due to the relative high cost of bone-forming drug since there is only one bone-forming drug i.e. teriparatide is available. The Osteoporosis Society of Hong Kong (OSHK) was the first to recommend the use of this bone-forming drug as one of the first-line drugs for the treatment of established osteoporosis in the 2013 OSHK Guideline. However, the use of this drug in the public setting is subject to a number of high-level criteria such that bone-forming therapy remains not a popular treatment even in patients with very serious type of established osteoporosis.

Over the last three years, 2 other bone-forming drugs with different mechanism of action were approved by the FDA for treatment of established osteoporosis. Abaloparatide, a parathyroid hormone-related protein (PTHrP) analogue, was approved in 2017 whereas romosozumab, an antiscerostin monoclonal antibody, was approved last year. Both drugs demonstrated significant bone-forming effects in randomised controlled trials and subsequent extension studies also showed superior fracture risk reduction.

Recent epidemiological studies have also identified an “imminent risk” period which is the initial one to two years of an incident major osteoporotic fracture (MOF), within which the risk of a second MOF is particularly very high. The early use of potent bone-forming drugs in this period of imminent risk is expected to offer much better fracture risk reduction to this high-risk patient population.
The Lower the Better? Evolving Concepts of Dyslipidemia, Diabetes and Cardiovascular Disease

According to statistics from the International Diabetes Federation, cardiovascular disease is accountable for one-third to one-half of all diabetes-related deaths. Dyslipidemia, especially high low-density lipoprotein (LDL) cholesterol levels, further increases cardiovascular risks in patients with diabetes. LDL cholesterol lowering therapy has long been an integral part of management in diabetes, but this is accompanied by an ongoing debate regarding treatment goals and safety. On one hand, there is strong evidence to support a linear relationship between LDL cholesterol levels and cardiovascular risks in both primary and secondary preventions, suggesting that the lower the LDL cholesterol levels the better. On the other hand, there is also concerns regarding the adverse consequences of excessive LDL suppression, especially hemorrhagic stroke and neuropsychiatric effects. In 2019, the European Society of Cardiology (ESC) and European Atherosclerosis Society (EAS) updated their joint guidelines on the management of dyslipidemias based on the latest evidence. This talk will focus on the evidence supporting the efficacy and safety of LDL cholesterol lowering in patients belonging to different risk categories, and on the pharmacotherapeutic options (including monotherapy using statin, combination therapy using statin and ezetimibe, plus or minus proprotein convertase subtilisin/kexin type 9 (PCSK9) inhibitors) that can help to achieve the LDL cholesterol goals.
Dr. Joanna Tung  
Associate Consultant  
Department of Paediatrics  
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Paediatric Osteoporosis

Bone health is an important yet often neglected issue among children. While fragility fractures are common among children with genetic metabolic bone diseases (e.g. osteogenesis imperfecta), with better survival of patients suffering from various chronic diseases and increased use of osteotoxic medications, the incidence of secondary osteoporosis in children is also increasing. Fracture does not only result in acute bone pain, but other permanent morbidities, including premature loss of ambulation, deformity, progressive kyphosis, restrictive lung disease and even fat embolism. Compromised peak bone mass accrual in childhood would also contribute to early-onset osteoporosis in adulthood. It is paramount to identify the at-risk children for early interventions, with the management focuses on both improvement in bone mineral density and functional outcome. This talk will discuss on the burden of osteoporosis in children, as well as the diagnosis and management in this group of children based on the available evidence.
Dr. Elaine Kwan  
Consultant  
Department of Paediatrics & Adolescent Medicine  
Pamela Youde Nethersole Eastern Hospital

Big Children are NOT Small Adults

Young people are neither children nor adults. They are a unique and vulnerable population with specific physical, social, educational, vocational and developmental needs. Some people termed this specific age range between 18 to 25 years as 'Emerging Adulthood'. They are at risk of giving health care a low priority compared with other competing dimensions of their adult transition. Conversely, their feelings of invulnerability and a tendency to reject adult control further limit their receptiveness to recommendations for chronic disease management. They are at high risk of falling between the cracks of pediatric and adult health care systems, resulting in adverse health outcomes. Transitional care programs are developed to support emerging adults during the transition to adult care.

Transitional care should be a purposeful, structured and planned movement of adolescents and young adults with chronic medical conditions from a child-centered to an adult-oriented health care system. It should be an individualized and collaborative process between the youth, the family, pediatric and adult care providers. There are usually six core elements of health care transition:

1. Establish a transition policy  
2. Have a youth registry to identify those who is ready and to track progress and outcomes  
3. Transition preparation and transition readiness assessment  
4. Transition planning  
5. Transition and transfer of care  
6. Transition completion

In this lecture, we will take a look at some effective intervention strategies as well as barriers to successful transition; with special interest on 4 groups of endocrine problem including patients with diabetes, Turner syndrome, congenital adrenal hyperplasia and survivors of cancer treated during childhood. We will also share our experience of a transitional program for children with diabetes in a local hospital.
Thyrotropin Receptor Antibodies

Antibodies (Ab) to the thyrotropin (TSH) receptor (TSH-R) play an important role in the pathogenesis of autoimmune thyroid disease (AITD). TSH-R-Ab may mimic or block the action of TSH or be functionally neutral. Stimulating TSH-R-Ab are specific biomarkers for Graves' disease (GD) and responsible for many of its clinical manifestations. TSH-R-Ab may also be found in patients with Hashimoto's thyroiditis in whom they may contribute to the hypothyroidism of the disease. Measurement of TSH-R-Ab in general, and functional Ab in particular, is recommended for the rapid diagnosis of GD, differential diagnosis and management of patients with AITD, especially during pregnancy, and in AITD patients with extra-thyroidal manifestations such as orbitopathy. Measurement of TSH-R-Ab can be done with either immunoassays that detect specific binding of Ab to the TSH-R or cell-based bioassays that also provide information on their functional activity and potency. Application of molecular cloning techniques has led to significant advances in methodology that have enabled the development of clinically useful bioassays. When ordering TSH-R-Ab, clinicians should be aware of the different tests available and how to interpret results based on which assay is performed. The availability of an international standard and continued improvement in bioassays will help promote their routine performance by clinical laboratories and provide the most clinically useful TSH-R-Ab results. In conclusion, measurement of TSH-R-Ab in general, and functional (especially stimulating) Ab in particular, is recommended for the rapid diagnosis, differential diagnosis, and management of patients with GD, related thyroid eye disease, during pregnancy, as well as in Hashimoto's thyroiditis patients with extra-thyroidal manifestations and/or thyroid-binding inhibiting immunoglobulin positivity.
Maturity-onset diabetes of the young (MODY) is the most common type of monogenic diabetes. It is an autosomal dominantly inherited non-insulin dependent form of diabetes classically presenting in adolescence or young adults. MODY is a rare cause of diabetes accounting for 3-5% of DM patients. The percentage may just be underestimated as it is frequently misdiagnosed as Type 1 diabetes (T1DM) or Type 2 diabetes (T2DM). A precise molecular diagnosis is essential and provides guidance to optimize patient’s treatment and allows early diagnosis for their asymptomatic family members and reproductive planning. In this presentation, we will discuss the molecular pathway of different MODY entities, specific clinical presentations in different types of MODY and their molecular tests. Lastly we will share a few MODY cases presented to Clinical Genetic Service (CGS, DH) for discussion.
Prof. Brian Lang  
Clinical Professor  
Department of Surgery  
The University of Hong Kong  

