# 115 年度
# 台灣腎臟醫學會春季學術演講會
# 2026 Spring Academic Conference of Taiwan Society of Nephrology

# Program Book

## 2026/4/19 SUN

嘉義長庚紀念醫院

綜合醫學大樓 G 棟 B1 國際會議廳會議區

# 台灣腎臟醫學會
# 115 年度春季學術演講會

## 目錄

# 會員報到、教育積分注意事項

## 報到：

**會員及準會員務必攜帶身份證親自刷卡報到，才給予積分認定。**

## 醫師會員及準醫師會員報到

時間：115 年 4 月 19 日(星期日)上午 8:30 至下午 1:00

地點：嘉義長庚紀念醫院綜合醫學大樓 G 棟 B1 國際會議廳會議區報到處

**積分認定**

✓腎臟醫學會積分：A類15分

✓內科醫學會積分：B類4分

## 透析護理人員及腎臟照護衛教師報到及刷退

- 已報名且完成繳費者
- 請務必攜帶身分證親自報到-刷到及刷退，才給予積分

**【報到及刷退時間】**

| | 刷到時間 | 刷退時間 |
|---|---|---|
| 4 月 19 日(星期日) | 上午 8 時 30 分至上午 10 時 00 分 | 下午 2 時 45 分至 3 時 15 分 |
| 地點：嘉義長庚紀念醫院綜合醫學大樓 G 棟 B1 國際會議廳會議區報到處 | | |

**積分認定：**

✓透析繼續教育積分：1.5 次

✓慢性腎臟病繼續教育積分：15 小時

✓衛福部護產積分分申請中

## 參展廠商攤位展示區

時間：115 年 4 月 19 日(星期日)

地點：嘉義長庚紀念醫院綜合醫學大樓 G 棟 B1 國際會議廳會議區廣場

## 午餐資訊

時間：115 年 4 月 19 日(星期日)中午 12:30 至下午 1:20

【已登記用餐之會員請於報到時領取餐券】

地點：嘉義長庚紀念醫院綜合醫學大樓 G 棟 B1 國際會議廳用餐區

領取地點：各午餐演講會議室外領取【憑券領取餐盒】

用餐區：第二會議室、第三會議室、廣場用餐區 (第一、第二國際會議廳內無法飲食)

## 接駁車資訊

- 本次會議學會備有中型巴士來往嘉義高鐵站至嘉義長庚紀念醫院

嘉義高鐵上車地點：高鐵 2 號出口大客車上車處
嘉義長庚上車地點：綜合大樓 B1 乘車處

***實際發車時間依照實際交通狀況而定，時刻表僅供參考**

| 115年度台灣腎臟醫學會春季學術演講會 | | | |
|---|---|---|---|
| 乘車地點：嘉義高鐵站2號出口 大客車上車處 | | | |
| 發車時間 | | | |
| 08:00 | 08:30 | 09:00 | 09:30 |
| 10:00 | 10:30 | 11:00 | 11:30 |
| 12:00 | 13:00 | 13:30 | 14:00 |
| 14:30 | 15:00 | 15:30 | |

| 115年度台灣腎臟醫學會春季學術演講會 | | | |
|---|---|---|---|
| 乘車地點：嘉義長庚醫院 綜合大樓 B1乘車處 | | | |
| 發車時間 | | | |
| 08:15 | 08:45 | 09:15 | 09:45 |
| 10:15 | 10:45 | 11:15 | 11:45 |
| 12:45 | 13:15 | 13:45 | 14:15 |
| 14:45 | 15:15 | 15:45 | |

# 台灣腎臟醫學會
# 115 年度春季學術演講會
# 節目議程表

時間：115 年 4 月 19 日(星期日)
地點：嘉義長庚紀念醫院 綜合醫學大樓 G 棟 B1 國際會議區

| 日期 | | 115 年 4 月 19 日(星期日) | | | | |
|---|---|---|---|---|---|---|
| 時間＼會場 | | 第一國際會議廳 | 第二國際會議廳 | 第三會議室 | 第二會議室 | 廣場 |
| 上午 | 08:30 \| 13:00 | 報到 | | | | |
| | 9:00 9:10 | 開幕致詞 | | | | 廠商展示區 |
| | 09:10 \| 10:40 | 【專題演講 1】<br>2026 年美國最新飲食指南 | 【專題演講 4】<br>KDIGO guideline—Anemia, ADPKD | 【專題演講 7】<br>Dialysis Care Strategies for Resilient Healthcare | 【病例報告 1】 | |
| | 10:50 \| 12:20 | 【專題演講 2】<br>從腎到心到代謝：CKM 跨科合作新時代 | 【專題演講 5】<br>KDIGO guideline—2025 IgAN 與 2024 AAV | 【專題演講 8】<br>掌握生命線：血液透析通路照護與優質穿刺技術之臨床指引 | 【病例報告 2】 | |
| 中午 | 12:30 \| 13:20 | Lunch Symposium 1<br>百靈佳 | Lunch Symposium 2<br>太暘生技 | Lunch Symposium 3<br>葛蘭素史克 | Lunch Symposium 4<br>阿斯特捷利康 | |
| 下午 | 13:30 \| 15:00 | 【專題演講 3】<br>CKLM 時代：CKD 合併肥胖與 MASLD/MASH 的整合照護 | 【專題演講 6】<br>基因醫學於腎臟學之最新進展與應用 | 【專題演講 9】<br>肥胖症與慢性腎臟病之整合照護與精準治療策略 | | |

## 【第一國際會議廳】

【開幕致詞】
**09:00-09:10**
陳金順 理事長
台灣腎臟醫學會

## 【專題演講 1】
## The 2026 U.S. Dietary Guidelines
## 2026 年美國最新飲食指南

*Chair(s):* 林威宏 醫師

**09:10 – 09:15**
Opening Remarks
林威宏 醫師
成大醫院 腎臟科

**09:15 – 09:40**
The 2026 U.S. Dietary Guidelines: A Dietitian's Perspective
2026 年美國最新飲食指南：營養師觀點
吳紅蓮 營養師
成大醫院 營養部

**09:40 – 10:05**
The 2026 U.S. Dietary Guidelines: A Physician's Perspective
2026 年美國最新飲食指南：醫師觀點
邱鼎育 醫師
高雄長庚紀念醫院 腎臟科

**10:05 – 10:30**
Implications for Chronic Kidney Disease: What Should We Adopt and What Should We Avoid?
對慢性腎臟病的啟示：我們該採納什麼，又該避免什麼？
吳秉勳 醫師
高雄醫學大學附設醫院 腎臟科

**10:30 – 10:35**
Q&A

**10:35 – 10:40**
Closing Remarks: What Should Nephrologists Change on Monday Morning?
腎臟科醫師下週一早上該改變什麼？
林威宏 醫師
成大醫院 腎臟科

## 【第一國際會議廳】

## 【專題演講 2】
## From Kidney to Heart to Metabolism: A New Era of CKM Cross-Specialty Collaboration
## 從腎到心到代謝：CKM 跨科合作新時代

*Chair(s):* 林俊良 醫師
劉冠宏 醫師

**10:50 – 10:55**
Opening: CKM Syndrome — Breaking Down Specialty Barriers
開場：CKM Syndrome — 打破科別的圍牆
林俊良 醫師
嘉義長庚紀念醫院 腎臟科

主持：劉冠宏 醫師
**10:55 – 11:20**
The CKM Era: Redefining the Nephrologist's Role Beyond the Kidney
CKM 時代：腎臟科醫師的新角色定位
蔡尚峰 醫師
台中榮民總醫院 腎臟科

主持：林俊良 醫師
**11:20 – 11:45**
Dear Nephrologists: What Your Cardiologist Wishes You Knew
Dear 腎臟科同事：這些心臟的事，我希望你知道
陳柏偉 醫師
成大醫院 心臟科

主持：劉冠宏 醫師
**11:45 – 12:10**
Upstream of the Metabolic Storm: Key Messages from Endocrinology to Nephrologists
代謝風暴的上游：內分泌科想告訴腎臟科的關鍵訊息
沈峰志 醫師
高雄長庚紀念醫院 新陳代謝科

**12:10 – 12:20**
Q&A

## 【第一國際會議廳】

### 【專題演講 3】
### CKD in the CKLM Era: Integrating Management of Obesity and MASLD/MASH
### CKLM 時代：CKD 合併肥胖與 MASLD/MASH 的整合照護

*Chair(s):* 邱炳芳 醫師
張家築 醫師
曾進忠 醫師

**13:30 – 13:35**
Opening
陳金順 理事長

主持：邱炳芳 醫師
**13:35 – 14:00**
Managing Obesity in CKD: Patient Stratification and Goal-Directed Weight Loss
林思涵 醫師
彰化基督教醫院 新陳代謝科

主持：張家築 醫師
**14:00 – 14:25**
Modern Metabolic Therapy for CKD: GLP-1–Based Agents, Dual Incretin Therapy, and Renoprotection
曾士婷 醫師
光田綜合醫院 新陳代謝科

主持：曾進忠 醫師
**14:25 – 14:50**
Implementing MASLD/MASH Care in CKD: Screening, Fibrosis Risk Triage, and When to Refer
簡世杰 醫師
成大醫院 胃腸科

**14:50 – 15:00**
QA & Closing

## 【第二國際會議廳】

## 【專題演講 4】
## KDIGO guideline—Anemia, ADPKD
## KDIGO 臨床指引更新：Anemia, ADPKD

*Chair(s):* 方德昭 醫師
張明揚 醫師

主持：方德昭 醫師
**09:10 – 09:50**
KDIGO 2025 Clinical Practice Guideline for Anemia update
KDIGO 2025 貧血臨床實務指引更新
黎思源 醫師
台北榮民總醫院 腎臟科

主持：張明揚 醫師
**09:50 – 10:30**
KDIGO 2025 Clinical Practice Guideline for ADPKD
KDIGO 2025 多囊腎臨床診療指引
高芷華 醫師
衛生福利部雙和醫院 腎臟科

**10:30 – 10:40**
Panel Discussion

## 【第二國際會議廳】

## 【專題演講 5】
## Update of KDIGO Guidelines: 2025 IgA nephropathy and 2024 ANCA-associated vasculitis
## KDIGO 臨床指引更新：2025 IgAN 與 2024 AAV

*Chair(s):* 吳家兆 醫師
楊如燁 醫師
賴俊夫 醫師

**10:50 – 10:55**
Opening
吳家兆 醫師
三軍總醫院 腎臟科

主持：吳家兆 醫師
**10:55 – 11:20**
KDIGO 2025 IgA nephropathy/IgA vasculitis guideline
KDIGO 2025 IgA 腎病與 IgA 血管炎臨床指引
賴台軒 醫師
台大醫院腎臟科

主持：楊如燁 醫師
**11:20 – 11:45**
KDIGO 2024 ANCA-associated vasculitis guideline：Clinical challenges in Taiwan
KDIGO 2024 ANCA 相關血管炎臨床指引與台灣實務的挑戰
潘思宇 醫師
台大醫院 腎臟科

主持：賴俊夫 醫師
**11:45 – 12:10**
TCR commentary on the KDIGO IgA and AAV guidelines
風濕病醫學會意見分享
李克仁 醫師
中華民國風濕病醫學會理事長

**12:10 – 12:20**
Panel Discussion

## 【第二國際會議廳】

## 【專題演講 6】
## Precision Medicine in Kidney Disease
## 腎臟疾病的精準醫療

*Chair(s):* 林志慶 醫師
游棟閔 醫師

**13:30–14:00**
Precision Medicine in Inherited Kidney Diseases: From Gene to Therapy
遺傳性腎臟病的精準醫學：從基因到治療
楊豐榮 醫師
台大醫院 基因醫學部

**14:00–14:30**
Utilizing TPMI Genomic Data for Personalized Prediction Models of Kidney Diseases
運用 TPMI 基因數據建立腎臟病之個人化預測模型
陳一銘 醫師
台中榮民總醫院 醫學研究部轉譯醫學科

**14:30–15:00**
Perspectives on TMA: Precision Medicine and Long-term Management of aHUS
透視血栓性微血管病變：aHUS 的精準醫療與長期預後管理
蔡宜蓉 醫師
台大醫院 小兒科

