台灣腎臟醫學會

111 年度春季學術演講會

Taiwan Society of Nephrology
2022 Spring Academic Conference

節目手冊

Program Book

時間：111 年 4 月 17 日(星期日)

地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心
(高雄市三民區十全一路 100 號)

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Experience the freedom
感受自由*

新一代2合1可溶性雙胰島素
同時提供基礎及餐後血糖控制[2-5]

Ryzodeg® FlexTouch®
健保給付自2020年11月1日起生效

*可彈性調整於正餐時給藥的時間[1]

本圖片並非真實病人

**感受全新的 Ryzodeg® 諾胰得
您與您的患者，準備好了嗎?**

Ryzodeg [簡易仿單]
諾胰得 諾特筆®
本藥限由醫師使用
100 U/ml
衛部菌疫輸字第001053號
注射液劑

仿單資訊摘要
適應症
適用於一歲以上罹患糖尿病病人，以改善血糖控制。使用限制: 不建議用於治療糖尿病酮酸中毒。不建議用於需要小於5個單位Ryzodeg®的兒童病人。
用法用量
一般給藥說明
對於成人病人，每天1次或2次於任何正餐時皮下注射Ryzodeg®；對於兒童及青少年病人，每天 1 次於任何正餐時。根據病人個人的代謝需求、血糖監測結果及血糖控制目標設計並調整Ryzodeg®的劑量。根據早餐前(空腹)血糖測量值調整Ryzodeg®劑量。建議每次增加劑量應間隔3至4天的時間。可能需要隨著體能活動、用餐型態(亦即巨量營養素含量或食物攝取的時間)、腎臟或肝臟功能的改變、或在急性疾病期間調整劑量，以降低低血糖或高血糖的發生風險。若忘記注射一劑Ryzodeg®，應於當天下一次正餐時注射，並於之後恢復給藥時程，無須注射額外劑量以補足忘記注射之劑量。在其他正餐時視需要投予速效型或短效型胰島素。第1型糖尿病人在不投予Ryzodeg®的其他正餐時，通常需要使用速效型或短效型胰島素，以達到最佳血糖控制。
用於不曾接受胰島素治療病人的起始劑量
在不曾接受胰島素治療的第1型糖尿病病人，建議的Ryzodeg®起始劑量約為每日胰島素總劑量的1/3至1/2，其餘的每日胰島素總劑量則應投予短效型或速效型胰島素，並於每日三餐分次投予。一般情況下，可使用每公斤體重0.2至0.4單位胰島素來計算初始治療的每日胰島素總劑量。在不曾接受胰島素治療的第2型糖尿病人，建議的Ryzodeg®起始劑量為10單位每天1次。
用於已接受基礎(basal)、預混(premix)或自混(self-mix)胰島素每日1至2次(單獨治療或每日多次注射之療程的一部分)之第1型或第2型糖尿病病人的起始劑量
第1型與第2型糖尿病之成人病人
對於從僅含基礎胰島素每天1次或2次單獨治療療程轉換治療的第2型糖尿病病人，以相同劑量單位數和注射時程開始Ryzodeg®治療。對於從基礎胰島素每天1次轉換為Ryzodeg®每天1次的病人，由於Ryzodeg®含速效型胰島素成分，應於開始治療後監測血糖。對於從含基礎胰島素及餐時使用短效型或速效型胰島素之每天多次注射療程轉換治療的病人，於正餐時使用與基礎胰島素相同的劑量單位數開始Ryzodeg®治療。在不注射Ryzodeg®的正餐時，繼續使用相同劑量的短效型或速效型胰島素。對於使用預混或自混胰島素的病人，則使用與預混或自混胰島素相同的劑量單位數和注射時程開始Ryzodeg®治療。同時於餐時使用短效型或速效型胰島素的病人，在不注射Ryzodeg®的正餐時繼續使用相同劑量的短效型或速效型胰島素。
第1型與第2型糖尿病之兒童(一歲以上)病人