Managing Thyroid Cancers – Updates and Future Advances

Population-based studies have demonstrated that an increasing number of incidental thyroid nodules are being identified. The corresponding increase in thyroid-based diagnostic procedures, such as ultrasound guided fine-needle aspiration cytology, has led to an increase in the diagnoses of thyroid cancers and to more thyroid surgeries being performed. Small papillary thyroid cancers (PTCs) account for most of this increase in diagnoses. These cancers are considered to be low risk because of the excellent patient outcomes, with a 5-year disease-specific survival of >98%. As a result, controversy remains regarding the optimal management of newly diagnosed differentiated thyroid cancer, as the complications related to thyroidectomy (primarily recurrent laryngeal nerve injury and hypoparathyroidism) have a considerable impact on patient quality of life. As a result, the 2015 American Thyroid Association (ATA) guidelines have recommended a more conservative approach in the treatment of low-risk thyroid cancers. This lecture will highlight the current debates, including undertaking active surveillance versus thyroid surgery for papillary thyroid microcarcinoma, the extent of thyroid surgery and lymphadenectomy for low-risk PTC, and the use of molecular testing to guide decision-making about whether surgery is required and the extent of the initial operation. The lecture will also include a discussion of current consensus guideline recommendations regarding these topics in patients with differentiated thyroid cancer. Additionally, various innovative thyroidectomy techniques (including robotic and transoral approaches) and non-surgical approaches will be discussed, with an emphasis on patient preferences around decision-making and outcomes following thyroidectomy.
Latent Autoimmune Diabetes

Latent autoimmune diabetes is an admixture of immune-mediated and type 2 diabetes genetically and phenotypically. In the recently updated diabetes classification scheme of the WHO, this condition is renamed as "slowly evolving immune mediated diabetes of adults", under a new diabetes category of “hybrid forms of diabetes”. A number of studies have shown that patients with latent autoimmune diabetes have higher frequencies of type 1 and type 2 diabetes-susceptibility genotypes compared with healthy controls. Among Chinese, 6% of adults and 8% of youth with non-type 1 diabetes have evidence of anti-islet autoimmunity. Ant-glutamic acid decarboxylase (GAD) auto-antibodies are the most sensitive in identifying immune-mediated disease although the levels may decline over time. In general, patients with latent autoimmune diabetes have fewer metabolic risk factors compared with those with type 2 diabetes but still, up to half are obese and half have family history of diabetes. It should also be recognised that even among patients with latent autoimmune diabetes, a spectrum exist in the intensity of autoimmunity and metabolic burden, the former can be determined based on anti-GAD titre and the presence of other anti-islet and organ-specific auto-antibodies. Prognosis of patients with latent autoimmune diabetes is distinguished from type 2 diabetes by a lower incidence of cardiovascular disease and mortality, whilst their risks of microvascular complications are higher. Studies on the efficacy of glucose-lowering treatments in latent autoimmune diabetes are extremely limited. In general, glycaemic response to insulin is greater in latent autoimmune diabetes than type 2 diabetes but there is no available evidence for early versus late initiation of insulin with respect to long-term outcome. Limited data indicate a faster deterioration of beta-cell function with sulphonylurea and this class of drugs should be discouraged. In patients with residual beta-cell function, DPP-4 inhibitors may be an alternative to insulin for maintaining glycaemic control. In our gradual movement towards personalised medicine, there is a strong argument for regular testing of anti-islet autoimmunity especially in younger patients with diabetes because this delineation of disease aetiology has implications on guiding treatment and prognosis.
Radioactive Iodine: Thyrotoxicosis and Thyroid Cancer

Radioactive iodine-131 is commonly used in diagnosis and management of thyroid disease (both thyrotoxicosis and thyroid cancer).

Graves' disease and toxic nodular goitre account for most cases of thyrotoxicosis associated with hyperthyroidism. The main therapeutic options are antithyroid drugs, radioactive iodine and surgery. In Graves' disease, carbimazole and propylthiouracil achieve long-term remission in approximately 35% of cases. Iodine-131 can be used as a first-line therapy for thyrotoxicosis or as second-line treatment after failure of anti-thyroid medications. It is administered orally, in capsule or liquid form. Around 90% patients after radioactive iodine-131 can achieve cure in 6 months. However, it is contraindicated in pregnancy (as it may ablate the fetal thyroid) and in breastfeeding (because of concentration in breast milk).

Thyroid cancer is the tenth most common cancer in Hong Kong. The incidence of thyroid cancer has increased substantially worldwide in the past decade. Surgery is the mainstay of treatment in well-differentiated thyroid cancer. Radioactive iodine is recommended for patients with intermediate and high risk of recurrence. It helps to eradicate the remnant thyroid cells to increase the sensitivity of serum thyroglobulin for disease monitoring and future surveillance with iodine scan. It also reduces risk of local and distant recurrence. The use of external beam radiotherapy is mainly for older patients with R2 resection. Radioactive iodine is not indicated in medullary thyroid carcinoma or anaplastic thyroid carcinoma.
Intensify Treatment in Type 2 Diabetes Mellitus with GLP-1 Receptor Agonists

Pathophysiology of Type 2 diabetes mellitus (T2DM) is complex and consists of at least eight mechanisms causing hyperglycaemia (“The Ominous Octet”). The modern concepts of clinical diabetes management should base therapy on pathophysiology. The progressive natural history of T2DM often necessitates the use of multiple anti-diabetic drugs to achieve therapeutic targets. Glycogen-like peptide 1 receptor agonists (GLP-1 RAs), which correct six of the eight components of the Ominous Octet is a very attractive option in management of T2DM. GLP-1 RAs, in addition, prevent or reverse the progressive β-cell failure and rise in HbA1c, and reduce cardiovascular risk in T2DM independent of their glucose-lowering ability.

According to their pharmacokinetic and pharmacodynamic profiles, GLP-1 RAs can be classified as short-acting (Exenatide and Lixisenatide) or long-acting (Exenatide-LAR, Liraglutide, Dulaglutide and Semaglutide). Long-acting GLP-RAs can maintain high GLP-1 levels and provide continuous stimulation on insulin secretion for 24 hours, even during fasting periods, resulting in a greater reduction in fasting hyperglycaemia. The effect of these GLP-1 RAs on postprandial hyperglycaemia can be attributed mainly to suppression of glucagon secretion, reduced appetite, and slower gastric emptying. On the other hand, the effect of short-acting GLP-1 RAs on insulin secretion during the fasting period is less pronounced than that of long-acting ones, whereas the effect on gastric emptying is more pronounced.

In the perspectives of personalized treatment of patients with T2DM, the increasing availability of GLP-1 RAs with different pharmacokinetic properties means that the selection of the drug should be determined by a series of factors: the patient’s specific needs and characteristics, pharmacokinetic properties, anti-hyperglycaemic efficacy, effects on associated disease processes and safety profile.
Dr. Sofie Yung  
Clinical Assistant Professor  
Department of Obstetrics & Gynaecology  
The University of Hong Kong  

Polycystic Ovary Syndrome  

Diagnosis  
Polycystic ovary syndrome (PCOS) is a common endocrine disorder affecting reproductive aged women. Main clinical features include menstrual irregularities, hyperandrogenism and polycystic ovaries on ultrasound. The diagnosis is based on fulfilling two out of these three criteria, together with exclusion of other conditions which can give similar clinical features.