## 【第三會議室】

## 【專題演講 7】
## Dialysis Care Strategies for Resilient Healthcare
## 打造韌性醫療的透析照護策略

*Chair(s):* 吳家兆 醫師
賴台軒 醫師
林素如 醫師

**09:10 – 09:15**
Opening
「韌性醫療」的透析照護策略
陳金順 理事長

主持：吳家兆 醫師
**09:15 – 09:40**
A Strategic Shift Toward Home-Based Therapies and Value-Based Outcomes
邁向居家治療與價值導向成效的策略轉型
鄭本忠 醫師
高雄長庚醫院 腎臟科

主持：賴台軒 醫師
**09:40 – 10:05**
Digital PD: Innovation for Better Living
數位化腹膜透析：以創新提升生活品質
鍾牧圻 醫師
台中榮民總醫院 腎臟科

主持：林素如 醫師
**10:05 – 10:30**
PD Beyond the Hospital: Community Clinics as Both Hubs and Spokes
走出醫院的 PD：以社區診所建立樞紐—輻射式照護模式
王介立 醫師
台北市柏安診所

**10:30 – 10:40**
QA & Closing

## 【第三會議室】

## 【專題演講 8】
## Mastering the Life-Line: A Clinical Guide to Dialysis Access Care and Cannulation Excellence
## 掌握生命線：血液透析通路照護與優質穿刺技術之臨床指引

*Chair(s):* 林承叡 醫師

**10:50 – 11:20**
Session I: Decoding the Life-Line: From Anatomical Foundation to Functional Evaluation
解碼生命線—從解剖基礎到功能評估
陳盈穎 醫師
台北馬偕醫院 腎臟科

**11:20 – 11:50**
Session II: The Sonographic Sentinel: Ultrasound's Role in the Proactive Management of AVF Complications
超音波守門人：超音波在動靜脈瘻管（AVF）併發症前瞻性管理中的角色
吳重寬 醫師
新光紀念醫院 腎臟科

**11:50 – 12:20**
Session III: Beyond the Surface: Breakthrough Strategies for Challenging Cannulation
突破表層：困難穿刺的創新策略
韓雲楷 醫師
輝德診所

## 【第三會議室】

## 【專題演講 9】
## Integrated Care and Precision Therapeutic Strategies for Obesity and Chronic Kidney Disease
## 肥胖症與慢性腎臟病之整合照護與精準治療策略

*Chair(s):* 蔡宜純 醫師

**13:30 – 13:35**
簡介

**13:35 – 14:00**
Obesity-Associated Metabolic Imbalance and Kidney Injury
肥胖相關代謝失衡與腎臟病變
張以承 醫師
台灣大學醫學院附設醫院 新陳代謝科

**14:00 – 14:25**
Obesity × Kidney Disease: Emerging Guidelines for Weight Management from CKD to Dialysis — From Weight Reduction to Cardiorenal Protection
肥胖 × 腎臟病：從 CKD 到透析的體重管理新指引—從『減重』出發到『心腎保護』
林威宏 醫師
成功大學醫學院附設醫院 腎臟內科

**14:25 – 14:50**
Bridging the Gap: How Metabolic-Bariatric Surgeons and Nephrologists Can Co-manage Obesity-Driven Chronic Kidney Disease
重塑照護模式：代謝減重外科與腎臟科攜手管理肥胖驅動之慢性腎臟病
張博智 醫師
高雄醫學大學附設中和紀念醫院 胸腔外科

**14:50 – 15:00**
Q&A

# Lunch Symposium

## 【Lunch Symposium 1】

會場：第一國際會議廳
時間：中午 12:30-13:20

主持人：方德昭 醫師

主講人：林威宏 醫師
成大醫院 腎臟科

講　題：Beyond RAAS: The New Standard of Care for CKD

贊助單位：台灣百靈佳殷格翰股份有限公司

## 【Lunch Symposium 2】

會場：第二國際會議廳
時間：中午 12:30-13:20

主持人：林俊良 醫師

主講人：塗昆樺 醫師
林口長庚紀念醫院 腎臟科

講　題：Navigating from MGUS to MGRS: The Critical Role of Serum Free Light Chains in Renal Involvement and Clinical Decision Making
從 MGUS 到 MGRS：血清游離輕鏈在腎臟損傷中的臨床價值與決策關鍵

贊助單位：太暘生物科技股份有限公司

## 【Lunch Symposium 3】

會場：第三會議室
時間：中午 12:30-13:20

主持人：張淀榮 醫師

主講人：郭弘典 醫師
高雄醫學大學附設醫院 腎臟科

講　題：糖心腎患者的關鍵感染風險：帶狀疱疹與 RSV 防護策略

贊助單位：荷商葛蘭素史克藥廠股份有限公司台灣分公司

## 【Lunch Symposium 4】

會場：第二會議室
時間：中午 12:30-13:20

主持人：陳金順 醫師

主講人：楊智超 醫師
高雄長庚紀念醫院 腎臟科

講　題：Precision Nephrology: Resolve the Safety-Prognosis Conflict to Sustain Cardiorenal Benefit
護腎不煞車—強化醫病信心，鎖定長期腎臟保護效益

贊助單位：台灣阿斯特捷利康股份有限公司

# 病例報告

時間：09:15~11:30
地點：嘉義長庚紀念醫院 綜合醫學大樓 G 棟 B1 國際會議區第二會議室

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【病例討論 1】 主持人：王憲奕 醫師

09:15 － 09:30 1. 三酸甘油酯–葡萄糖指數在新開始腹膜透析患者中的預後意義
Prognostic Significance of the Triglyceride–Glucose Index in Patients Starting Peritoneal Dialysis
張禎祐[1] 許高鳴[2] 謝堯棚[2] 張玉君[3] 蔡詩梅[4] 邱炳芳[2]
[1]彰化基督教兒童醫院兒童腎臟科 [2]彰化基督教醫院腎臟內科 [3]彰化基督教醫院大數據中心 [4]彰化基督教醫院護理部

09:30 － 09:45 2. 組織血栓溶解劑(rTPA)引發之動脈粥狀硬化栓塞性腎病模仿非典型溶血性尿毒症候群之病例報告
Atheroembolic Renal Disease (AERD) Mimicking Atypical Hhemolytic Uremic Syndrome (aHUS) Following rTPA administration: A Case Report
羅奕涵[1] 陳建良[1]
[1] 高雄榮民總醫院腎臟科

09:45 － 10:00 3. 嚴重高血鈣、急性腎損傷及肝膽侵犯為表現之 IgG4 相關疾病
IgG4-Related Disease Presenting with Severe Hypercalcemia, Acute Kidney Injury, and Hepatobiliary Involvement
劉曉盈[1] 蔡尚峰[1] 張振義[2] 彭彥鈞[3]
[1]台中榮民總醫院 內科部 腎臟科 [2]台中榮民總醫院 家庭醫學部 家庭醫學科 [3]台中榮民總醫院 內科部 胃腸肝膽科

10:00 － 10:15 4. 一位診斷高血壓性血栓性微血管病變的年輕男性
Malignant Hypertension Presenting as Thrombotic Microangiopathy: A Biopsy-Proven Case in a Young Adult with CD61-Positive Platelet Aggregation
林鈺雯[1] 王憲奕[1] 簡志強[1]
[1]奇美醫院腎臟內科

10:15 – 10:30　5. 環孢素誘發之可逆性後腦病變症候群於原發性膜性腎病變患者之病例報告
Cyclosporine-induced Posterior Reversible Encephalopathy Syndrome in a Patient With Primary Membranous Nephropathy
沈家銘[1] 顏正杰[1,2,3] 蘇勤雅[1,2]
嘉義基督教醫院 [1]內科部 [2]腎臟內科 [3]敏惠醫護管理專科學校長期照顧與健康促進管理科

【病例討論 2】　主持人：陳建良 醫師

10:30 – 10:45　6. 局部檸檬酸抗凝法使用於患有嚴重肺炎鏈球菌相關溶血性尿毒綜合症並接受接受連續性腎臟替代治療之個案報告
Regional Citrate Anticoagulation In A Patient With Severe Streptococcus Pneumoniae-Associated Hemolytic Uremic Syndrome Requiring Continuous Kidney Replacement Therapy
張婷媛[1] 蔡宜蓉[2] 莊國燦[3] 黃厚瑄[4]
[1]台大兒童醫院小兒腎臟科 [2]高雄醫學大學小兒腎臟科

10:45 – 11:00　7. 積水背後的真相：以難治性漿膜腔積液為表現之移植後原發性積液淋巴瘤
More Than Just Fluid Overload: Primary Effusion Lymphoma Presenting as Refractory Effusion in a Kidney Transplant Recipient
林唯尹[1] 陳泰迪[2] 許翔皓[1] 田亞中[1] 方基存[1] 楊智偉[1] 塗昆樺[1]
[1]林口長庚醫院腎臟科 [2]林口長庚醫院病理科

11:00 – 11:15　8. 肺移植後受贈者發生原生腎 BK 病毒性腎病變：一例罕見病例報告與文獻回顧
Native Kidney BK Virus Nephropathy After Lung Transplantation: A Rare Case Report and Literature Review
郭怡珩[1] 陳泰迪[2] 許翔皓[1] 田亞中[1] 方基存[1] 楊智偉[1] 塗昆樺[1]
[1]林口長庚醫院腎臟科 [2]林口長庚醫院病理科

11:15 – 11:30　9. 腎小球毛細血管內脂蛋白血栓：一名十四歲大女童的脂蛋白腎小球病早期表現
Lipoprotein Thrombi Within The Glomerular Capillaries: An Early Presentation of Lipoprotein Glomerulopathy In A 14-Year-Old Girl
邱偉倫[1] 蔡政道[1] 陳冬英[2] 曾敏華[3]
[1]馬偕兒童醫院兒科部 [2]馬偕紀念醫院病理科 [3]林口長庚紀念醫院兒科部

# 專題演講
# 及
# Lunch Symposium
# 摘要

# 專題演講 1

## The 2026 U.S. Dietary Guidelines: A Dietitian's Perspective

## 2026 年美國最新飲食指南：營養師觀點

吳紅蓮 營養師
成大醫院 營養部

美國飲食指南長期為全球營養論述的重要參考依據，強化蔬菜與真食物攝取的重要性，減少加工品的攝取，這些觀點是值得倡議的，但美國新版飲食指南是舊版的倒金字塔，在蛋白質與動物性食品的建議上出現明顯調整，同時是有爭議性。在營養師的觀點:1.長久以來臨床研究顯示紅肉與加工肉品對健康的危害，紅肉屬於高磷的食物及含高甲硫胺酸易產生 IS 與 PS 造成腎臟與心血管及其他內臟器官的危害，加工肉品含高鹽高磷高鉀的食品添物及含高熱量、高糖(尤其是果糖)、高鹽、高油、高飽和脂肪、低營養素、低纖維易造成高血壓、高血脂、高血糖等而造成肥胖、心臟病、血管疾病、糖尿病、肝臟疾病如脂肪肝與肝硬化、腎臟病等，應明確限制攝取的份量，

尤其不可與蔬果並列。這幾年慢性病的防治與治療都在提倡以植物蛋白質取代部分的動物性蛋白質。2.新指引建議多攝取全脂乳品，但又一直強低飽和脂肪的攝取，不超過總熱量的 10% ，兩個理念是完全相違背，因全脂奶含高油脂尤其含高飽和脂肪，飽和脂肪的攝取會超過 10% ，應限制分量對有心血管疾病病人或高血脂的病人或風險的病人應限制全脂乳品的攝取改低脂奶，尤其慢性腎臟病的病人或透析病人更不應建議攝取。3.蔬菜與水果富含纖維及各種營養素如維生素與礦物質，尤其是蔬菜每日應至少 3 份( 1.5 碗)，對腸道黏膜的保護及益生菌的滋養及排便都是有利的，水果因有糖份且纖維含量遠低於蔬菜，其建議量(2 份)應低於蔬菜，尤其對糖尿病病人需限量，對腎臟病後期病人或透析病人尤其血液透析病人更需加強注意，圖示的酪梨與香蕉都是極高鉀的水果。 4.澱粉與全穀類是人體主要的熱能來源也是增加蛋白質利用率的食物，全穀類含高纖維及高 B 群建議應以品質為導向，而非比例，應依個人的活動、體重、疾病、工作量來建議每個人適當的攝取量，不是一味的建議減少攝取，健康飲食的熱能不足反而進食更多不健康的食物。5.健康油脂來自原型食物如紅肉、家禽、全蛋、富含Ω-3 的海產、全脂奶、堅果、種子、橄欖油:有些理念是錯的，動物脂肪含全蛋的蛋黃及氫化植物是含高量的飽和脂肪，只有植物性的油脂除棕梠油及椰子油外才是富含不飽和脂肪，故烹調用油盡量採用植物油，動物肉類應建議限量食用以減少飽和脂肪攝取。 6.新指引建議減少攝取高度加工食品，強化環境永續及健康標示是一件好事，我們都知高度加工品對身體的傷害，環境永續低碳飲食需減少動物性食物的攝取增加植物性食物的攝取，健康標示可讓民眾選擇適合自己的食品，但須教育民眾食品標示各項的內涵與意義，以上期望為民眾與病人帶來更正確的營養知識以維護健康的身體。