對於使用基礎胰島素的病人，以長效型或中效型胰島素每日總劑量80%的劑量單位數開始Ryzodeg®治療，以避免低血糖風險。每天1次併正餐使用，同時於餐時使用短效型或速效型胰島素的病人，在不注射Ryzodeg®的正餐時繼續使用相同劑量的短效型或速效型胰島素。對於使用預混或自混胰島素的病人，則以混合型胰島素每日總劑量的80%劑量單位數開始Ryzodeg®之治療，以避免低血糖風險。每天1次併正餐使用。同時於餐時使用短效型或速效型胰島素的病人，在不注射Ryzodeg®的正餐時繼續使用相同劑量的短效型或速效型胰島素。
禁忌症
低血糖發作時，對Ryzodeg®或其任一種賦形劑過敏的病人。
警語與注意事項
病人間切勿共用Ryzodeg®FlexTouch注射筆。胰島素、製造商、類型或投藥方法改變時，可能會影響血糖控制，而容易發生低血糖或高血糖。應謹慎進行此類改變，並且必須在醫療監督下進行，也應提高血糖監測的頻率。對於第2型糖尿病病人，可能需要調整併用的口服抗糖尿病治療。從其他胰島素治療轉換為Ryzodeg®治療時，請遵循用藥建議。低血糖為胰島素(包括Ryzodeg®)最常見的不良反應。Ryzodeg®或任何胰島素均不應於低血糖發作時使用。低血糖可能突然發生，且症狀存在個體差異；在長期患有糖尿病的病人、患有糖尿病神經疾病的病人、使用藥物阻斷交感神經系統(如β阻斷劑)的病人、或是反覆發生低血糖的病人中，對低血糖的症狀意識可能較不顯著。注射後低血糖的風險與胰島素作用的持續時間相關，通常在胰島素的降血糖效果最大時風險也最高。腎功能不全或肝功能不全的病人、低血糖的風險可能較高。為了避免Ryzodeg® 與其他胰島素間的用錯失誤，請指示病人在每次注射前務必查看胰島素的標籤。Ryzodeg® 禁用於對 insulin degludec、insulin aspart或任一種賦形劑曾發生過敏反應的病人。在有低血鉀症風險的病人應監測鉀濃度。併用PPAR-γ促進劑時，可能導致劑量相關的液體滯留而引發充血性心臟衰竭或使其惡化。應觀察接受胰島素(包括Ryzodeg®)及PPAR-γ促進劑治療的病人是否出現充血性心臟衰竭的徵兆和症狀。
不良反應
使用Ryzodeg®的病人可能會出現低血糖 (包括用藥失誤引起的低血糖)、注射部位反應、脂肪代謝障礙、過敏與過敏反應及低血鉀症。低血糖與低血鉀症請參照上述警語與注意事項。胰島素藥品(包括Ryzodeg®)治療中可能發生重度、危及生命、全身性的過敏，包括全身過敏性反應(anaphylaxis)。若發生過敏反應，請立即停用Ryzodeg®並就醫。依標準照護治療並監測病人至症狀緩解為止。臨床試驗中觀察到的不良反應包括鼻咽炎、頭痛、上呼吸道感染、流感、低血糖過敏反應、注射部位反應、 體重增加、 周邊水腫、出現 Insulin aspart 抗體 、出現 Insulin degludec抗體，詳情請參考衛福部核准仿單。
藥物交互作用
請參考衛福部核准仿單。
特殊族群使用
目前並無Ryzodeg®用於懷孕女性中的資料說明藥物相關的重大先天缺陷或流產風險。目前並無關於Ryzodeg®或insulin degludec會分泌到人類乳汁中、對被哺乳的嬰兒、或對於泌乳影響的資料。 無足夠資料以了解insulin aspart 對泌乳及哺乳嬰兒的影響 。尚未確立 Ryzodeg® 在一歲以下的兒童控制血糖的安全性與有效性。臨床試驗資料顯示年齡逾65歲受試者與年輕受試者的安族群分析並無安全性或療效的差異。然而，為老年病人投予Ryzodeg®時仍應謹慎以避免低血糖。如同所有的胰島素藥品，在肝、腎功能不全病人中應加強血糖監測，並根據個人需求調整劑量。
用藥過量
當使用過量胰島素時，可能導致重度並且有時為長期且危及生命的低血糖和低血鉀症，過量處理請參考衛福部核准仿單。
建議儲存方式
未使用的Ryzodeg®注射筆應儲存於冰箱(2°C至8°C)。請勿冷凍。若經冷凍則請勿使用。若儲存於冰箱、未開封的注射筆可使用至保存期限，使用中的注射筆應冷藏(2°C至8°C)或保存於室溫環境(低於30°C)至28天(4週)，並遠離熱源及避光。
使用前請詳閱衛福部核准仿單