Associated health conditions  
Women with PCOS have increased risk of obesity, type 2 diabetes, dyslipidaemia and possibly cardiovascular diseases. Chronic anovulation may result in infertility, and increases the risk of endometrial hyperplasia and cancer. They also have higher rates of depression and anxiety.

Management  
The treatment goals of PCOS are amelioration of symptoms, identifying and reducing metabolic risks, prevention of endometrial hyperplasia and cancer, and inducing ovulation for those pursuing pregnancy. Lifestyle modification and weight reduction for overweight and obese women are important to reduce metabolic risks and improve reproductive outcomes. Cardiometabolic risks should be assessed and managed accordingly. Hyperandrogenic symptoms can be treated with various medical treatments in women not pursuing pregnancy. Combined oral contraceptive pills or progestogens, depending on fertility wish and patient preference, are used to prevent endometrial hyperplasia and cancer. In women with PCOS-associated anovulatory infertility, letrozole is now the first-line ovulation induction drug over clomiphene citrate. Gonadotrophins and laparoscopic ovarian drilling are second-line treatment to induce ovulation. If ovulation induction fails to achieve pregnancy, in-vitro fertilization is the third-line treatment. Depressive and anxiety symptoms should also be screened.
Sellar & Suprasellar Tumor for Endocrinologists

Pituitary adenoma is among one of the most common encountered intracranial tumor. More than half of them are secretory. Prolactin, growth hormone, adrenocorticotropic hormone are by far the common secreting hormones. Treatment includes surgery, medical treatment and stereotactic radiosurgery. MRI is the imaging modality of choice, unless contraindicated. Radiological assessment should include suprasellar extension, hence compression to the optic chiasm or hypothalamus. Parasellar extension reveals cavernous sinus invasion, internal carotid artery involvement. Hemorrhage is not uncommon encounters for pituitary adenoma. It ranges from asymptomatic to pituitary apoplexy. Surgical approaches for pituitary adenoma include frontal and endoscopic transsphenoidal approach. Important normal variants should be remembered upon reviewing the MR images of the pituitary adenoma, which might have paramount effect to the surgical outcome. Invasive prolactin-producing pituitary adenoma is uncommon, however, should be recognized. They can present as incidental findings as the prolactin related symptomatology as reduced libido can be very subtle and not recognized by both the patients and their family doctors. Lesions not to be touched upon include hypothalamic hamartoma, ectopic posterior pituitary gland, enlarged pituitary gland secondary to other pathology as intracranial hypotension. For pituitary microadenoma, contrast MRIs have a sensitivity of 90%. Inferior petrosal sinus venous sampling can be used to lateralize pituitary microadenoma prior to surgery; it can also be used to confirm the presence of the microadenoma if the MRI is not revealing.

Other than pituitary gland, endocrinologists might also be interested in the pineal gland. Pineal cysts are the most common imaging encounters. The vast majority of the pineal cysts are small and free of symptoms. Pineal germinomas accounts for about half of the pineal tumors. Paraventricular location of germinomas, ie involvement of basal ganglia and thalami, are not uncommon in our population. Germinoma has propensity to bleed, might have cystic component and enhance vividly on MR upon administration of contrast.
Dermatosis in Endocrinology

Similar to other systemic diseases, endocrine disorders can present with cutaneous manifestations. It is important to recognize these cutaneous manifestations as they may be clues to underlying diseases. Some common endocrinology diseases with cutaneous presentations, its differentials, and treatments will be discussed. Rare cutaneous diseases will also be discussed - as it is important not to miss them!
Prof. Gilberto Leung
Clinical Professor
Department of Surgery
The University of Hong Kong

Pituitary Surgery for Endocrinologists: A Contemporary Perspective

Harvey Cushing once abandoned the transsphenoidal approach because of unsatisfactory patient outcomes. It was only after endocrinologists had come to know more about the pituitary axis that transsphenoidal surgery had had its renaissance - no longer was relief of mass effect and visual symptoms the only gauge of therapy; hormonal normalisation and preservation and replacement of pituitary function had become attainable goals. Neurosurgeons have not looked back since and soon established endoscopic transsphenoidal surgery as the standard treatment for pituitary tumours in one of the most sophisticated and rewarding physician-surgeon partnerships in the modern era.

An often unheeded but pertinent question is whether a pituitary lesion should primarily be understood and treated as an endocrinological condition, or simply a “unwanted mass” to be taken out. Like most things in medicine, the practice of pituitary surgery has swung the pendulum from the overly cautionary to the mindlessly aggressive, ending at the extremes with either unrelenting hypersecretion and metabolic conundrums or total pituitary failure and neurological catastrophes, or both. Between good intention, patient safety, surgical prowess, and the endocrinological ideals is a fine balance awaiting and important lessons to be learnt.

What is (not) so “minimally invasive” about transsphenoidal surgery? What are the realistic goals and limits? When is craniotomy a sensible alternative? And radiosurgery? What can patients and endocrinologists reasonably expect? This paper aims to examine these issues in light of the contemporary principles and practices of pituitary surgery.
Role of Fixed-Ratio Combination of Basal Insulin and GLP-1 RA in Type 2 Diabetes Management

The year 2021 marks the 100th anniversary of insulin discovery by Dr Banting. Insulin is not only essential, but also crucial in glycemic control, especially under certain clinical conditions such as type 1 diabetes or gestational diabetes. Despite the numerous anti-diabetic agents being invented and developed into clinical use over the past decades, insulin still plays a pivotal role in diabetes care.

Basal insulin is highly effective in reducing fasting blood glucose. However, it is associated with considerable risk of hypoglycaemia and weight gain. Owing to concerns of these untoward side effects, basal insulin initiation and intensification are sometimes delayed by clinicians. On the other hand, glucagon like peptide 1 receptor agonist (GLP-1 RA) has often been used recently as the first injectable among patients with diabetes, attributable to its efficacy in terms of glycemic control (both fasting and postprandial) and the associated benefits of weight loss and low hypoglycaemic risk. The dose-related gastrointestinal side effects, especially nausea, may be minimized through a slow dose escalation.

The latest American Diabetes Association guideline set prevention of complication and optimization of quality of life as the goals of diabetes care. A management plan is tailored to each individual patient based on the assessment of his/her key characteristics. The guideline encourages the incorporation of those anti-diabetic agents with cardiovascular and/or renal benefits beyond glucose-lowering effects, such as sodium-glucose co-transporter-2 inhibitor and GLP-1 RA, into the treatment regimens of high risk patients.