# 專題演講 1

The 2026 U.S. Dietary Guidelines: A Physician's Perspective
2026 年美國最新飲食指南：醫師觀點

邱鼎育 醫師
高雄長庚紀念醫院 腎臟科

2026 年初，美國政府發布了最新的 《2025–2030 年美國居民膳食指南》 (Dietary Guidelines for Americans)，這份指南被視為數十年來聯邦營養政策最重大的「重啟」。新指南由美國衛生及公共服務部(HHS)主導，核心口號為「吃真正的食物」(Eat Real Food)。我們將從多層面向探討其可能的衝擊：各專科疾病預防策略與病理生化指標，對不同年齡及族群的影響。希望能夠幫助個人、家庭、社區、國家思考建立最合適自己的精準膳食指南。

# 專題演講 1

## Implications for Chronic Kidney Disease: What Should We Adopt and What Should We Avoid?

對慢性腎臟病的啟示：我們該採納什麼，又該避免什麼？

吳秉勳 醫師
高雄醫學大學附設醫院 腎臟科

近年來，飲食指南的更新顯示公共衛生營養策略正從傳統的營養素導向逐漸轉向原型食物的概念。然而，這些主要針對一般健康族群設計的建議，未必完全適用於慢性腎臟病患者。2026 年美國飲食指南(Dietary Guidelines for Americans, DGA)強調高蛋白攝取、限制添加糖、降低超加工食品以及適量鈉攝取等原則，但若直接套用於 CKD 族群，可能在某些情境下產生潛在風險，例如高蛋白飲食可能增加腎絲球過濾壓力並加速腎功能惡化。在糖與超加工食品方面，DGA 與腎臟病營養指引(KDIGO/KDOQI)具有高度共識。大量研究顯示，含糖飲料與高糖飲食可透過促進胰島素阻抗、慢性發炎及糖尿病風險增加，進而加速腎臟病進展；而超加工食品因含有高鈉、添加磷與多種食品添加物，亦與 CKD 進展及死亡率上升相關。因此，減少添加糖並避免超加工食品，是對一般族群與 CKD 患者皆適用的核心策略。蛋白質攝取則是最具爭議的部分。DGA 建議成人蛋白質攝取量約為 1.2 – 1.6 g/kg/day，但 KDIGO 指南建議非透析 CKD G3 – G5 患者應控制於約 0.8 g/kg/day，以降低腎絲球高過濾與蛋白尿惡化風險。對於透析患者或營養不良族群，則可能需要較高蛋白攝取。因此，蛋白質管理需依疾病階段進行分層處方，而非採取單一標準。此外，蛋白質來源亦十分重要。植物性蛋白通常伴隨較低的淨酸負荷與較少的尿毒素前驅物，並可能透過腸腎軸（gut – kidney axis）改善腸道菌相與降低尿毒素生成，因此相較於紅肉，植物性蛋白與魚類更有利於腎臟與心血管健康。在電解質管理方面，鈉攝取過量仍是全球性問題。KDIGO 建議 CKD 患者每日鈉攝取應低於 2 g（約 5 g 食鹽），以降低血壓與心血管風險。然而，對於鉀的管理，近年研究指出全面性低鉀飲食缺乏充分證據支持。植物來源的鉀吸收率較低，且富含纖維與鹼性成分，對腎臟與代謝健康可能具有保護作用。因此，現代策略逐漸從全面限制轉向監測血鉀並個別化調整。

綜合而言，2026 年飲食指南為一般族群提供了重要的健康飲食框架，但在 CKD 族群中需經由 KDIGO 與 KDOQI 指引進行個別化調整。臨床實務中應採取三項原則：採納原型食物與減少超加工食品、調整鈉、鉀與磷的攝取，以及避免不適合 CKD 患者的高蛋白飲食策略。透過個人化醫療營養治療，才能在維持營養品質的同時，降低 CKD 進展與心血管併發症風險。

# 專題演講 2

## The CKM Era: Redefining the Nephrologist's Role Beyond the Kidney

## CKM 時代：腎臟科醫師的新角色定位

蔡尚峰 醫師
台中榮民總醫院 腎臟科

心臟－腎臟－代謝（cardio–kidney–metabolic, CKM）疾病概念的提出，重新塑造了慢性疾病的整體照護架構，強調心血管疾病、慢性腎臟病與代謝異常（如糖尿病、肥胖與胰島素阻抗）之間高度交互且相互促進的病理生理關係。這些疾病不再被視為各自獨立的器官疾病，而是構成一個連續且相互影響的疾病光譜。腎臟在此系統中扮演關鍵節點，腎功能受損不僅是多重代謝與心血管風險因子的結果，同時也會進一步加劇全身性發炎、氧化壓力、血流動力學改變與器官損傷，形成惡性循環。因此，在 CKM 時代，腎臟科醫師的角色正逐漸由傳統的「腎衰竭照護者」轉變為「慢性疾病整合管理者」。

隨著大量臨床試驗與真實世界研究的累積，包括 SGLT2 抑制劑、非類固醇型礦物皮質素受體拮抗劑、GLP-1 受體促效劑等疾病修飾治療，已證實能同時改善腎臟、心血管與代謝相關結局，使 CKM 疾病的治療策略出現重大轉變。這些療法不僅能延緩腎功能惡化，也能降低心衰竭、心血管事件及死亡風險，進一步突顯腎臟科醫師在整體 CKM 治療中的核心地位。此外，腎臟科醫師在慢性腎臟病的早期辨識、風險分層、蛋白尿與腎功能監測，以及多重藥物整合治療方面具有豐富經驗，使其成為推動 CKM 整合照護的重要橋樑。

因此，在 CKM 醫學逐漸成為慢性疾病管理核心架構的背景下，腎臟科醫師需要重新定位自身角色，從過去以透析與末期腎病為中心的照護模式，轉向涵蓋疾病早期預防、風險評估與跨專科整合治療的全病程管理。透過與心臟科、內分泌科及基層醫療的合作，腎臟科醫師將在 CKM 整合照護體系中扮演關鍵樞紐角色，促進跨器官疾病的整體管理，並最終改善患者的長期預後與生活品質。

# 專題演講 2

## Dear Nephrologists: What Your Cardiologist Wishes You Knew

## Dear 腎臟科同事：這些心臟的事，我希望你知道

陳柏偉 醫師
成大醫院 心臟科

The emerging concept of Cardio-Kidney-Metabolic (CKM) syndrome represents a paradigm shift in chronic disease management, highlighting the interconnected pathophysiology of cardiovascular disease, chronic kidney disease, and metabolic disorders. Rather than viewing these conditions in isolation, CKM emphasizes a shared biological network driven by inflammation, oxidative stress, endothelial dysfunction, and neurohormonal activation. Within this framework, vascular calcification has emerged as a critical yet often underrecognized mediator linking kidney dysfunction to adverse cardiovascular outcomes.

Vascular calcification, particularly in patients with chronic kidney disease and diabetes, is no longer considered a passive degenerative process but an actively regulated phenomenon resembling osteogenesis. Dysregulation of mineral metabolism—including phosphate retention, fibroblast growth factor-23 (FGF-23) elevation, and reduced Klotho expression—plays a central role in promoting vascular smooth muscle cell transformation and arterial stiffness. Clinically, this translates into increased pulse wave velocity, systolic hypertension, left ventricular hypertrophy, and ultimately heightened risks of heart failure and cardiovascular mortality.

Recent advances in pharmacotherapy have opened new opportunities to modify the CKM trajectory. Sodium-glucose cotransporter-2 (SGLT2) inhibitors have demonstrated robust benefits across cardiovascular, renal, and metabolic domains, including reductions in heart failure hospitalization and slowing of kidney disease progression, independent of glycemic control. Similarly, nonsteroidal mineralocorticoid receptor antagonists such as finerenone have shown efficacy in reducing both renal and cardiovascular events, likely through anti-inflammatory and antifibrotic mechanisms. Emerging lipid-lowering agents, including PCSK9 inhibitors and novel therapies targeting lipoprotein(a), further contribute to residual cardiovascular risk reduction in high-risk populations.

Importantly, these therapies may also exert indirect effects on vascular calcification through modulation of systemic inflammation, oxidative stress, and mineral metabolism. However, direct therapeutic strategies targeting calcification pathways remain an unmet need. Ongoing research into inhibitors of phosphate transport, modulators of calcification signaling pathways, and imaging-based risk stratification tools may help refine future interventions.

In the CKM era, clinical practice must evolve toward integrated, cross-specialty collaboration. Nephrologists, cardiologists, and endocrinologists must work together to identify high-risk patients early, implement guideline-directed medical therapy, and address shared pathophysiological mechanisms such as vascular calcification. By bridging traditional specialty boundaries and incorporating novel therapeutics, we can move toward a more holistic and effective approach to reducing the global burden of CKM syndrome.

# 專題演講 2

## Upstream of the Metabolic Storm: Key Messages from Endocrinology to Nephrologists

代謝風暴的上游：內分泌科想告訴腎臟科的關鍵訊息

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高雄長庚紀念醫院 新陳代謝科

The emergence of Cardiovascular-Kidney-Metabolic (CKM) syndrome marks a pivotal shift toward a holistic understanding of chronic disease. From the perspective of an endocrinologist, the “metabolic storm” that eventually ravages the heart and kidneys does not begin with an elevated creatinine or a cardiac event; it originates far upstream in the dysfunctional crosstalk between adiposopathy (sick fat) and insulin resistance. The cornerstone of primary prevention lies in recognizing adipose tissue as a volatile endocrine organ. When lipid storage capacity is overwhelmed, ectopic fat deposition triggers a cascade of lipotoxicity, chronic low-grade inflammation, and systemic oxidative stress. Effective intervention requires a paradigm shift from “glucocentrism” to “organ-protective metabolic remodeling.” We advocate for the early deployment of SGLT2 inhibitors and GLP-1 receptor agonists—not merely for glycemic control, but for their pleiotropic ability to reduce glomerular hyperfiltration, dampen systemic inflammation, and restore metabolic flexibility. By integrating these pharmacological advancements with precision lifestyle prescriptions aimed at weight loss and “metabolic resetting,” we can intervene during CKM Stages 1 and 2.Ultimately, the goal of the endocrinologist is to act as the gatekeeper of the CKM axis. By stabilizing the metabolic foundation and mitigating upstream drivers, we can effectively quench the fires of the metabolic storm, preventing the progression to end-stage renal disease and heart failure, and ensuring long-term multi-organ resilience.