References:
1. 衛福部核准仿單 2. Vijan S, et al. J gen Intern Med. 2005;20(5):479-482. 3. Fulcher G, et al. Diabetes Care. 2014;37(8):2084-2090. 4. Haahr H, et al. Clin Pharmacokinet. 2017;56(4):339-354. 5. IQVIA MIDAS® data. January 2020. 6. Kumar A, et al. Int J Clin Pract. 2016;70(8):657-667. 7. Philis-Tsimikas A, et al. Diabetes Res Clin Pract. 2019;147:157-165. 8. Onishi Y, et al. Diabetes Obes Metab. 2013;15(9):826-832.

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70% insulin degludec and 30% insulin aspart
[rDNA origin] injection

台灣腎臟醫學會
111 年度春季學術演講會

目錄

台灣腎臟醫學會 111 年度春季學術演講會

會員報到、教育積分注意事項

因應疫情，進入學校請先實名制登記。

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。

醫師會員報到

時間：111 年 4 月 17 日(星期日)上午 8:30 至下午 2:30

地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心報到處

積分認定

✓腎臟醫學會積分：A類15分

✓內科醫學會積分：B類10分

透析護理人員及腎臟照護衛教師報到

時間：111 年 4 月 17 日(星期日)上午 8:30 至下午 2:30

地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心報到處

注意事項：請攜帶身份證刷卡報到，不需刷退

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

參展廠商攤位展示區

時間：111 年 4 月 17 日(星期日)

地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心走廊區

午餐資訊

時間：111 年 4 月 17 日(星期日)中午 12:00 至下午 1:30【報到時領取餐券】

地點：高雄醫學大學①國際學術研究大樓 B2 國際會議中心電梯出口右側

②勵學大樓 A1 講堂、A2 講堂門口

【憑券領取餐盒】

停車資訊

同盟路路口地下停車場

✓由同盟路車道入口進出

✓免費

因應嚴重特殊傳染性肺炎(Covid-19)，與會者請自備口罩並全程配戴，若出現發燒、咳嗽或急性呼吸道症狀，應儘速就醫後在家休養，避免參加集會活動。

台灣腎臟醫學會
111 年度春季學術演講會
議程一覽表

時間：111 年 4 月 17 日(星期日)
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心
(高雄市三民區十全一路 100 號) ➔**請由同盟路正門進入**

日期		111 年 4 月 17 日(星期日)			
時間＼會場		會議室 A	會議室 B	會議室 C	走廊區
上午	08:30 14:30	報到處：國際學術研究大樓 B2 國際會議中心(08:30-14:30)			
	09:00 10:30	【專題演講 1】 Covid-19 疫苗施打在各族群之可能副作用	【專題演講 4】 腎性貧血治療之更新	【專題演講 7】 急性腎損傷生物標記	廠商展示區
	10:30 12:00	【專題演講 2】 疫情後的浴火重生	【專題演講 5】 阻斷急性腎損傷—急性腎疾病—慢性腎臟病發展進程：從精準醫學至醫療政策	【專題演講 8】 單株抗體於腎臟領域之應用 (10:30-12:10)	
中午	12:20 13:10	【Lunch Symposium 1 及 2】 會場移至勵學大樓 A1 及 A2 講堂		Lunch Symposium 3 華安及吉立亞	
		Lunch Symposium 1 諾華 勵學大樓 A1 講堂	Lunch Symposium 2 阿斯特捷利康 勵學大樓 A2 講堂		
下午	13:30 14:20	【專題演講 3】 鈉-葡萄糖共同轉運器-2 抑制劑之心腎糖對話	Industry Lecture 費森尤斯卡比	【教育演講】 國際腎臟病防治的田野行動 (13:30-14:00)	
	14:20 15:00		【專題演講 6】 腎臟介入性技術建立執行及優化經驗分享	【病例報告 2】 (14:00-15:48)	
	15:00 15:50	【病例報告 1】 (15:00-15:48)			