The fixed-ratio combination (FRC) of GLP-1 RA and insulin requires only one injection per day which could obviate the need to have multiple daily injections and allow gradual dose titration. In my presentation, basic information and trials of FRC will be discussed, and clinical experience in initiating FRC will also be shared.
Graves’ Orbitopathy

Graves' orbitopathy or thyroid eye disease (TED) is a complex autoimmune disorder which causes substantial morbidity. It can result in orbital disfigurement, double vision, and visual loss. Consequently, it has a substantial negative effect on quality of life, mental health, and socioeconomic status. Most signs and symptoms of TED can be explained by the expansion of the orbital contents. Steroids are the mainstay of treatment in TED. However, recurrence may occur once steroids are withdrawn. Furthermore, in most cases, normal orbital anatomy is not restored, and skilled rehabilitative surgery is required to reduce disfigurement, double vision, and to preserve vision. Therefore, novel, causal, and more efficacious treatment strategies are warranted. In the last decade, the pathophysiology of TED has also been revised with the identification of new potential therapeutic targets. Recent clinical trials have shown that considerable benefit may be derived from the addition of antiproliferative agents (e.g., mycophenolate sodium) in preventing deterioration after steroid cessation. In addition, targeted biologic therapies have shown promise, including teprotumumab (anti-IGFR) which appears to substantially reduce proptosis, rituximab (anti-CD20) which reduces inflammation and tocilizumab (anti-IL-6) which potentially benefits both of these parameters.
Realise the Potential of the New GLP-1 RA in DM Treatment

GLP-1 receptor agonists (GLP-1RAs) are an incretin-based class of drugs that have been shown to have high glucose-lowering efficacy and to induce decreases in bodyweight. GLP-1RAs are included in current national and international guidelines as second-line treatment especially in patients with type 2 diabetes and concomitant cardiovascular disease (CVD). First-generation GLP-1RAs (exenatide, lixisenatide, liraglutide) were given once or twice daily, but longer-acting GLP-1RAs have now been developed for once-weekly administration – e.g., exenatide ER, dulaglutide and semaglutide. The efficacy and safety of semaglutide have been investigated in an extensive clinical development program including more than 9,000 patients with type 2 diabetes. Semaglutide has been compared head-to-head with a dipeptidyl peptidase-4 (DPP4)-inhibitor, sodium-dependent-glucose transporter-2 (SGLT2)-inhibitor, other GLP-1RAs and basal insulin. In these studies, semaglutide was found to provide significant and clinically relevant reductions in HbA1c, fasting plasma glucose (FPG), glucose excursions, body weight and blood pressure. The reduction in glycaemic parameters was more pronounced than that in the comparator GLP-1RAs. The rate of hypoglycemia is very low during treatment with semaglutide if not combined with sulphonylureas or insulin. In a cardiovascular outcome trial (SUSTAIN 6) not only noninferiority was confirmed, but also superiority compared with placebo in a population of patients with type 2 diabetes and high risk of CVD. The safety profile of semaglutide is well established and indicates that gastrointestinal side effects dominate, as observed with other GLP-1RAs. As also observed with other GLP-1RAs, side effects such as nausea and vomiting diminished over time during continuous treatment. Regarding microvascular complications, an unexpected increase in diabetes-related retinopathy was observed in the CVOT (SUSTAIN 6), most likely due to a nonspecific effect of a rapid decrease in glycaemic parameters in patients with preexisting retinopathy with high HbA1c at the start of the treatment. Overall, semaglutide was efficacious across the spectrum of type 2 diabetes and more effective than comparators for glycaemic control and weight loss.
Addressing the Unmet Need in Diabetes: Role of Antithrombotic Therapy

Diabetes is one of the major risk factors for atherosclerotic cardiovascular disease, including coronary artery disease and peripheral artery disease. Despite a broad armamentarium of effective glycemic control and lipid-lowering medications, diabetes still contributes to a prothrombotic state and residual cardiovascular risk. Antiplatelet therapy, including dual antiplatelet therapy, represents the mainstay of treatment for secondary prevention of ischemic events. Until recently, a strategy of dual antithrombotic therapy with an antiplatelet and an anticoagulant has been developed to further reduce the risks of adverse ischemic cardiac and limb events in patients with established coronary artery disease and peripheral artery disease. As a result, multiple international guidelines recommend addition of a second antithrombotic agent to aspirin in patients at high ischemic risk and low bleeding risk. In summary, antithrombotic therapy is critical to improving outcomes in patients with atherosclerotic cardiovascular disease and should be individualized based on patient thrombotic risk and bleeding risk.
Bone Health Assessment

Osteoporosis is a systemic skeletal disease hallmarked by compromised bone mass and quality with subsequent bone fragility. It is a major public health concern and its prevalence has been increasing over the years. Fragility fractures result in significant mortality and morbidity.

It is vital that clinicians are able to perform bone health assessment, in order to identify patients who are at risk of osteoporotic fracture and those who would benefit from anti-osteoporotic pharmacotherapy.

Bone densitometry is widely adopted to predict the risk of future osteoporotic fracture in an individual. It is also used to diagnose osteoporosis. However, a substantial number of patients with normal bone mineral density sustain fragility fracture. It has become apparent that bone mineral density only quantifies bone mass, which is one of the many properties that contributes to bone strength. However, bone strength is a factor of both bone mass and bone quality. Fracture risk assessment tool incorporating clinical risk factors, bone mineral density and trabecular bone score can better predict the risk of future osteoporotic fracture in an individual.
When are Your Patients' Abnormal Lipid Levels Not Their Fault?

Elevated plasma cholesterol and high triglycerides are frequently considered to be primarily the fault of a patient's lifestyle habits. However, inherited lipid disorders are not rare. Awareness of when hyperlipidemia is likely inherited rather than the result of an unhealthy lifestyle, or secondary to other medical conditions, is critical in the management of these patients. When to suspect an inherited condition based on history, physical examination, and laboratory measurement of lipid levels; and the approach to managing these disorders including genetic testing will be discussed.
Dr. CH Choi
Consultant
Department of Medicine
Queen Elizabeth Hospital

Dynamic Tests in Endocrinology

Why we need dynamic tests?
MCQ:
1. Instinct (like oculostenotic or oculostent reflex in cardiologist)
2. Special affiliation with Chemical Pathologist
3. Romanticism
4. Avoid Dunning-Kruger effect
5. Others

Content of the talk:
1. We all (should) have our own cook book
2. Noise everywhere
3. Good answer only come from good question
4. Between Scylla & Charybdis
5. 'Where is the knowledge we have lost in information?' - T.S.Eliot
6. 'Uncertainty is the only certainty there is.' - J.A.Paulos
7. Don't lost in Oblivion
8. Continua a lottare
Prof. Chris Wong
Professor
Department of Biology
Hong Kong Baptist University

Effects of Perfluorinated Compounds on Animal Metabolic Functions

The prevalence of metabolic diseases is on an uprising trend. Although susceptible genetic background, over-nutrition, and physical inactivity are identified as significant risk factors contributing to the disease development, they are insufficient to account for the high prevalence. Over the last 20 years, converging epidemiological and experimental data have pointed out that the risk of developing metabolic syndrome is associated with early-life exposure to endocrine-disrupting chemicals (EDCs). Apparently, EDC-elicited suboptimal maternal-intrauterine environment perturbs fetal metabolic programming, thereby increasing the susceptibility to developing metabolic diseases (i.e., obesity, type-2 diabetes) in adulthood. Although the risk of EDC exposure during fetal development is utmost, the underlying effects have not been fully elucidated.