# 專題演講 3

## Managing Obesity in CKD: Patient Stratification and Goal-Directed Weight Loss

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肥胖不僅是慢性腎臟病（CKD）常見共病，更可透過腎絲球高過濾、發炎、脂毒性與肥胖相關腎絲球病變，直接促進蛋白尿與腎功能惡化。 近年 CKD 肥胖照護已走向病人分層與目標導向減重，需依 eGFR、蛋白尿、糖尿病、心血管風險與肌少症風險擬定策略。 目前多數證據支持，體重下降 5–10% 即可帶來臨床效益，若達 10% 以上，對代謝與腎臟相關指標改善更明顯。 本演講將聚焦肥胖致腎機轉、臨床分層評估與目標設定，並分享生活型態、藥物與代謝治療在台灣腎臟科實務上的整合應用。

# 專題演講 3

## Modern Metabolic Therapy for CKD: GLP-1–Based Agents, Dual Incretin Therapy, and Renoprotection

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糖尿病與慢性腎臟病（CKD）的交集構成全球最具破壞性的疾病負擔之一。傳統標準照護以 RAAS 抑制劑為主，但患者進展至末期腎病的殘餘風險仍然極高。近年來，腸泌素（Incretin）基礎療法的崛起帶來了心血管-腎臟-代謝（CKM）症候群整合治療的典範轉移。

GLP-1 受體激動劑透過多重抗發炎、抗氧化應激機制及促進尿鈉排泄，展現直接的腎臟保護作用，並與 SGLT2 抑制劑在血流動力學上形成互補。GIP/GLP-1 雙重受體激動劑更進一步透過「脂質重分配」效應，減少腎臟周邊異位脂肪堆積，消除「脂肪腎」病理，改善腎臟缺氧狀態。

FLOW 試驗（n=3,533）證實 Semaglutide 1.0 mg 在高風險糖尿病腎病患者中，使主要複合腎臟終點風險降低 24%（HR 0.76），eGFR 斜率差異達 1.16 mL/min/1.73m$^2$/year，UACR 下降 40%，全死因死亡率降低 20%（NNT=39）。SELECT 試驗進一步於無糖尿病肥胖族群中證實，Semaglutide 2.4 mg 使複合腎臟終點風險降低 22%（HR 0.78），確認腎臟保護機制獨立於血糖控制（Glucose-independent）。

Tirzepatide 在 SURPASS-4 事後分析中，複合腎臟終點風險降低 42%（HR 0.58），新發巨量白蛋白尿降低 59%。TREASURE-CKD 試驗正以 BOLD-MRI 探索脂肪腎與腎臟缺氧之影像學證據。新世代三重激動劑 Retatrutide 已啟動萬人規模 TRIUMPH-Outcomes Phase 3 試驗，CagriSema（Amylin+GLP-1）亦進入 Phase 2 腎臟試驗（NCT06131372）。

KDIGO 2024 指南已將 GLP-1Ras 正式納入 CKD 標準治療框架（Recommendation 3.9.1, 證據等級 1B），並啟動聚焦更新以擴展適應症至非糖尿病 CKD 族群。臨床實務中需注意腸胃道副作用引發之急性腎損傷風險，落實「病假用藥法則」（Sick Day Rules），在最大化長期腎臟保護效益與防範短期風險之間取得個別化平衡。

References: NEJM 2024 (FLOW, NEJMoa2403347); Eur Heart J 2024;46(12):1096; Kidney Int/PMC11271413 (SELECT); Diabetes Care 2022 (SURPASS-4); KDIGO 2024 CKD Guideline; NCT05536804 (TREASURE-CKD); NCT06383390 (TRIUMPH-Outcomes)

# 專題演講 3

## Implementing MASLD/MASH Care in CKD: Screening, Fibrosis Risk Triage, and When to Refer

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代謝異常脂肪性肝病(MASLD)為重要的肝臟疾病，全世界來說，大約有 25%-30%的盛行率。有 MASLD 疾病的病人，除了肝臟本身的影響之外，其身體的器官也會受到很大的影響，包含心臟血管疾病、慢性腎臟病(CKD)、退化性關節炎，肌少症與惡性腫瘤的發生率增加等等。其中對慢性腎臟病的影響非常的顯著。據統計有 MASLD 疾患的病人其罹患 CKD 的風險會增加 1.5 倍，且若伴隨有多重代謝異常，則其風險會顯著上升。因此在 MASLD 病人早期篩檢出高風險 CKD 的次族群病人就非常的重要。以 MASLD 疾病來說，肝臟纖維化的程度對於病人預後(包含肝臟與非肝臟相關)是最為重要的。因此篩檢出嚴重肝纖維化的病人是首要任務。以非侵入性的篩檢方式，如 FIB-4 score 與肝纖維掃描(vibration-controlled transient elastography, VCTE) 是目前各學會建議的篩檢工具，然而卻有其診斷上的限制。以 FIB-4 來說，會受到年齡的影響而減少其篩檢的敏感度。在過去的研究來說，病人有代謝異常疾病，譬如糖尿病、高血壓、肥胖等等，皆會影響 FIB-4 的特異性，導致有肝纖維化的病人無法被篩檢出來。此次演講旨在介紹 MASLD 對 CKD 的影響，與如何篩檢出高風險的病人，以及篩檢出來後，以腸胃科醫師的角度，又會怎麼樣的去建議治療。

# 專題演講 4

KDIGO 2025 Clinical Practice Guideline for Anemia update
KDIGO 2025 貧血臨床實務指引更新

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繼 2012 年版本後，KDIGO 在 2026 年發表慢性腎臟病貧血臨床實踐指南。本次更新彙整了 RCT 臨床證據，也引進了對病理生理學的理解。指南最顯著的變革之一在於術語的更新，捨棄「絕對」與「功能性」等詞彙，改用更具生理描述性的名稱，系統性鐵缺乏 Systemic iron deficiency： 原稱「absolute IDA」，代表患者儲存鐵量皆已枯竭。鐵受限紅血球生成 Iron-restricted erythropoiesis： 原稱「功能性鐵缺乏」。這是指患者體內並非沒鐵，而是因為發炎引起 Hepcidin 升高，導致鐵被「鎖」在巨噬細胞中，無法支應骨髓造血。針對不同分期的建議篩檢頻率為: CKD G3 每年一次;CKD G4 每年兩次; CKD G5 每三個月一次。若篩檢未發現病因，應擴展檢查範圍到 reticulocyte、Vit B12、folic acid、CRP、LDH、Haptoglobin 等。這次更新的另一個重點在鐵劑治療的啟動與主動式 proactive 策略，根據 PIVOTAL 試驗的實證，將血液透析患者與其他類別區隔開來。血液透析病人 Ferritin ≤ 500 ng/ml 且 TSAT ≤ 30%，靜脈注射定期給藥以維持鐵狀態穩定，而非等數值掉落才反應。 腹膜透析病人則是 Ferritin < 100 ng/ml 且 TSAT < 40% 或. Ferritin 100–300 ng/ml 且 TSAT < 25%，可以口服或靜脈注射補充。所有病人在 Ferritin ≥ 700 ng/ml 或 TSAT ≥ 40% 時，暫停鐵劑治療。這次更新的第 3 個重點在 ESA 與新型藥物 HIF-PHI 的定位，指南給予了明確的優先級建議： 儘管 HIF-PHI 為口服選項，指南仍建議將 紅血球生成刺激劑 (ESA) 作為首選治療方案。同時不建議臨床上同時併用 ESA 與 HIF-PHI。這份 2025 年更新指南透過生理化的術語與更靈活的給藥策略，保護患者免於心血管風險與輸血副作用。臨床醫師在應用此指南時，應優先考量 Shared Decision-making，根據患者的症狀、潛在併發症風險及未來移植計畫，量身打造最合適的貧血治療計畫。

# 專題演講 4

KDIGO 2025 Clinical Practice Guideline for ADPKD
KDIGO 2025 多囊腎臨床診療指引

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本課程介紹 KDIGO2025 年的 ADPKD 指引，一共有十章，內容包括介紹 ADPKD 的定義、命名、盛行率、診斷方法、預後因子、血壓控制，以及腎結石、痛風、血尿和囊腫感染的處理方式。治療方面，除介紹 tolvaptan 的適應症和劑量外，也強調以共享決策來決定透析方式、腎臟移植和手術方式等。此外，也介紹多囊肝、血管瘤及其它腎外臨床表現。再來針對生活方式、心理社會面向、懷孕與生殖、兒童病人等做討論，最後以病人的綜合照護做總結。

Chapter 1: Nomenclature, diagnosis, prognosis, and prevalence

Chapter 2: Kidney manifestations

Chapter 3: Chronic kidney disease management and progression, kidney failure, and kidney replacement therapy

Chapter 4: Therapies to delay the progression of kidney disease

Chapter 5: Polycystic liver disease

Chapter 6: Intracranial aneurysms and other extrarenal manifestations

Chapter 7: Lifestyle and psychosocial aspects

Chapter 8: Pregnancy and reproductive issues

Chapter 9: Pediatric issues

Chapter 10: Approaches to the management of people with ADPKD

# 專題演講 5

KDIGO 2025 IgA nephropathy/IgA vasculitis guideline
KDIGO 2025 IgA 腎病與 IgA 血管炎臨床指引

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台大醫院腎臟科

2025 年發表的 KDIGO 針對 IgA 腎病變（IgAN）與 IgA 血管炎（IgAV）的照護指引指引，相較於 2021 年版本並無太大不同，但本次更新反映近年最新的臨床試驗與治療進展，提出更積極的診斷與治療策略。

在診斷方面，IgAN 仍須依賴腎臟切片確診，目前尚無經驗證的血液或尿液生物標記。2025 年指引建議對疑似 IgAN 且蛋白尿≥0.5 g/day 的成人採取較寬鬆的腎切片策略，以利早期診斷與介入。預後評估則建議結合臨床與病理資訊，並使用國際 IgAN 預測工具進行風險分層。

在治療目標上，指引強調應將蛋白尿控制至<0.5 g/day（理想<0.3 g/day），並維持腎功能穩定，目標為減緩腎功能下降至接近生理速度。核心治療概念為「雙軌策略」：一方面抑制致病性 IgA 及免疫複合體的形成與作用，另一方面處理既有腎元損傷所引發的慢性腎病變進展。

針對免疫機轉，目前建議使用標靶釋放型 budesonide（Nefecon）或減量全身性類固醇治療；在特定族群（如中國患者）可考慮 mycophenolate mofetil。對於腎臟保護，所有患者皆應接受生活型態介入（低鹽飲食、體重控制、戒菸、運動）及藥物治療，包括腎素-血管張力素系統抑制劑（RASi）、SGLT2 抑制劑及雙重內皮素-血管張力素受體阻斷劑（如 sparsentan）。

指引亦強調新藥可針對不同致病機轉，未來可能需採取合併與維持治療策略，以達到最佳長期腎臟保護效果。然而，目前仍缺乏最佳用藥順序與組合的高品質證據。
特殊情境（如快速進展型 IgAN、腎病症候群、急性腎損傷、妊娠與兒童患者）之建議大致延續 2021 年版本，主要因缺乏新的臨床試驗資料。IgAV 方面，目前尚無足夠證據支持將 IgAN 新療法直接套用。

總結而言，IgAN 不再被視為良性疾病，多數患者若未適當治療將進展至腎衰竭。新指引強調早期診斷、嚴格蛋白尿控制及機轉導向治療，並指出多重藥物組合可能為未來治療趨勢，以延緩疾病進展並維持終身腎功能。

# 專題演講 5

KDIGO 2024 ANCA-associated vasculitis guideline：Clinical challenges in Taiwan
KDIGO 2024 ANCA 相關血管炎臨床指引與台灣實務的挑戰

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抗嗜中性白血球相關之血管炎(Anti-neutrophil Cytoplasmic Autoantibody-Associated Vasculitis, AAV)是小血管發炎而造成器官損傷的一群疾病，可以侵犯全身多個不同器官系統，而腎臟是常見受影響的器官。

本次報告，聚焦於 KDIGO 2024 ANCA guideline 更新的內容，並針對台灣臨床實務的挑戰進行討論。重點包含抗嗜中性白血球相關之血管炎的診斷、引導(induction)治療、維持(maintenance)治療、以及血漿治療(therapeutic plasma exchange)，並提出實際臨床案例分享。

根據目前研究以及報告人臨床經驗，及時的診斷與治療、適切調整 cyclophosphamide 劑量、考慮 rituximab 使用、使用 reduce-dose glucocorticoid regimen、選擇合適病患進行血漿治療、並監測疾病復發，是重要的治療考量。

# 專題演講 5

TCR commentary on the KDIGO IgA and AAV guidelines

風濕病醫學會意見分享
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中華民國風濕病醫學會理事長

As a rheumatologist, I find the convergence of the 2025 BSR, 2024 KDIGO, and 2022/2024 EULAR guidelines highly encouraging, though subtle differences reflect their respective specialty focuses. A major point of consensus is the shift toward B-cell depletion. All three guidelines recommend Rituximab (RTX) or Cyclophosphamide (CYC) for induction, with a clear preference for RTX in relapsing disease. Furthermore, they all embrace steroid-sparing strategies, specifically the reduced-dose glucocorticoid (GC) tapering regimens (derived from the PEXIVAS trial) and the incorporation of Avacopan to minimize GC-related toxicity. However, the management of severe renal disease remains a point of divergence. While EULAR and BSR are increasingly comfortable using RTX even in severe renal impairment, KDIGO 2024 remains more conservative, noting limited prospective data for RTX when serum creatinine (SCr) exceeds 4 mg/dl (354 μmol/l) and often favoring CYC or a CYC+RTX combination in this high-risk setting. Regarding Plasma Exchange (PLEX), the guidelines have moved toward more restrictive use. BSR and EULAR align by suggesting PLEX only for severe renal disease (SCr >300 μmol/l) and recommending against its routine use for pulmonary hemorrhage alone. Conversely, KDIGO still allows for PLEX in patients with diffuse alveolar hemorrhage (DAH) accompanied by hypoxemia. For maintenance, RTX is the preferred agent across the board

In conclusion, while KDIGO emphasizes renal-specific protection, BSR and EULAR provide a more systemic rheumatological framework. These updates collectively move us toward a precision-medicine approach that prioritizes long-term remission while aggressively reducing treatment-related damage.