➲ 春季學術演講會摘要不再印製紙本
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台灣腎臟醫學會 111 年度春季學術演講會

台灣腎臟醫學會
111 年度春季學術演講會
節目表

時 間：111 年 4 月 17 日(星期日)
地 點：高雄醫學大學國際學術研究大樓 B2 國際會議中心

專題演講 1

時間：111 年 4 月 17 日(星期日) 09:00-10:30
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心會議室 A

Covid-19 疫苗施打在各族群之可能副作用
The possible side effect of Covid-19 in different populations

主持人：田亞中 陳呈旭

09:00-09:30 Covid-19 疫苗施打在透析腎友之可能副作用
The possible side effect of Covid-19 in dialysis population
陳冠興 醫師
林口長庚醫院 腎臟科

09:30-10:00 Covid-19 疫苗施打在慢性疾病患者可能副作用
The possible side effect of Covid-19 in patients with chronic disease
洪思群 醫師
台北慈濟醫院 腎臟科

10:00-10:30 Covid-19 疫苗施打在移植受贈者可能副作用
The possible side effect of Covid-19 in transplant recipients
鍾牧圻 醫師
臺中榮民總醫院 腎臟科

專題演講 2

時間：111 年 4 月 17 日(星期日) 10:30-12:00
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心會議室 A

疫情後的浴火重生
How we survived the pandemic?

主持人：吳志仁 邱怡文

10:30-11:00 血液透析感染管制到導入先驅研究報告
盧柏樑 院長
高雄醫學大學附設醫院

11:00-11:30 血液透析室如何因應 Covid 19
吳培甄 醫師
馬偕紀念醫院 腎臟科

11:30-12:00 腹膜透析室如何因應 Covid 19
陳怡婷 醫師
臺大醫院 綜合診療部血液淨化科

台灣腎臟醫學會 111 年度春季學術演講會

專題演講 3

時間：111 年 4 月 17 日(星期日) 1330-1500
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心會議室 A

鈉-葡萄糖共同轉運器-2 抑制劑之心腎糖對話
Cross Talk among Cardio-Kidney-Diabetes (CKD) Through SGLT2 inhibitor

13:30 - 13:35 Opening 開幕致詞
姜至剛 醫師
台大醫院 腎臟科

主持人：陳思嘉
13:35 - 14:00 SGLT2 inhibitors for kidney protection: diabetic kidney disease and beyond
何立鈞 醫師
義大醫院 腎臟科

主持人：黃尚志
14:00 - 14:25 T2D caring- Move from conventional to comprehensive
李美月 醫師
高雄醫學大學附設醫院 內分泌科

主持人：鄭正一
14:25 - 14:50 SGLT2 Inhibitors and Heart Failure in Cardiorenal Spectrum
鍾昇穎 醫師
高雄長庚醫院 心臟科

14:50-15:00 討論及閉幕致詞
Discussion and Closing Remarks
姜至剛 醫師
台大醫院 腎臟科

專題演講 4

時間：111 年 4 月 17 日(星期日) 09:00-10:30
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心會議室 B

腎性貧血治療之更新
Update on the Treatment of Renal Anemia

主持人：吳明儒 張哲銘

09:00-09:30 Overview the treatment guideline and unmet needs of renal anemia.
邱怡文 醫師
高雄醫學大學附設醫院 腎臟科

09:30-10:00 The emerging treatment of renal anemia: HIF stabilizer.
蔡尚峰 醫師
臺中榮民總醫院 腎臟科

10:00-10:30 Recent and emerging therapies for iron deficiency in anemia of CKD
吳家麟 醫師
彰化基督教醫院 腎臟科