Humans are exposed to complex mixtures of environmental contaminants. Perfluorinated compounds (PFCs) are EDCs and environmental obesogens. In the European research project OBELIX, exposure to PFCs was proposed to be a risk factor in the alternation of metabolic programming. There is a considerable amount of human data correlating PFC exposure to the risk of metabolic diseases. Multiple numbers of cohort studies using paired maternal-fetal samples reported the association of maternal plasma PFCs with neonatal anthropometry and adiposity. PFCs are measurable in human plasma, umbilical blood, and breast milk. There is a strong association between maternal and cord blood PFC concentrations, suggesting the placental transfer to affect growth in early life. Experimental studies in animal and cell models have demonstrated the effects of PFCs to alter glucose and lipid metabolism, via perturbation to pancreatic -cells, adipocyte, and liver functions. Presumably, PFCs could alter fetal metabolic programming through similar effects. In this talk, we discuss the health impacts of PFCs on animal metabolism.
Supporting Organizations

- Association of Hong Kong Diabetes Nurses
- Hong Kong Obesity Society
- The Hong Kong Society for Rehabilitation
- The Hong Kong Society of Paediatric Endocrinology and Metabolism
- Youth Diabetes Action
Prolia® (Denosumab) Abbreviated Prescribing Information

Prolia® (denosumab) Solution for injection in Pre-Filled Syringe 60 mg/mL. INDICATIONS Prolia is indicated for: (i) treatment of postmenopausal women with osteoporosis at high risk for fracture, defined as a history of osteoporotic fracture, or multiple risk factors for fracture; or patients who have failed or are intolerant to other available osteoporosis therapy; (ii) treatment to increase bone mass in men at high risk for fracture receiving androgen deprivation therapy for nonmetastatic prostate cancer. In these patients Prolia also reduced the incidence of vertebral fractures; (iii) treatment to increase bone mass in women at high risk for fracture receiving adjuvant aromatase inhibitor therapy for breast cancer. DOSAGE AND ADMINISTRATION: The recommended dose of Prolia is 60 mg administered as a single subcutaneous injection once every 6 months. Administer Prolia via subcutaneous injection in the upper arm, the upper thigh, or the abdomen. All patients should receive calcium 1300 mg daily and at least 600 IU vitamin D daily. CONTRAINDICATIONS: Hypersensitivity to any component of the product. SPECIAL WARNINGS AND PRECAUTIONS FOR USE (Denosumab): Clinically significant hypocalcemia including asymptomatic hypocalcemia has been reported with Prolia. Symptoms have included hypocalcemia, dysphonia, throat tightness, voice change, and paresthesia. Hypocalcemia typically occurs within hours to several days of administration of the initial dose of Prolia. If hypocalcemia occurs, calcium should be administered and symptomatic hypocalcemia should be treated. If asymptomatic hypocalcemia occurs, calcium should be administered and discontinuation of Prolia should be considered. Prolia is not indicated for the treatment of Paget’s disease of bone. Hypocalcemia may occur following administration of human bisphosphonates. Prolia administration is a significant risk in patients with severe renal impairment (creatinine clearance < 30 mL/min) or receiving dialysis. Adequately supplement all patients with calcium and vitamin D. Suppression of parathyroid hormone (PTH) has been reported in patients receiving Prolia. The onset of treatment or a dose change of treatment may lead to an unexplained increase in serum calcium in the range of 2-3 mg/dL. A decline in serum calcium with preventive therapy is not recommended prior to treatment with Prolia in patients with concurrent risk factors. All patients should be encouraged to maintain good oral hygiene, undergo routine dental check-ups, and, immediately report any oral symptoms such as dental mobility, pain or swelling, or non-healing sores or discharge during treatment with Prolia. While in treatment, invasive dental procedures should be performed with caution and avoided in close proximity to Prolia treatment. Osteonectin Substitution and Osteonal Remodeling, Hypocalcemia, Hypocalciuria, or low urinary excretion of the shaft have been reported in patients receiving Prolia. Patients should be advised to report new or unusual thigh, hip, or groin pain. Multiple Myeloma, Fractures: Risks Following Discontinuation of Prolia Treatment. Following discontinuation of Prolia treatment, fracture risk increases, including the risk of multiple vertebral fractures. If Prolia treatment is discontinued, consider transitioning to an alternative antiresorptive therapy. Serious infections: Serious infections leading to hospitalization were reported in clinical trials. Advise patients to seek prompt medical attention if they develop signs or symptoms of severe infection, including cellulitis. Dermatologic Adverse Reactions: Dermatitis, eczema, and rash. Most of these events were not specific to the injection site. Consider discontinuing Prolia if severe symptoms develop. Musculoskeletal Flud: Swelling and/or occasionally unexplained bone, joint, or muscle pain. Consider discontinuing use if severe symptoms develop. Supersensitization of Bone Disease: In clinical trials treatment with Prolia resulted in significant suppression of bone remodeling as evidenced by markers of bone turnover and bone histomorphometry. Osteonecrosis of the external auditory canal: Osteonecrosis of the external auditory canal has been reported with denosumab. Possible risk factors include staring use and chemotherapy and/or local risk factors such as infection or trauma. INTERACTIONS: In subjects with postmenopausal osteoporosis, Prolia 60 mg subcutaneously injected did not affect the pharmacokinetics of ibandronate, which is metabolized by cytochrome P450 3A4 (CYP3A4), indicating that it should not affect the pharmacokinetics of drugs metabolized by this enzyme in this population. PREGNANCY AND LACTATION: Pregnancy: Category B. Lactation: It is not known whether Prolia is excreted into human milk. PEDIATRIC USE: In a radiographic study comparing multiple doses of denosumab in the pediatric group, denosumab produced a similar incidence of osteonecrosis to the adult group. Renal Impairment: Prolia is not recommended in pediatric patients. Safety, its overall differences in safety or efficacy were observed in clinical studies between elderly patients and younger patients and other reported common adverse reactions has not identified differences in responses between the elderly and younger patients, but greater sensitivity of some older individuals cannot be ruled out. Renal impairment: No dose adjustment is necessary in patients with renal impairment. UNREASSRABLE EFFECTS: The most common adverse reactions reported with Prolia in patients with postmenopausal osteoporosis are back pain, pain in extremity, musculoskeletal pain, hypocalcemia, arthralgia, and tendinitis. The most common adverse reactions reported with Prolia in patients with bone loss receiving androgen deprivation therapy for prostate cancer or adjuvant aromatase inhibitor therapy for breast cancer are arthralgia and back pain. Pain in extremity and musculoskeletal pain have also been reported in clinical trials. The most common adverse reactions leading to discontinuation of Prolia in patients with postmenopausal osteoporosis are back pain and constipation. OVERDOSE: There is no experience with overdosage with Prolia. Abbreviated Prescribing Information Version: 03/2019

References: 1. Henry Q Bore, Rachell B Wagenman, Marie L. Brandt, et al. The Journal Diabetes & Endocrinology 2017;7(6): S15-32. 2. Please read the full prescribing information prior to administration and full prescribing information is available upon request. This material is for the reference and use by healthcare professionals only. For medical inquiries and adverse event reporting, please contact Medical Information at 800-661-1412 (English only). Prolia® and RANKL® are registered trademarks owned or licensed by Amgen Inc., its subsidiaries, or affiliates.
For people with Diabetes

Contour plus ONE
Blood Glucose Monitoring System

- Simple to use and remarkable Accuracy, going beyond the minimum Accuracy requirements of the ISO15197:2013 standard
- Results easily understood with the smartLIGHT® feature
- Second-Chance® sampling: Accurate reading, even after applying more blood to the same strip
- Smart diabetes management with the CONTOUR® DIABETES App

Contour plus

- Simple to use and remarkable Accuracy, going beyond the minimum Accuracy requirements of the ISO15197:2013 standard
- The Second-Chance® sampling feature:
  - Reduces frustration about having to re-apply**
  - Could save up to 100 strips per year**

For detection of hypoglycemia, meeting current ISO15197:2013 standards is not enough

It's time for a change: choose Accuracy and Simplicity.