# 專題演講 6

Precision Medicine in Inherited Kidney Diseases: From Gene to Therapy
遺傳性腎臟病的精準醫學：從基因到治療

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Inherited kidney diseases collectively account for 10–15% of adult end-stage kidney disease (ESKD) and up to 70% of pediatric chronic kidney disease (CKD), with over 600 disease-associated genes identified to date. Advances in next-generation sequencing — from targeted gene panels to whole exome and whole genome sequencing — have transformed our ability to establish precise genetic diagnoses, with diagnostic yields reaching 25–35%.

This lecture highlights how genotype–phenotype correlations drive precision medicine across the spectrum of inherited nephropathies. Fabry disease exemplifies this paradigm: GLA genotype determines phenotype severity, renal prognosis, and therapeutic strategy — including eligibility for oral chaperone therapy (migalastat) in amenable mutations versus enzyme replacement therapy (ERT) for classical variants. Taiwan's newborn screening program has revealed a uniquely high prevalence of the later-onset IVS4+919G>A variant (up to 1:1,250), demanding tailored surveillance protocols.

Beyond Fabry disease, genotyping informs clinical decision-making in Alport syndrome (COL4A5 truncating variants predicting earlier ESKD and post-transplant anti-GBM risk), ADPKD (PKD1 versus PKD2 prognostication guiding tolvaptan eligibility), and genetic FSGS (avoiding months of futile immunosuppression). Emerging RNA therapeutics — notably lumasiran for primary hyperoxaluria type 1 — have revolutionized treatment and transplant strategy. In aHUS, complement genotyping directly guides the duration of C5 blockade (eculizumab/ravulizumab) and transplant planning.

Genetic testing has also become indispensable in pre-transplant evaluation: determining disease recurrence risk, screening living related donors, and guiding peri-transplant strategies. Gene therapy and mRNA-based approaches are rapidly entering clinical trials, expanding the precision therapeutic landscape.

Taiwan's genomic infrastructure — including the Taiwan Precision Medicine Initiative (TPMI), comprehensive newborn screening, and the Rare Disease Act — uniquely positions us to advance precision nephrology. This talk will provide a comprehensive overview from gene to therapy, equipping nephrologists with practical knowledge to integrate genetic medicine into everyday clinical practice.

# 專題演講 6

## Utilizing TPMI Genomic Data for Personalized Prediction Models of Kidney Diseases

## 運用 TPMI 基因數據建立腎臟病之個人化預測模型

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隨著台灣精準醫療計畫（TPMI）成功建立百萬人規模的基因體數據庫 ，醫療模式正從傳統的病後治療轉向預測與預防。本演講將介紹如何利用 TPMI 資源，針對腎臟與自體免疫疾病開發創新應用。

演講重點首先聚焦於單基因與多基因風險分數(PRS)的臨床整合。我們發現透過 TPMI 特有的族群風險評分，能早期發現全身性紅斑狼瘡的高風險群，我們將分享前瞻性召回（Prospective Recall）研究經驗，透過基因篩選召回高風險 SLE 受試者進行早期免疫特徵分析，達成「病前精準醫療」。以單細胞定序探索疾病發生前期的免疫異常。特別的是，本研究團隊開拓了基因與環境交互作用的領域，揭露了 PRS 與環境因子對免疫腎臟疾病發病風險的加乘效應 。

最後，我們已成功將這些研究轉譯為臨床實務，開發出單基因與多基因遺傳的疾病風險檢測，為腎臟科及風濕免疫科醫師提供科學化的決策工具，實踐精準健康管理。

# 專題演講 6

Perspectives on TMA: Precision Medicine and Long-term Management of aHUS
透視血栓性微血管病變：aHUS 的精準醫療與長期預後管理

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Thrombotic Microangiopathy (TMA) is a clinical emergency characterized by microangiopathic hemolytic anemia, thrombocytopenia, and acute vital organ injury. Among the diverse spectrum of TMA, atypical Hemolytic Uremic Syndrome (aHUS) remains a particularly challenging in the diagnosis and treatment, that is driven by dysregulation of the alternative complement pathway.

This session provides a comprehensive overview of aHUS, focusing on the paradigm shift toward precision medicine through the integration of genetic insights. Central to this approach is the identification of germline mutations in complement genes—such as CFH, CFI, CD46, and CFB—which not only confirm the diagnosis but also play a pivotal role in risk stratification and predicting the likelihood of post-transplant recurrence.

We will also discuss the clinical "red flags" that differentiate aHUS from other TMAs and examine how genetic profiling informs the transition from empirical therapy, such as plasma exchange to targeted intervention with terminal complement inhibitors (C5 inhibitors). Furthermore, the presentation will address long-term management strategies, including the optimization of treatment duration, the feasibility of guided treatment withdrawal in patients with specific low-risk genetic variants, and the necessity of vigilant monitoring for relapse. By bridging the gap between molecular genetics and clinical practice, we will focus on the practical insights into implementing individualized, evidence-based care to preserve long-term renal function and improve patient outcomes in the era of precision nephrology.

# 專題演講 7

## A Strategic Shift Toward Home-Based Therapies and Value-Based Outcomes

## 邁向居家治療與價值導向成效的策略轉型

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As the burden of CKD and ESKD continues to rise, renal care must evolve beyond a facility-centered model toward approaches that are more sustainable, flexible, and patient-centered. The COVID-19 era further exposed the limitations of in-center hemodialysis (HD) and reinforced the value of home-based therapies. In this context, a PD-preferred strategy—meaning Peritoneal dialysis (PD)first when clinically appropriate, not PD only—offers an important direction for modern kidney care.

PD provides more than an alternative dialysis modality. It supports patient empowerment, greater control over daily life, and treatment decisions shaped through shared decision-making. For some patients, PD serves as a bridge to transplantation; for others, it is a long-term lifestyle-based therapy.

The clinical rationale is strong. Evidence from international registries and Taiwanese data shows that PD offers comparable or superior early survival versus HD in incident dialysis patients. PD is also associated with better preservation of residual renal function, an important factor linked to improved volume control, solute clearance, cardiovascular stability, and future transplant readiness. In addition, PD reduces exposure to vascular access-related bloodstream infections commonly seen with catheter-based HD. In Taiwan, peritonitis rates have remained within international quality targets, reflecting progress in PD care and monitoring.

Technological advances are making PD even more feasible. Remote patient monitoring, tele-nephrology, and multidisciplinary team support can improve follow-up, detect complications early, and strengthen home-based care delivery. However, successful expansion of PD requires more than technology alone. Persistent barriers include limited physician experience, uneven patient awareness, and system-level reimbursement challenges.

A key opportunity lies in addressing unplanned dialysis starts. Too many patients still begin treatment emergently with central venous catheters and become locked into in-center HD. Urgent-start PD can safely disrupt this default pathway and expand access to home dialysis from the outset.

A PD-preferred model is therefore not simply a modality choice, but a strategic redesign of renal care. By aligning clinical evidence, patient empowerment, digital support, professional training, and policy reform, nephrologists and renal nurses can help advance a more resilient, value-based, and humane future for kidney care.

# 專題演講 7

Digital PD: Innovation for Better Living
數位化腹膜透析：以創新提升生活品質

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台中榮民總醫院 腎臟科

本演講將探討智能化自動腹膜透析遠端監測（RM-APD）系統在臨床上的實務應用與臺灣在地經驗 。首先，根據臺灣本土的回顧性研究顯示，新發透析病患（Incident PD patients）在導入 RM-APD 系統後，其三年內的腹膜炎發生率有顯著改善（HR 0.38, 95% CI 0.15-0.96）。此外，國際大型隨機對照試驗亦指出，RM-APD 能有效降低全死因死亡率與心血管相關死亡率 。其次，分享臺中榮民總醫院如何透過智能化系統建立標準化臨床路徑。利用結構化的異常警訊處置流程（Flag alerts procedure），將雲端數據轉化為具體的臨床行動，成功解決了護理人力吃緊時的照護瓶頸，並能針對多重共病或缺乏支持系統的特殊病患提供精準守護 。最後，將討論智能化系統如何成為吸引病患選擇居家透析的誘因，並配合健保署最新的「病人遠端管理（RPM）」給付計畫，強化病患教育與醫病協作，達成「Think Big, Start Small」的轉型目標，提升病患的治療品質與生活自主性。

# 專題演講 7

PD Beyond the Hospital: Community Clinics as Both Hubs and Spokes
走出醫院的 PD：以社區診所建立樞紐—輻射式照護模式

王介立 醫師
台北市柏安診所

台灣約 6,200 名腹膜透析病人中，僅 43 人（0.7%）於基層診所接受完整的慢性腹膜透析照護。腹膜透析屬居家治療，病人生活於社區，卻幾乎不在社區接受照護。此一「常識」是否合乎邏輯？本演講訪談一家開業僅一年的社區透析診所，其團隊照護 20 餘位腹膜透析病人，以門診靜脈抗生素治療腹膜炎、將原本需住院者留在社區，照護功能與醫院腎臟科團隊無本質差異；且其末期腎臟病前期（Pre-ESRD）門診中，約半數新進末期腎臟病個案選擇腹膜透析。本演講主張一項認知典範轉移：具備條件的社區診所可發展為具樞紐功能之腹膜透析中心，協同輻射診所與醫院形成分散式網絡，值得納入政策討論。

# 專題演講 8

Session I: Decoding the Life-Line: From Anatomical Foundation to Functional Evaluation

解碼生命線—從解剖基礎到功能評估

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Arteriovenous fistula (AVF) is the preferred vascular access for hemodialysis, traditionally evaluated based on anatomical suitability, maturation, and access patency. This lecture provides a clinically oriented framework to reassess vascular access, starting from conventional functional evaluation and extending toward a broader physiological perspective.

We will briefly review upper limb vascular anatomy and the process of AVF maturation, including shear stress–mediated remodeling and common causes of maturation failure. Functional evaluation will then be discussed from a practical standpoint, including standardized physical examination and access flow measurement. In daily practice, parameters such as Qa and dialysis-derived flow data (e.g., HD03) are primarily used to assess access patency and detect dysfunction.

Building on this foundation, the lecture will further explore how access flow can be reinterpreted beyond patency. By relating Qa to cardiac output (Qa/CO ratio), we highlight its role as an indicator of systemic hemodynamic burden rather than a purely local parameter. Ultrasound will be briefly addressed as a complementary tool for anatomical and functional assessment.

By conceptualizing AVF as a low-resistance shunt, we will discuss its impact on preload, cardiac output, and pulmonary circulation, and their clinical implications in conditions such as heart failure, pulmonary hypertension, and dialysis-related hemodynamic instability. A representative case of pulmonary hypertension exacerbation following AVF creation will be briefly presented to illustrate this concept.

This lecture emphasizes a shift from an access-centered to a patient-centered approach, highlighting that optimal vascular access should balance patency with overall physiological tolerance.