台灣腎臟醫學會 111 年度春季學術演講會

專題演講 5

時間：111 年 4 月 17 日(星期日) 10:30-12:00
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心會議室 B

阻斷急性腎損傷—急性腎疾病—慢性腎臟病發展進程：從精準醫學至醫療政策
Stop AKI-AKD-CKD continuum in Taiwan: From precision medicine to policy decision

主持人：陳金順 洪士元 李建德

10:30-10:35 Opening remark
陳金順 醫師
高雄榮民總醫院 腎臟科

10:35-10:55 Delineating AKI-AKD-CKD continuum in Taiwan
吳麥斯 醫師
衛生福利部雙和醫院 腎臟科

10:55-11:15 Intensified AKD care to reduce CKD
陳佑瑋 醫師
衛生福利部雙和醫院 腎臟科

11:15-11:35 Epidemiological perspectives on first dialysis
廖家德 醫師
衛生福利部雙和醫院 腎臟科

11:35-11:55 Role of genetics and immunity in AKI-AKD-CKD continuum
吳美儀 醫師
衛生福利部雙和醫院 腎臟科

11:55-12:00 Closing remark
李建德 醫師
高雄長庚醫院 腎臟科

專題演講 6

時間：111 年 4 月 17 日(星期日) 14:20-15:50
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心會議室 B

腎臟介入性技術建立執行及優化經驗分享
Experience Sharing: how to establish, practice and refine for interventional nephrology in Taiwan

14:20-14:25 Opening remark
陳金順 醫師
高雄榮民總醫院 腎臟科

主持人：陳金順
14:25-14:50 腎臟穿刺
李明峰 醫師
陳信佑 醫師
高雄榮民總醫院 放射線部
高雄榮民總醫院 腎臟科

主持人：周哲毅
14:50-15:15 腹膜透析經皮植管技術執行與經驗分享
鄭本忠 醫師
高雄長庚醫院 腎臟科

主持人：方昱偉
15:15-15:40 上肢周邊血管超音波檢查與應用
吳重寬 醫師
新光紀念醫院 透析血管通路管理中心

15:40-15:50 問題與討論

台灣腎臟醫學會 111 年度春季學術演講會

專題演講 7

時間：111 年 4 月 17 日(星期日) 09:00-10:30
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心會議室 C

急性腎損傷生物標記
Acute Kidney Injury (AKI) Biomarkers

主持人：陳永昌 張志宗

09:00-09:20 急性腎損傷生物標記綜論
Overview Biomarkers for AKI
黃俊德 醫師
臺中榮民總醫院 重症醫學部

09:20-09:40 肝型脂肪酸結合蛋白在急性腎衰竭的臨床應用
Clinical use of urinary liver-type fatty acid binding protein in acute kidney injury
洪啓智 醫師
高雄醫學大學附設醫院 腎臟科

09:40-10:00 嗜中性白血球明膠酶相關運載蛋白
Neutrophil Gelatinase-Associated Lipocalin (NGAL)
張智翔 醫師
林口長庚醫院 腎臟科

10:00-10:20 胱蛋白 C
Cystatin C (CysC)
黃道民 醫師
臺大醫院 腎臟科

10:20-10:30 問題與討論

專題演講 8

時間：111 年 4 月 17 日(星期日) 10:30-12:10
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心會議室 C

單株抗體於腎臟領域之應用
The coming era of monoclonal antibody use in nephrology field

主持人：許永和 張浤榮

10:30-10:55 Overview of therapeutic monoclonal antibodies
黃道民 醫師
臺大醫院 腎臟科

10:55-11:20 Use of monoclonal antibodies in primary glomerulonephritis
塗昆樺 醫師
林口長庚醫院 腎臟科

11:20-11:45 Use of monoclonal antibodies in immune related kidney diseases
楊皇煜 醫師
林口長庚醫院 腎臟科

11:45-12:10 Use of monoclonal antibodies in renal transplantation
游棟閔 醫師
臺中榮民總醫院 腎臟科