For more information, please visit www.contourplusone.hk or contact us at 8100 6386 and diabetescare@ascensia.com

* 99% of the measured glucose values needed to fall within either ±15 mg/dL (±0.83 mmol/L) of the average measured values of the reference measurement procedure at glucose concentrations ≤100 mg/dL (≤5.56 mmol/L), or within ±15% at glucose concentrations ≥100 mg/dL (≥5.56 mmol/L), 99% of individual glucose measured values needed to fall within zones A and B of the Consensus Error Grid (CEG) for type 1 diabetes.
*** See 6.3.3 para 1a.
**** 96% of the measured glucose values needed to fall within ±15 mg/dL (±0.83 mmol/L), of the average measured values of the reference measurement procedure at glucose concentrations ≤5.56 mmol/L, ≤100 mg/dL, ≤5.56 mmol/L, ≥5.56 mmol/L, ≤100 mg/dL, ≤5.56 mmol/L, ≥5.56 mmol/L, 96% of individual glucose measured values needed to fall within zones A and B of the Consensus Error Grid (CEG) for type 1 diabetes.
***** Millward Research. Ascensia Diabetes Care. Tel 6 & Partners online market research, conducted April/May 2015.
********** Average area-waste estimated by healthcare professionals and indicated as a potential benefit of the Second-Chance® sampling feature. Data and quotes collected through online survey of 400 HCPs (nephrologists with diabetes in US, CAN, DE, and UK).
Ascensia Diabetes Care Hong Kong Limited Room 3102-13, Millennium City 1 Tower 1, 388 Kwan Tong Road, Kwan Tong, Hong Kong T: +852 8100 6386 diabetes.ascensia.com Ascensia, the Ascensia Diabetes Care logo, CONTOUR®, Second-Chance are trademarks of Ascensia Diabetes Care Holdings AG.
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Xarelto®
Tailored Protection for Your Cardio-Vascular Patients

INDICATED FOR CAD OR SYMPTOMATIC PAD

Xarelto 2.5 mg film-coated tablet
Abbreviated Prescribing Information
(Please refer to full prescribing information before prescribing.)
Composition: Active ingredient: 2.5 mg rivaroxaban. Excipients: Microcrystalline cellulose, croscarmellose sodium, lactose monohydrate, hypromellose 2910, sodium lauryl sulphate, magnesium stearate, martragel 3350, titanium dioxide [E171], iron oxide red [E172].

Indication and Posology: Prevention of atherothrombotic events in adult patients with coronary artery disease (CAD) or symptomatic peripheral artery disease (PAD) at high risk of ischemic events, co-intervened with aspirin (ASA). The recommended dose is 2.5 mg once daily, with a daily dose of 75 - 100 mg ASA. Duration of treatment should be determined for each individual patient based on regular evaluations and should consider the risk for thrombotic events versus the bleeding risk.

Contraindications: Hypersensitivity to the active substance or any of the excipients; active clinically significant bleeding; recent or condition considered a significant risk for major bleeding; concurrent treatment with any other anticoagulants except under specific circumstances of switching anticoagulant therapy or when unfraccionated heparin is given at doses necessary to maintain an APTT ratio of 1:1 with an aminocaproic acid; concurrent treatment of CAD/PAD with ASA in patients with previous haemorrhage or lacunar stroke, or any stroke within a month, hepatic disease associated with coagulopathy and clinically relevant bleeding risk including cardiac patients with PCI, PPI and C. pneumonia and breastfeeding.

Warnings and Precautions: Clinical surveillance in line with anticoagulation practice is recommended throughout treatment. Xarelto should be discontinued in case severe haemorrhage occurs. Increasing age may increase haemorrhagic risk. Xarelto should be discontinued in case the risk is balanced by potential benefit or other anticoagulants in conjunction with a medical lesion. Not recommended: in patients with severe renal impairment (creatinine clearance <15 ml/min), in patients receiving concurrent systemic treatment with strong CYP3A4- and P-gp inhibitors, i.e. azole-antimicrobials or H2 blockers also inhibitors, in patients with increased bleeding risk, in patients receiving concurrent treatment with strong CYP3A4 inhibitors unless the patient is closely observed for any signs of thrombosis, not recommended due to lack of data: treatment in combination with antipillar agents other than ASA, in patients below 18 years of age; in patients concomitantly treated with warfarin; in patients with synthetic heart valves. Use with caution: in conditions with increased risk of haemorrhage; in patients with severe renal impairment (creatinine clearance 15-29 ml/min) or with moderate renal impairment (creatinine clearance 30-49 ml/min) concomitantly receiving other medicinal products which increase rivaroxaban plasma concentrations; in patients treated concomitantly with medicinal products affecting haemostasis. In patients ≤75 years of age or with lower body weight; when neurectomy anaesthesia or surgical puncture is employed. Patients on treatment with Xarelto and ASA should only receive concurrent treatment with NSAIDs if the benefit outweighs the bleeding risk. In patients at risk of ulcerative gastrointestinal disease prophylactic treatment may be considered. Although treatment with rivaroxaban does not require routine monitoring of exposure, rivaroxaban levels measured with a calibrated quantitative anti-Factor Xa assay may be useful in exceptional situations. Xarelto contain lactose.

Undesirable effects: Common: anaemia, dizziness, headache, ear haemorrhage, hypotension, haematuria, epistaxis, haemoptysis, gingival bleeding, gastrointestinal tract haemorrhage, gastrointestinal and abdominal pain, dyspepsia, nausea, constipation, diarrhoea, vomiting, increase in transaminases, pruritis, rash, eczema, urticaria, and subcutaneous haemorrhage, pain in extremity, oesophageal tract haemorrhage, renal impairment, fever, lower pulmonary, decreased general strength and energy, post-procedural haemorrhage, conduction, void retention, constipation, thrombocytopenia, allergic reaction, dermatitis allergic, angioedema and allergic oedema, cerebral and intracranial haemorrhage, syncope, asthenia, dry mouth, hepatic impairment, increase in bilirubin, blood alkaline phosphatase and GGT, urticaria, haemorrhage, feeling unwell, increase in ESR, lipase, amylase. Rare: jaundice, bile duct conjugated increased, cholestasis, hepatitis (with hepatitis B and C liver failure secondary to a bleeding). Reference: 1. Xarelto® Hong Kong Reference Prescribing Information (February 2019).