# 專題演講 8

Session II: The Sonographic Sentinel: Ultrasound's Role in the Proactive Management of AVF Complications

超音波守門人：超音波在動靜脈瘻管（AVF）併發症前瞻性管理中的角色

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在無症狀的透析病人中，動靜脈瘻管（AVF/AVG）仍需定期接受超音波評估，以早期發現潛在異常並預防通路失敗。檢查時通常以表格化方式系統性紀錄，包括血流量（flow volume）、血管直徑、尖峰收縮期速度（PSV）、PSV 比值、彩色都卜勒影像是否出現疊影（aliasing）、血流方向以及是否存在血栓或假動脈瘤等。這些指標可協助判斷是否有狹窄、血流過高或早期阻塞等問題，即使病人尚未出現症狀，也能及早介入處理。

上肢周邊血管超音波主要用於評估透析通路之功能與結構。檢查內容涵蓋動脈 inflow、吻合處（anastomosis）以及靜脈 outflow，並觀察血流速度、流量與血管型態變化。正常情況下，血管應具有良好血流與適當管徑，而瘻管成熟則需具備足夠血流量與可供穿刺的靜脈條件。若瘻管未成熟，常見原因包括血流不足或靜脈狹窄。

在併發症方面，最常見為血管狹窄與血栓形成，其機轉多與內膜增生有關。超音波診斷狹窄時，可見局部血流速度上升、PSV 比值增加及彩色都卜勒出現疊影現象。若進一步惡化，可能導致完全阻塞，出現無血流或高阻力波形。除此之外，長期使用亦可能形成動脈瘤或假動脈瘤，其典型影像包括「陰陽圖樣」與「來去血流」。其他併發症尚包括血腫、感染及液體聚集。

此外，部分病人可能發生竊血症候群，表現為遠端動脈血流逆轉；而當瘻管血流過高時，則可能導致高輸出量心衰竭。這些情況皆需透過超音波早期辨識。總體而言，上肢周邊血管超音波在透析通路的建立、追蹤與併發症診斷中扮演關鍵角色，而定期監測無症狀病人更是維持瘻管功能的重要策略。

# 專題演講 8

Session III: Beyond the Surface: Breakthrough Strategies for Challenging Cannulation
突破表層：困難穿刺的創新策略

韓雲楷 醫師
輝德診所

The primary challenge for patients undergoing hemodialysis via an arteriovenous (AV) access is successful cannulation. Based on our experience over the past six years in managing challenging cannulations, we have identified eight underlying causes: (1) bifurcations within the arteriovenous fistula (AVF); (2) AV access that is excessively deep and/or narrow; (3) curved AV access; (4) irregular AV access diameter; (5) difficulty identifying the AV access; (6) thrombi within the AV access; (7) protrusions within the AVF; and (8) stents within the AV access. A review of the literature reveals a lack of studies that systematically identify and address these challenges. Therefore, we have organized and summarized our experiences through a series of case reports to illustrate these issues and propose potential solutions. Cannulation of AV access is an ongoing skill that merits further investigation to improve outcomes.

# 專題演講 9

## Obesity-Associated Metabolic Imbalance and Kidney Injury

肥胖相關代謝失衡與腎臟病變

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東亞族群特有之 ALDH2 功能性變異（rs671, Glu504Lys）廣泛存在於華人、日本與韓國族群中，約影響超過 5 億人口，並使粒線體醛去氫酶活性下降至約 30–45%，導致反應性脂質醛類（特別是 4-hydroxy-2-nonenal, 4-HNE）清除能力顯著受損。近年人類遺傳學研究指出，ALDH2 不僅參與酒精代謝，更為東亞族群代謝疾病與器官功能異常的重要遺傳決定因子。

在全身代謝層面，ALDH2 功能缺失導致 4-HNE 在粒線體內累積並對關鍵代謝蛋白產生共價修飾，特別影響粒線體脂肪酸氧化（FAO）與電子傳遞鏈（ETC）。在棕色脂肪組織中，此種修飾使 FAO 與粒線體呼吸能力顯著下降（約 70%），造成能量消耗降低與脂質利用受阻，進一步導致肥胖、脂肪肝、胰島素阻抗與葡萄糖代謝異常。

在高能量需求器官中，此一機制對心肌影響尤為顯著。4-HNE 的累積會損害心肌粒線體呼吸鏈與鈣離子調控，並促進氧化壓力與發炎反應，最終導致心肌收縮與舒張功能受損。在糖尿病心肌病變模型中，ALDH2 功能低下與心肌能量代謝障礙、纖維化與功能衰退密切相關。

進一步地，4-HNE 的累積可導致腎絲球與腎小管細胞粒線體功能受損，促進足細胞結構破壞、發炎反應與纖維化進展，最終導致慢性腎臟病（CKD）發展。在急性腎損傷（AKI）情境下，4-HNE 亦會加劇粒線體失能與細胞損傷，並在 AKI 向 CKD 轉變過程中驅動 maladaptive repair 與持續性纖維化，形成不可逆的腎功能惡化。

在治療策略上，ALDH2 活化提供一項可同時介入多器官病理的關鍵切入點。新一代 ALDH2 活化劑 AD-9308（其活性代謝物 AD-5591）可透過穩定酵素結構提升其催化效率，降低 4-HNE 累積並恢復粒線體功能。在代謝疾病模型中，AD-9308 可改善肥胖、脂肪肝、胰島素阻抗與血糖控制；在心肌病變中，可改善粒線體呼吸與鈣離子調控並恢復心臟功能；在肥胖肌少症模型可改善肌肉質量與力量，在腎臟疾病模型中，則可降低腎臟氧化壓力、改善粒線體功能並抑制纖維化進展。特別是在 AKI 向 CKD 轉變過程中，ALDH2 活化可有效阻斷疾病惡化。

ALDH2–4-HNE–粒線體軸將東亞族群特異性遺傳變異轉譯為可被精準逆轉的跨器官病理機制，並提供同時重塑代謝、肌肉與腎臟疾病進程的統一治療節點。

# 專題演講 9

## Obesity × Kidney Disease: Emerging Guidelines for Weight Management from CKD to Dialysis — From Weight Reduction to Cardiorenal Protection

肥胖 × 腎臟病：從 CKD 到透析的體重管理新指引—從『減重』出發到『心腎保護』

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本演講聚焦肥胖與慢性腎臟病之密切交互作用，從早期 CKD 至透析階段，系統整理近年體重管理的新指引與實證進展。內容將說明肥胖如何影響腎功能惡化、心血管風險與代謝失衡，並進一步探討臨床策略如何由傳統「減重」思維，轉向兼顧體重控制、心腎保護與整體預後改善。演講亦將涵蓋生活型態介入、藥物治療及透析病人的特殊考量，協助臨床醫師建立以病人為中心的整合照護模式。

# 專題演講 9

Bridging the Gap: How Metabolic-Bariatric Surgeons and Nephrologists Can Co-manage Obesity-Driven Chronic Kidney Disease

重塑照護模式：代謝減重外科與腎臟科攜手管理肥胖驅動之慢性腎臟病

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Obesity-driven chronic kidney disease (CKD) has emerged as a major clinical consequence of the modern metabolic epidemic, linking visceral adiposity, insulin resistance, hypertension, chronic inflammation, oxidative stress, and glomerular hyperfiltration to progressive renal injury and obesity-related glomerulopathy. In patients with severe obesity, early hyperfiltration may obscure underlying kidney damage, and conventional creatinine-based estimates of glomerular filtration may be less reliable, making timely recognition and risk stratification more challenging. At the same time, despite contemporary medical therapy including RAAS blockade, intensive glycemic and blood pressure control, SGLT-2 inhibitors, and GLP-1 receptor agonists, a substantial residual risk of CKD progression remains in high-risk obese and diabetic populations.

This lecture will review the pathophysiologic links between obesity, metabolic syndrome, and CKD, and examine the growing evidence that metabolic-bariatric surgery (MBS) may provide kidney-relevant benefits beyond weight reduction alone. A systematic review and meta-analysis in patients with CKD and severe obesity found that MBS was associated with improved eGFR, reduced serum creatinine, and a lower likelihood of albuminuria above 30 mg/g, although effects on CKD stage were less clear. In addition, a recent meta-analysis of randomized controlled trials showed that the presence of CKD did not significantly diminish the safety, weight-loss efficacy, or metabolic benefits of metabolic-bariatric surgery in patients with obesity and type 2 diabetes. Taken together, these data support an earlier, structured co-management model in which nephrologists and metabolic-bariatric surgeons collaborate on patient selection, perioperative optimization, and longitudinal monitoring of renal, nutritional, and cardiometabolic outcomes before irreversible kidney decline occurs.

# Lunch Symposium 1

Beyond RAAS: The New Standard of Care for CKD

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慢性腎臟病（CKD）已成為全球重要的公共衛生議題，尤其在糖尿病、肥胖、高血壓與高齡化盛行的背景下，CKD 患者的心血管事件、腎功能惡化及進入透析之風險持續攀升。本演講以「Beyond RAAS: The New Standard of Care for CKD」為題，將從現代 CKD 治療策略的演進出發，探討臨床實務如何由過去以 RAAS blockade 為基礎的傳統模式，進一步邁向以 SGLT2i 為核心的新照護標準。演講內容將結合 KDIGO 最新觀點與近年重要臨床證據，說明肥胖與代謝異常如何透過腎絲球高過濾、慢性發炎、氧化壓力、脂肪毒性及纖維化等多重機轉，加速 CKD 的發生與進展，並強調及早阻斷心腎代謝連鎖反應的重要性。

此外，本演講將聚焦 SGLT2i 在 CKD 患者中的臨床價值，包括延緩 eGFR 下降、減少蛋白尿、降低心衰竭與腎臟替代治療風險，以及在糖尿病與非糖尿病腎病患者中的保護效果，說明其已不再只是降血糖藥物，而是 CKD 治療的重要基石。演講亦將進一步介紹 CKD 合併第二型糖尿病之「四本柱」整合治療概念，包括 RASi、SGLT2i、ns-MRA 與 GLP-1 RA，並討論如何透過快速序列啟動與個人化監測，在更早期、更完整地落實實證醫療，以期延後透析、改善心腎終點，並建立真正以病人為中心的慢性腎臟病整合照護新模式。

# Lunch Symposium 2

## Navigating from MGUS to MGRS: The Critical Role of Serum Free Light Chains in Renal Involvement and Clinical Decision Making

## 從 MGUS 到 MGRS：血清游離輕鏈在腎臟損傷中的臨床價值與決策關鍵

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林口長庚紀念醫院 腎臟科

單株免疫球蛋白血症（Monoclonal Gammopathy）具有高度的臨床異質性，從相對良性的 MGUS（意義不明的單株免疫球蛋白血症）到具侵略性的多發性骨髓瘤，期間的準確診斷與風險層級至關重要。近年來「具有腎臟意義的單株免疫球蛋白血症（MGRS）」的概念陸續被提出，強調即使漿細胞異常增生，而尚未達到惡性腫瘤診斷標準，其產生的單株免疫球蛋白或游離輕鏈（Free Light Chain, FLC）仍可能對腎臟造成不可逆的損傷。

本次講座將深入解析血清游離輕鏈（sFLC）檢測在腎臟病學中的核心應用。首先，我們將討論在慢性腎臟病（CKD）背景下，如何正確判讀 sFLC 比值，並強調應用「腎功能校正參考區間（Renal-specific Reference Range）」進行判讀的必要性，以優化診斷的特異性並減少不必要的切片檢查。

其次，演講重點將論述 sFLC 在 MGRS 早期診斷中的關鍵地位。MGRS 患者往往因分泌極少量的輕鏈即可導致嚴重的腎病變（如 AL-amyloidosis 或單株免疫球蛋白沉積症 MIDD），傳統電泳常難以察覺，而 sFLC 檢測提供了極高的敏感度，是早期篩選與追蹤療效的必備工具。最後，結合臨床實體案例分享，探討 sFLC 在潛伏的多發性骨髓瘤（SMM）中的預後價值，協助醫師評估何時該從「觀察」轉向「積極介入」。透過本次專題，聽眾將能掌握如何運用 FLC 指標，在複雜的漿細胞疾病中精準定位腎臟損傷風險，建立更完善的臨床決策路徑。