Bayer Healthcare Limited
149, Oxford House, Tai Kok Tsui, 979 King’s Road, Quarry Bay, Hong Kong
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- A ready-to-use pen designed with patients in mind

Automatic dose delivery
Each pen contains 1 dose of Trulicity®
- No reconstitution or priming required
- Pre-attached, hidden needle

Trulicity®

T2DM = Type 2 diabetes mellitus

Trulicity® is indicated in adults with type 2 diabetes mellitus to improve glycemic control as Monotherapy: When diet and exercise alone do not provide adequate glycemic control in patients for whom the use of metformin is considered inappropriate due to intolerance or contraindications. Add-on therapy. In combination with other glucose-lowering medicinal products including insulin, when these, together with diet and exercise, do not provide adequate glycemic control. Doseage: Adult Monotherapy: 0.75 mg once weekly. Add-on therapy: 1.5 mg once weekly. Elderly ≥75 years old: Initial 0.75 mg once weekly. Renal impairment: No dosage adjustment is required in patients with mild, moderate, or severe renal impairment (cGFR ≥20 to ≤89 mL/min/1.73 m²). Administration: To be injected subcutaneously in the abdomen, thigh, or upper arm. It should not be administered intravenously or intramuscularly. The dose can be administered at any time of day, with or without meals. Contraindications: Hypersensitivity to dulaglutide or any of its excipients. Special Precautions: Do not use in patients with type 1 diabetes mellitus or for the treatment of diabetic ketoacidosis. Do not administer IV. Acute pancreatitis. Hypoglycemia. Limited experience in patients with congestive heart failure. Adverse Drug Reactions: Hypoglycemia, nausea, diarrhea, vomiting, abdominal pain, decreased appetite, dyspepsia, constipation, flatulence, abdominal distention, gastroesophageal reflux disease, eructation, fatigue, injection site reactions, acute pancreatitis, sinus tachycardia, first-degree atrioventricular block.

Full prescribing information is available upon request.

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Meets ISO standard 15197:2013¹ and delivers even more precise 10/10 accuracy²

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Easier monitoring
Strip port light, wide yellow application area and touch-free strip removal

Smartly stored data
Wirelessly sends results to the mySugr app

¹. ISO 15197:2013 In vitro test systems requirements for blood glucose monitoring systems for self-testing in managing diabetes mellitus.

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HELP MORE PATIENTS
PUT HIGH PERCENTAGES BEHIND THEM

74% OAD UNCONTROLLED PATIENTS AT GOAL

50% SHORTEN TIME TO ACHIEVE TARGET

2X GREATER A1C DROP THAN GLARGINE ALONE IN BASAL UNCONTROLLED PTS

No ADDITIONAL RISK OF HYPOGLYCEMIA

STUDY DESIGN

LIXILAN-L was an open-label, randomised, parallel-group, multinational, multicentre phase 3 clinical trial designed to evaluate the efficacy and safety of SOLIQUA™ vs insulin glargine 100 Units/ml in 736 patients ≥18 years of age with type 2 diabetes for ≥1 year before screening and uncontrolled on basal insulin with or without up to 2 oral antidiabetics (OADs) for ≥28 months. The primary efficacy endpoint was change in HbA1c from baseline to Week 30. Eligible patients (n=1018) entered a 6-week run-in phase in which patients remained on or switched to insulin glargine 100 Units/ml, in case they took another basal insulin, and had their insulin dose titrated/stabilised while continuing metformin (if previously taken). Any other OADs were discontinued. At the end of the run-in period, patients with an HbA1c between 7% and 10%, FPG ≥7.77 mmol/L, and insulin glargine daily dose of 20 to 50 Units, were randomised to either SOLIQUA™ (n=367) or insulin glargine (n=369). Soliqua showed significantly greater reduction in HbA1c compared to insulin glargine (-1.1% vs -0.6%, p<0.0001). The safety profile of SOLIQUA™ generally reflected the established safety profiles of its components after 30 weeks of treatment.1,2

LIXILAN-0 was an open-label, randomised, parallel-group, multinational phase 3 clinical trial designed to evaluate the efficacy and safety of SOLIQUA™ vs insulin glargine 100 Units/ml and lixisenatide in 1170 patients ≥18 years of age with type 2 diabetes for ≥18 before screening and uncontrolled with metformin +/- a second oral antidiabetic ≥3 months. The primary endpoint was change in HbA1c from baseline to Week 30. SOLIQUA™ demonstrated significantly greater HbA1c reduction at Week 30 vs insulin glargine 100 Units/ml and vs lixisenatide (-1.1% vs -1.2% vs -1.3% vs -0.85%, p<0.0001). The safety profile of SOLIQUA™ generally reflected the established safety profiles of its components after 30 weeks of treatment.1,2

A post hoc analysis of LIXILAN-0 compared efficacy and hypoglycaemia outcomes at early study visits with IGlarLixi (insulin glargine U100 (IGlar) and lixisenatide) vs IGlar alone in patients with type 2 diabetes uncontrolled on oral antidiabetic therapy. FPG control, defined as days to achieve glycated haemoglobin (HbA1c) ≦7% or fasting plasma glucose (FPG) ≤7.7 mmol/L, was estimated using the Kaplan-Meier method.

REFERENCES

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Your Trustable Partner with Years of Experience and Confidence

- Effective HbA1c reduction
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HbA1c=glycated haemoglobin. NPH=neutral protamine Hagedorn insulin.


Prescribing information:
Presentation: 100 IU/ml Insulin glargine solution for injection. Indications For the treatment of adults, adolescents and children aged 2 years and above with diabetes mellitus. Dosage Once daily (at the same time every day), with adjusted individual dosage. Administration Subcutaneous injection. Lantus is NOT INTENDED FOR INTRAVENOUS USE since it could result in severe hypoglycaemia. Contraindications Hypersensitivity to insulin glargine or to any of the excipients. Precautions Lantus has not been studied in children below the age of 2 years. Elderly: progressive deterioration of renal function may lead to a steady decrease in insulin requirements. Renal impairment: insulin requirements may be diminished due to reduced insulin metabolism. Hepatic impairment: insulin requirement may be diminished due to reduced capacity for gluconeogenesis and reduced insulin bioavailability. Intermittent illness. Combination of Lantus with glitazones. Fertility, pregnancy and lactation Animal studies do not indicate direct harmful effects with respect to fertility and reproductive toxicity. The use of Lantus may be considered during pregnancy if clinical needed. It is unknown whether insulin glargine is secreted in human milk. Overdose Insulin overdose may lead to severe and sometimes long-term and life-threatening hypoglycaemia. Mild episodes of hypoglycaemia can usually be treated with oral carbohydrates. More severe episodes with coma, seizure or neurologic impairment may be treated with glucagon (intramuscular or subcutaneous) or concentrated glucose solution (intravenous). Interactions Effects enhanced by oral antidiabetics, AEDs, diazoxide, fibrates, fluoxetine, MAOs, pentoxifylline, propoxyphene, salicylates, sulfonamide antibiotics. Effects reduced by corticosteroids, danazol, diazoxide, duretics, glucagon, ibuprofen, oestrogens and progestogens, phenothiazine derivatives, somatropin, sympathomimetics, or thyroid hormones, atypical antipsychotics and protease inhibitors. Beta-blockers, clonidine, lithium or alcohol may either potentiate or weaken the effects of insulin. Pentamidine may cause hypoglycaemia, followed by hyperglycaemia. The signs of adrenergic counter-regulation may be reduced or absent under the influence of sympathomimetic medicinal products such as Beta-blockers, clonidine, guanethidine and reserpine. Undesirable effects Hypoglycaemia, Lipoatrophy, Injection site reactions, Lipoatrophy, Allergic reactions, Visual impairment, Retinopathy, Oedema, Dysesthesia, Myalgia. Storage: Before first use: Store in a refrigerator (0°C – 8°C). Do not freeze. After first use: Store below 30°C. Use within 28 days. Away from direct heat or light. Preparations Lantus SoloStar 5 x 3ml (3000U) pre-filled pens. Lantus Vial One 10ml (10000U) vial per box. Legal Classification: Part 1 Poison. Full prescribing information is available upon request. API-PK-GLA-17.03
Patients with type 2 diabetes should expect more after metformin