# Lunch Symposium 3

## 糖心腎患者的關鍵感染風險：帶狀疱疹與 RSV 防護策略

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對於糖尿病合併心血管與腎臟疾病的糖心腎族群而言，免疫功能本已受慢性發炎與代謝失衡影響，感染所帶來的衝擊往往遠高於一般人。帶狀疱疹不僅造成劇烈神經痛與長期帶狀疱疹後神經痛，更可能在急性期誘發心肌梗塞或中風風險上升；對心血管疾病患者而言，病毒再活化所引發的發炎反應與血管內皮不穩定，可能成為觸發事件。對腎臟病患者來說，疼痛與感染壓力也可能加速腎功能惡化，增加住院與醫療資源使用。

另一方面，**呼吸道融合病毒（RSV）**在中高齡慢性病患者中，可能導致嚴重下呼吸道感染與肺炎。糖心腎患者一旦感染 RSV，較易出現呼吸衰竭、心衰竭惡化或代謝失衡，甚至增加短期死亡風險。感染期間的全身性發炎反應與低氧狀態，也會加重心臟與腎臟負擔。

因此，對糖心腎族群而言，這些病毒不只是「感染問題」，而是可能引發連鎖併發症的風險事件。及早風險評估與預防策略，是降低急性事件與長期預後惡化的重要關鍵。

# Lunch Symposium 4

Precision Nephrology: Resolve the Safety-Prognosis Conflict to Sustain Cardiorenal Benefit

護腎不煞車—強化醫病信心，鎖定長期腎臟保護效益

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隨著 RAASi、SGLT2i、nsMRA 等藥物證據的完備，當前慢性腎臟病（CKD）的標準治療已臻成熟。不過臨床實務中，醫師常因患者出現急性狀況（Sick days）或高血鉀等藥物相關副作用而暫時停藥。國際共識皆認為目前臨床上最大的潛在風險在於「停藥後未能及時重新啟動治療」，導致患者失去長期的心腎保護效益。

本次演講將深入剖析台灣 CKD 照護中常見的「藥物副作用與長遠預後」之衝突困境，探討如何建立明確的臨床決策路徑與醫病溝通計畫。我們將分享在面對臨床常見不良事件時，如何透過精準的風險控管，而非全面性、永久性的停用藥物，來強化治療方案的韌性。核心目標在於建立「護腎不煞車」的治療共識，確保基礎治療（Standard of Care）能持續落實，進而共同最大化患者的長期心腎預後效益的同時，亦能兼顧臨床用藥安全性，鞏固醫病雙方對於長期用藥的信心。

While the integration of RAASi, SGLT2i, and nsMRA has redefined the standard of care for chronic kidney disease (CKD), a significant gap remains between clinical evidence and real-world implementation. Clinicians frequently suspend these essential therapies due to "sick days" or drug-related adverse events such as hyperkalemia. International consensus highlights that the primary clinical concern today is failure to restart therapy promptly following such interruptions, limiting the long-term cardiorenal protective benefits to patients.

This session goes into detail about the "Safety-Prognosis Conflict" in Taiwan's CKD landscape, emphasizing the need for structured clinical decision pathways and effective communication strategies with patients. By prioritizing "therapeutic resilience" through precise risk mitigation rather than permanent discontinuation, we aim to foster a "Don't Hit the Brakes" agreement to ensure the continuous delivery of evidence-based therapy, maximizing long-term patient outcomes while balancing clinical safety and restoring confidence in long-term treatment adherence for both physicians and patients.

# 病例報告
# 摘要

# 病例報告 1

1-1

## 三酸甘油酯–葡萄糖指數在新開始腹膜透析患者中的預後意義

## Prognostic Significance of the Triglyceride–Glucose Index in Patients Starting Peritoneal Dialysis

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**Abstracts**

**Background：**

The triglyceride–glucose (TyG) index is a simple surrogate marker of insulin resistance. Its prognostic value in incident peritoneal dialysis (PD) patients remains unclear. This study evaluated the association between TyG index and mortality risk in PD.

**Methods：**

We retrospectively enrolled 553 adult patients initiating PD between 2003 and 2017. Patients were categorized into TyG tertiles. Kaplan–Meier analysis and multivariable Cox regression were used to assess associations with all-cause and cardiovascular mortality.

**Results：**

During a mean follow-up of 3.83 years, 142 all-cause deaths and 89 cardiovascular deaths occurred. Higher TyG tertiles were associated with increased mortality risk. In fully adjusted models, the highest tertile showed hazard ratios of 2.12 for all-cause mortality and 2.78 for cardiovascular mortality compared with the lowest tertile. The optimal TyG cut-off values were 8.79 and 8.85 for all-cause and cardiovascular mortality, respectively.

**Conclusions：**

Higher TyG index independently predicts increased mortality risk in incident PD patients and may serve as a simple biomarker for risk stratification.

**Key words：**

Cardiovascular disease, Chronic kidney disease, Mortality, Peritoneal dialysis (PD), Triglycerideglucose(TyG) index

# 病例報告 1

1-2

## 組織血栓溶解劑(rTPA)引發之動脈粥狀硬化栓塞性腎病模仿非典型溶血性尿毒症候群之病例報告

## Atheroembolic renal disease (AERD) mimicking atypical hemolytic uremic syndrome (aHUS) following rTPA administration: a case report

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**Background**：The clinical presentation of atheroembolic renal disease (AERD) can closely mimic that of atypical hemolytic uremic syndrome (aHUS), as both conditions may involve immunothrombosis in their pathogenesis. Recent studies also indicate that complement activation (such as the C5a/C5aR pathway) plays a critical role in acute kidney injury and vascular thrombosis induced by cholesterol crystal embolism.
**Methods**：We describe a clinical case of a patient who presented with suspected aHUS after receiving recombinant tissue plasminogen activator (rTPA) therapy and was ultimately diagnosed with AERD via kidney biopsy.
**Results**：A 64-year-old female presented with a history of hypertension, coronary artery disease, and a recent ischemic stroke in September 2024. The patient received rTPA therapy following her stroke. Within a month, she developed abdominal pain, skin rash with scaly changes over her trunk and limbs (including plantar livedo reticularis), and bilateral toe gangrene. Laboratory investigations revealed acute kidney injury with uremic syndrome (creatinine up to 16.2 mg/dL) , the presence of schistocytes (1+) on the peripheral blood smear , decreased haptoglobin (<30 mg/dL) , and normal ADAMTS-13 activity (60%), and a possible MCP gene variant. Initially suspected to have aHUS complicated by microangiopathic hemolytic anemia (MAHA) and thrombocytopenia , the patient underwent hemodialysis, plasmapheresis, and Eculizumab therapy, which led to hematological remission but persistent renal dysfunction. However, a subsequent kidney biopsy revealed arteriosclerosis with lumens occluded by atheroemboli, accompanied by multiple cleft-shaped spaces left after cholesterol dissolution. The glomeruli were essentially normal with preserved tuft architecture. A computed tomography angiography (CTA) also revealed an 80% stenosis of the superior mesenteric artery (SMA), for which she received percutaneous transluminal angioplasty (PTA). She was ultimately diagnosed with AERD. Following the diagnosis, Eculizumab was discontinued, and she was switched to steroid therapy (prednisolone).
**Conclusions**：AERD can mimic aHUS, posing a significant challenge for clinical diagnosis. In this case, the patient achieved hematological remission after Eculizumab therapy despite persistent renal dysfunction, highlighting the complex interplay of immunothrombosis. Since both conditions share complement-mediated mechanisms, complement inhibitors may still hold potential therapeutic value for AERD. Close monitoring and tissue proof are crucial for the early differentiation and management of such complications following thrombolytic therapy.
**Key words**：
Atheroembolic renal disease (AERD), atypical hemolytic uremic syndrome (aHUS), rTPA, Eculizumab, Complement

# 病例報告 1

1-3

## 嚴重高血鈣、急性腎損傷及肝膽侵犯為表現之 IgG4 相關疾病

## IgG4-Related Disease Presenting with Severe Hypercalcemia, Acute Kidney Injury, and hepatobiliary involvement

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**Abstracts**
**Background:** IgG4-related disease (IgG4-RD) is a systemic immune-mediated fibroinflammatory disorder that can involve multiple organs.
**Methods:** We report a 54-year-old man with no underlying systemic disease or regular medication use who presented with severe hypercalcemia (calcium 18.54 mg/dL) and AKI (creatinine up to 5.14 mg/dL). Although both conditions was improved by supportive treatment, the patient subsequently developed thrombocytopenia, jaundice, and hepatitis afterwards. Laboratory testing revealed markedly elevated serum IgG4 (851 mg/dL), positive anti-mitochondrial antibody, and anti–smooth muscle antibodies.
**Results:** The patient was diagnosed with IgG4-related disease with hepatobiliary involvement and immune thrombocytopenia. His condition improved after corticosteroid therapy and hepatobiliary-directed treatment.
**Conclusions:** Severe hypercalcemia and acute kidney injury (AKI) are uncommon initial manifestations and may mimic malignancy or endocrine disorders.

**Key words:** IgG4-related disease (IgG4-RD); Hypercalcemia; Acute kidney injury (AKI); IgG4-sclerosing cholangitis (Ig-SC); Immune thrombocytopenia (ITP)

# 病例報告 1

1-4

## 病例報告：一位診斷高血壓性血栓性微血管病變的年輕男性

## Malignant Hypertension Presenting as Thrombotic Microangiopathy: A Biopsy-Proven Case in a Young Adult with CD61-Positive Platelet Aggregation

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**Abstracts:** Malignant hypertension (mHTN) can cause thrombotic microangiopathy (TMA). Herein, we present a young adult diagnosed as hypertensive TMA.
**Background:** According to the 2020 International Society of Hypertension Global Hypertension Practice Guidelines, mHTN is defined as severe blood pressure (BP) elevation (systolic BP/diastolic BP >200/120 mmHg) associated with advanced bilateral retinopathy. TMA is one of the complications of mHTN. TMA is composed of MAHA (decreased hemoglobin, elevated lactic dehydrogenase, low haptoglobin level, presence of schistocytes in a peripheral blood smear, a negative Coombs test), thrombocytopenia and end-organ damage. It is assumed that severely elevated BP induces damage to the vascular endothelium of kidney, which triggers platelet aggregation with vascular lumen occlusion, resulting in MAHA, thrombocytopenia and organ dysfunction.
**Case Report:** A 19-year-old previously healthy man presented to our Emergency Department for dizziness for 1 day. Physical examination revealed elevated BP (systolic BP/diastolic BP: 236/156mmHg). Blood tests showed anemia (hemoglobin: 9.9 g/dL), thrombocytopenia (platelet: 81,000/μL), elevated creatinine (2.29 mg/dL), elevated aldosterone (26.2 ng/dL) and hyperreninemia (8.28 ng/mL/hr). Urinalysis revealed microscopic hematuria (3+) and granular cast (20-29/low power field). Renal echo showed bilateral kidneys loss of corticomedullary junction. Fundoscopy revealed bilateral hypertensive papilledema. Heart echo showed concentric left ventricular hypertrophy. Hemolysis survey showed presence of schistocyte (1+), elevated lactic dehydrogenase (984 U/L), decreased haptoglobin (<3 mg/dL) and a negative Coombs test. Anti-hypertensive agents were added. The following laboratory test showed increasing creatinine (2.29 to 2.40 mg/dL). Based on MAHA, thrombocytopenia, and worsening renal function, TMA was suspected. Plasma exchange was performed, pending results of ADAMTS-13 activity and gene analysis. ADAMTS-13 activity revealed normal range (71.3%). The patient underwent a renal biopsy, and was discharged on day 12. Pathological report revealed onion-skin-like myoepithelial proliferation of arteriolar wall, narrow vessel lumen, CD61 positive platelet aggregation in small blood vessels, and vesicles in renal tubule epithelia. No pathogenic mutation of atypical hemolytic uremic syndrome was found in gene test. Diagnosis of TMA, secondary to mHTN, was confirmed. Abdominal computed tomography will be arranged in Outpatient Department after his renal function showed stable improvement (latest creatinine: 0.91 mg/dL).
**Conclusions:** TMA is a complication of mHTN. Kidney injury may present as an end-organ damage. Treatment for mHTN may alleviate kidney damage.
**Keywords:** malignant hypertension, hypertensive thrombotic microangiopathy

# 病例報告 1

1-5

## 環孢素誘發之可逆性後腦病變症候群於原發性膜性腎病變患者之病例報告

## Cyclosporine-induced Posterior Reversible Encephalopathy Syndrome in a Patient With Primary Membranous Nephropathy

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**Background**
Posterior Reversible Encephalopathy Syndrome (PRES) is a neurotoxic condition characterized by acute neurological manifestations and is commonly associated with uncontrolled hypertension and exposure to immunosuppressants, such as cyclosporine. We report a case of PRES developing in a patient with nephrotic syndrome undergoing immunosuppressive therapy.