REALISE THE POTENTIAL

The only once-weekly treatment unifying superior efficacy and CV benefits

SUPERIOR GLYCAEMIC CONTROL
Up to 1.8% HbA1c reduction

SUPERIOR AND SUSTAINED WEIGHT LOSS
Up to 6.5kg weight reduction

PROVEN CV BENEFITS
26% CV risk reduction

For adults with type 2 diabetes with established ASCVD or indicators of high ASCVD risk 2019 ADA/ESKD consensus report recommends a GLP-1 RA therapy with proven CV benefit

Abbreviations: GLP-1 receptor agonist; ASCVD—atherosclerotic cardiovascular disease; GLP-1—glucagon-like peptide-1 receptor agonist.

1 GLP-1 RA therapy may change the background therapy for patients with type 2 diabetes, including metformin. Consequently, the use of GLP-1 RA therapy may lead to changes in HbA1c and body weight. A significant proportion of patients with type 2 diabetes receive metformin. The effect of GLP-1 RA therapy on HbA1c and body weight is therefore likely to be small compared to what can be achieved with metformin alone.

2 A 1.8% decrease in HbA1c is associated with a 34% reduction in the risk of composite endpoints including non-fatal myocardial infarction (MI), non-fatal stroke, or death from cardiovascular causes (HbA1c 6.3% vs 7.0% at 3 years).

3 HbA1c 7.0% vs 7.5% at 3 years

4 The CV risk reduction is consistent across all subgroups and in all analyses. It is based on the HbA1c reduction that was observed in individual studies. The magnitude of the CV benefit may vary from study to study due to differences in baseline risk, duration of treatment, patient characteristics, and other factors.

5 Once-weekly treatment. The benefit of once-weekly GLP-1 RA therapy over daily GLP-1 RA therapy has not been definitively demonstrated in clinical trials. The use of once-weekly GLP-1 RA therapy is based on the assumption that once-weekly GLP-1 RA therapy is as effective as daily GLP-1 RA therapy in reducing HbA1c and body weight.

6 When added to SOTC, which included oral antidiabetic treatment, insulin, antihypertensive, and lipid-lowering drugs (where applicable).

7 Other diabetes treatments refer to sitagliptin, dipeptidyl peptidase-IV inhibitor,agliptin and enablesin U.S. Target refers to American Diabetes Association target of HbA1c <7%.

8 In SUSTAIN 6, Ozempic reduced CV risk (CV death, nonfatal myocardial infarction [MI], or nonfatal stroke) versus placebo in patients with type 2 diabetes at high CV risk treated with standard of care. For further details, please refer to Ozempic<sup>®</sup> across SUSTAIN trials, which included placebo, DPP-4, SGLT-2, GLP-1 RA and basal insulin.

9 Oral antidiabetic treatment, insulin, and antihypertensive and lipid-lowering drugs (where applicable).
DECLARE A BETTER TOMORROW

Forxiga® - Your Cardio-renal Guardian

START TODAY BETTER OUTCOME TOMORROW

Largest CVOT of SGLT2i with the broadest population from multiple risk factors to established ASCVD¹

Reduction in cardioenal events observed in T2DM patients¹

17% CV death or hospitalisation for HF²

24% Cardiorenal composite endpoint³

47% Renal-specific composite endpoint³

Reassured safety profile of Forxiga®³

Forxiga® is the trademark of the AstraZeneca group of companies.

¹HF alone was a separate, nominally significant exploratory endpoint in the DECLARE trial—the primary endpoint composite of CV death/MI was driven by HF.
²Nominally significant, prepecified exploratory outcome.


Abridged Prescribing Information (API) FORXIGA® (dapagliflozin) Compositions: Dapagliflozin—propanediol monohydrate 5 mg or 10 mg. Therapeutic Indications: For the treatment of inadequately controlled type 2 diabetes mellitus in adults as an adjunct to diet and exercise within an algorithm when metformin is considered inappropriate due to intolerance, or in addition to other medications for the treatment of type 2 diabetes. Dosage and Administration: Recommended dose is 10 mg to be taken orally once daily at any time of day with or without food. Tablets are to be swallowed whole. In patients with severe hepatic impairment, a starting dose of 5 mg is recommended. Contraindications: Hypersensitivity to the active substance or to any of its excipients. Warnings and Precautions: Renal function, risk of volume depletion and/or hypertension should be taken into account in patients. Dosage of insulin and sulphonylureas (SU) may need to be readjusted to reduce the risk of hypoglycaemia. May add to the diuretic effect of thiazide and loop diuretics and may increase the risk of dehydration and hypoglycaemia. Use with caution in patients with increased risk of diabetic ketoacidosis on anti-hypertensive therapy with a history of hypoglycaemia. Elderly (≥ 65 year). Treatment should be temporarily interrupted when volume depleted; when treating pheochromocytoma or unexplained in patients who are hospitalized for major surgical procedures or acute serious medical illnesses, until laboratory values are normal. Should not be initiated in patients with a GFR < 40 mL/min/1.73 m² with type 1 diabetes; with hereditary problems of glycosylated insulin, the total lactase deficiency, or glucose-galactose malabsorption. Discontinue: If GFR is persistently below 60 mL/min; if suspected or diagnosed diabetic ketoacidosis, if fournier’s gangrene is suspected, if pregnancy is detected, while breastfeeding. Limit or no use in cirrhosis; suicide; pregnancy, and pseudohypoparathyroidism. Advance Reactions: Very common: hyperglycemia when used with SU or insulin. Common: vulvulogenital, balanitis and related genital infections, urinary tract infection, dizziness, rash, back pain, diplopia, polyuria, dysphagia, decreased nighttime renal clearance (during initial treatment), and increased haematuria. Uncommon: Fungal infection, volume depletion, thirst, constipation, dry mouth, nocturia, vulvulogenital and genital pruritus, increased blood osmolality (during initial treatment), increased blood loss, and decreased weight. Rare: diabetic ketoacidosis. Very rare: necrotising fasciitis of the perineum (fournier’s gangrene), angioedema. Not known: acute kidney injury. Drug interaction: Co-administration with rifampicin may reduce dapagliflozin systemic exposure; co-administration with metformin may increase dapagliflozin systemic exposure. Monitoring glycaemic control with 1.5 A1C assay is not recommended in patients taking SGLT2 inhibitors. Storage: Store below 20°C. Local prescribing information is available upon request. APLHK-FOR-0729

Please contact HKPatientSafety@astrazeneca.com for reporting of Individual Case Safety Report (ICSR) to AstraZeneca Hong Kong Limited.

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Acknowledgements