**Case presentation**
A 45-year-old woman with a history of primary membranous nephropathy presented to the emergency department with acute severe headache, hypertensive crisis, and a seizure lasting approximately 2 minutes. The seizure was characterized by eye opening, impaired awareness, initial involuntary twitching of the left hand, followed by tonic movements involving both upper extremities. Approximately 5 weeks prior to this presentation, she had been initiated on prednisolone at a dose of 0.5mg/kg/day and cyclosporine 100mg/day for the treatment of primary membranous nephropathy.
Her baseline systolic blood pressure ranged from 100 to 120 mmHg, which acutely increased to 178/102 mmHg on presentation. The physical examination was unremarkable. Neurologic examination following the resolution of seizure revealed no focal deficits, with preserved muscle strength. Brain magnetic resonance imaging (MRI) demonstrated multifocal vasogenic edema involving the pons, cerebellum, and bilateral parieto-occipital regions. PRES was diagnosed and cyclosporin was promptly discontinued.
Although initial symptoms improved following discontinuation of cyclosporine and initiation of levetiracetam, the patient experienced recurrent episodes of severe headaches accompanied by hypertensive surges three days later. These episodes responded to osmotic therapy with mannitol, suggesting persistent impairment of cerebral autoregulation. Follow-up MRI performed 8 weeks later demonstrated complete resolution of the lesions, confirming the diagnosis of PRES.

**Conclusion**
This case highlights the occurrence of early-onset and recurrent PRES associated with cyclosporin, even at reduced doses. It underscores the importance of early recognition and prompt management of this severe adverse side effect in patients receiving cyclosporin therapy.

# 病例報告 2

2-1

## 局部檸檬酸抗凝法使用於患有嚴重肺炎鏈球菌相關溶血性尿毒綜合症並接受接受連續性腎臟替代治療之個案報告

## Regional citrate anticoagulation in a patient with severe Streptococcus pneumoniae-associated hemolytic uremic syndrome requiring continuous kidney replacement therapy

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**Abstracts**

**Background：**

In Taiwan, heparin is traditionally the first-line anticoagulant for pediatric patients requiring continuous kidney replacement therapy (CKRT). However, systemic heparinization carries significant risks, particularly the need for frequent monitoring of activated partial thromboplastin time (aPTT) and the potential for iatrogenic complications of bleeding. These risks are exacerbated in patients with thrombocytopenia or disseminated intravascular coagulation (DIC). In such critical scenarios, systemic anticoagulation may result in adverse outcomes. Herein, we present a case of Streptococcus pneumoniae-associated hemolytic uremic syndrome (SpHUS) complicated by thrombocytopenia, successfully managed with CKRT using regional citrate anticoagulation (RCA).

**Key words：**

case report, streptococcus pneumoniae-associated hemolytic uremic syndrome, continuous kidney replacement therapy, continuous venovenous hemodiafiltration, regional citrate anticoagulation, hemophagocytic lymphohistiocytosis, pediatric nephrology

# 病例報告 2

2-2

## 積水背後的真相：以難治性漿膜腔積液為表現之移植後原發性積液淋巴瘤

## More Than Just Fluid Overload: Primary Effusion Lymphoma Presenting as Refractory Effusion in a Kidney Transplant Recipient

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Primary effusion lymphoma (PEL) is a rare non-Hodgkin lymphoma which primarily occurs in immunocompromised individuals. Unlike typical lymphomas, PEL arises on serosal surfaces (e.g., pleura, pericardium, and peritoneum). It usually presented as neoplastic effusions within serosal cavities without distinct solid tumor masses. Herein, we reported a patient with atypical pleural and ascitic fluids, and finally diagnosed as PEL.
This 63-year-old male has underlying disease of hypertension and obstructive sleep apnea. He developed end-stage kidney disease (ESKD) due to chronic glomerulonephritis. He underwent peritoneal dialysis (PD) for 3 years and then received cadaveric kidney transplant with delayed graft function. Despite maintenance immunosuppression, he experienced several episodes of antibody-mediated rejection and/or T-cell mediated rejection. These required intensive rescue therapies, including pulse corticosteroids, plasma exchange or plasmapheresis, intravenous immunoglobulin (IVIG), and Rituximab.
Two years post kidney transplantation, his allograft function progressively declined to chronic kidney disease (CKD) stage 5. He presented to emergency room with decreased urine output, bilateral limbs edema, exertional dyspnea and nausea. The initial diagnosis was acute-on-CKD due to sepsis. Massive pleural effusion was found and CT scan revealed bilateral lower lobe consolidation, pneumonia and massive ascites. Exudative effusion with atypical cells were found in both pleural effusion and ascites, and both fluid were poorly response to convention therapy.
For allograft failure, he resumed peritoneal dialysis. Due to Tenckhoff catheter dysfunction, laparoscopy was performed, revealing extensive fibrinous occlusion. Excisional biopsy of the intra-abdominal soft tissue and PET scan confirmed HHV-8-positive PEL without evidence of nodal or extranodal involvement (Lugano stage IE). The patient received IVIG (5g) and oral prednisolone (30mg/day). Chemotherapy was initially planned but was deferred due to recurrent sepsis and frailty. He is now under supportive care.
This case highlights the diagnostic challenge of PEL in transplant recipients. PEL is a rare, aggressive B-cell non-Hodgkin lymphoma primarily driven by Human Herpesvirus-8 (HHV-8) in immunocompromised patients. Given the rarity of the disease, there is no established standard of care. Despite intensive chemotherapy, the prognosis remains poor, making early diagnosis critical.

**關鍵字:** 腎移植，免疫抑制，積液，原發性積液淋巴瘤，人類皰疹病毒第八型
**Keywords:** kidney transplant, immunocompromise, effusion, primary effusion lymphoma, HHV-8

# 病例報告 2

2-3

## 肺移植後受贈者發生原生腎 BK 病毒性腎病變：一例罕見病例報告與文獻回顧 Native Kidney BK Virus Nephropathy After Lung Transplantation: A Rare Case Report and Literature Review

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BK polyomavirus (BKPyV) DNAemia and BK virus nephropathy (BKVN) are significant yet under-recognized complications in non-renal solid organ transplant recipients. In lung transplant recipients (LTRs), the incidence of BKPyV is approximately 6%, which can lead to progressive native kidney injury and an increased risk of end-stage kidney disease (ESKD). We report a rare case of native kidney BKVN in an LTR and discuss the clinical challenges in diagnosis and management.

A 59-year-old male with severe COPD underwent bilateral lung transplantation in March 2023. Starting in December 2024, the patient experienced a progressive decline in renal function, with serum creatinine rising from 1.39 mg/dL to 2.10 mg/dL by October 2025. Diagnostic workup included serum and urine BK virus DNA quantification and a native kidney biopsy.

High-level BK virus replication was detected (Urine BKV DNA >10^8 IU/mL; Blood BKV DNA 382,000 IU/mL). Kidney biopsy revealed acute tubular injury with polyomavirus-specific cytopathic effects, and immunohistochemical staining for SV40 T-antigen was strongly positive (>10%), confirming BKVN. Management involved reducing immunosuppression, including the discontinuation of mycophenolate and a reduction in tacrolimus dosage. During the follow-up period, the patient's BKV viral load showed a decline, while his lung allograft function remained stable.

This case illustrates that BKVN is a critical cause of native kidney injury in LTRs. Literature shows that high-level BKPyV (>10,000 copies/mL) is strongly associated with the subsequent development of ESKD. A careful reduction in immunosuppression to treat BKVN can be achieved without a significant decline in lung allograft function. Early surveillance of BKV DNA levels is recommended for LTRs with unexplained renal impairment to preserve native kidney function and improve long-term outcomes.

**關鍵字**：BK 病毒性腎病變；肺移植；原生腎功能；免疫抑制；SV40 T 抗原
**Key words**：BK virus nephropathy (BKVN); Lung transplantation; Native kidney function; Immunosuppression; SV40 T-antigen.

# 病例報告 2

2-4

## 腎小球毛細血管內脂蛋白血栓：一名十四歲大女童的脂蛋白腎小球病早期表現

## Lipoprotein thrombi within the glomerular capillaries: an early presentation of lipoprotein glomerulopathy in a 14-year-old girl

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A previously healthy 14-year-old girl presented with persistent proteinuria detected during a school screening. Renal ultrasonography showed no abnormalities. Laboratory evaluation, including BUN (12 mg/dL), creatinine (0.5 mg/dL), C3, C4, ANA, IgA, and ASLO, was within normal limits. The patient had a notable family history: her maternal grandfather, aunt, and uncle had all developed renal disease requiring hemodialysis.

Despite treatment with Cabudan, the urine protein-to-creatinine ratio (UPCR) fluctuated between 0.228 and 1.705 g/g. A renal biopsy was performed in August 2025. Light microscopy demonstrated focal segmental sclerosis with mild mesangial proliferation and dilated capillary lumina containing pale intraluminal material. Immunofluorescence revealed diffuse mesangial positivity for IgM and C1q. Electron microscopy showed podocyte foot process effacement and variably sized lipid-rich granules and microbubbles within the glomerular capillary lumina, without evidence of immune complex deposition. Although the initial interpretation suggested IgM nephropathy with early focal segmental glomerulosclerosis (FSGS) changes, Oil Red O staining identified these deposits as lipoprotein thrombi, thereby confirming the diagnosis of lipoprotein glomerulopathy (LPG).

Genetic testing identified a heterozygous APOE variant, NM_000041.4(APOE):c.480_488del (Exon 4), corresponding to an in-frame deletion of three amino acids (p.Leu159_Arg161del) within the receptor-binding domain of apolipoprotein E. This 9–base pair deletion results in the loss of one LRK (Leu–Arg–Lys) repeat. The variant does not correspond to previously reported LPG-associated APOE mutations, such as Kyoto or Sendai, nor to the classic E1 deletion (del156–173), and likely represents a novel APOE variant not previously reported in LPG.

Lipoprotein glomerulopathy is a rare inherited kidney disease characterized by lipoprotein thrombi within dilated glomerular capillary lumina. Mutations in the APOE gene alter the structure of apolipoprotein E (ApoE) and its receptor-binding ability, leading to lipoprotein aggregation. Conventional immunosuppressive therapy is usually ineffective, making lipid-lowering therapy the mainstay of treatment.

Although hyperlipidemia was not initially evident, later tests showed increased total cholesterol (215–307 mg/dL) and triglycerides (121–159 mg/dL). After a brief loss to follow-up, the patient returned with bilateral lower extremity edema. Treatment was adjusted to tapered corticosteroids and lipid-lowering therapy (rosuvastatin and fenofibrate), resulting in marked improvement in proteinuria, with the UPCR decreasing to 0.072 g/g.

This case highlights LPG as a crucial differential diagnosis in pediatric patients with persistent proteinuria and poor steroid response. Renal biopsy remains essential for establishing the diagnosis, even in the absence of hyperlipidemia at presentation.

**Key words:** Lipoprotein glomerulopathy (LPG), pediatric proteinuria, delayed hyperlipidemia.

# 交通資訊

**嘉義長庚紀念醫院(嘉義縣朴子市嘉朴路西段 8 號)**

**1. 搭乘高鐵（嘉義站）**

- **BRT 接駁車**：至高鐵站 2 號出口搭乘 BRT(7211)往朴子路線，於「長庚醫院站」下車。
- **搭計程車至長庚醫院**。

**2. 搭乘台鐵（嘉義火車站）**

- **前站**：搭乘嘉義縣公車「往朴子路線」(7205、7209)於「長庚醫院站」下車。
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**4.停車資訊**

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