台灣腎臟醫學會 111 年度春季學術演講會

教育演講

時間：111 年 4 月 17 日(星期日) 13:30-14:00
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心會議室 C

主持人：彭渝森

13:30-14:00 國際腎臟病防治的田野行動
林建璋 計畫經理
財團法人國際合作發展基金會

Lunch Symposium 1、2

時間：111 年 4 月 17 日(星期日) 12:20-13:10
地點：高雄醫學大學勵學大樓

【Lunch symposium 1】 地點：A1 講堂

主持人：黃尚志

Burden and challenge for heart failure patients with kidney dysfunction
Nephrologists point of view for Heart Failure Treatment
姜至剛 醫師
臺大醫院 腎臟科

*本節目由台灣諾華股份有限公司贊助

【Lunch symposium 2】 地點：A2 講堂

主持人：李建德

Revolutionize CKD Therapy - Delay Dialysis and Reduce Mortality
蔡宜純 醫師
高雄醫學大學附設醫院 腎臟科

*本節目由臺灣阿斯特捷利康股份有限公司贊助

Lunch Symposium 3

時間：111 年 4 月 17 日(星期日) 12:20-13:10
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心

【Lunch symposium 3】 地點：會議室 C

主持人：宋俊明

如何治療及照護腎臟病 C 肝病患?
戴嘉言 醫師
高雄醫學大學附設醫院 肝膽胰內科

*本節目由華安藥品股份有限公司及香港商吉立亞醫藥有限公司台灣分公司贊助

台灣腎臟醫學會 111 年度春季學術演講會

Industry Lecture

時間：111 年 4 月 17 日(星期日) 13:30-14:20
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心

【Industry Lecture】 地點：會議室 B

主持人：吳明儒

The Role of Carbamylation in Patients with Kidney Disease
林祐賢 醫師
高雄市立大同醫院(委託高醫經營) 腎臟科

*本節目由台灣費森尤斯卡比股份有限公司贊助

病例報告 1

時間：15:00~15:48
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心會議室 A

【病例報告 1】　主持人：張敏育　王曦澔

15:00 － 15:12　1.以惡性高血壓表現的 41 歲女性，最終診斷為腎素分泌瘤
A 41-year-old female patient, with initial presentation of malignant hypertension, diagnosed to be renin secreting tumor : A case report
周子巽　顏銘佐
國泰綜合醫院腎臟內科

15:12 － 15:24　2.引起陣發型低血鈉及低血鉀的罕見原因
A rare cause of episodic hyponatremia and hypokalemia
鄭子明[1]　許智揚[1]
[1]高雄榮民總醫院內科部腎臟科

15:24 － 15:36　3.腎臟移植術後一週內併發急性排斥反應
An allograft kidney recipient complicated with active antibody mediated rejection after transplantation in one week
陳怡雯[1]　周康茹[1]
高雄榮民總醫院腎臟科[1]

15:36 － 15:48　4.產婦急性腎損傷及血小板低下症:非典型溶血性尿毒症
Pregnancy related Atypical Hemolytic Uremic Syndrome Successfully Treated by Eculizumab
羅翊中[1]　陳信佑[1]
[1]高雄榮民總醫院內科部腎臟科

台灣腎臟醫學會 111 年度春季學術演講會

病例報告 2

時間：14:00~15:48
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心會議室 C

【病例報告 2】　主持人：李柏蒼　吳建興

14:00 － 14:12　1.陣發性夜間血紅素尿症病人：可逆之急性腎臟病
Reversable acute kidney disease in a patient with paroxysmal nocturnal hemoglobinuria
于志業[1]　林維洲[2]　陳永銘[1]　黃道民[1]
[1]台灣大學附設醫院內科部腎臟科　[2]台灣大學附設醫院病理部

14:12 － 14:24　2. Everolimus 所致肺炎在腎移植患者
A Rapidly developed Everolimus-induced Pneumonitis in Renal Transplant Recipient
邵月珠　甄沛勤　陳錫賢
台北醫學大學附設醫院內科部腎臟內科

14:24 － 14:36　3. NK/T 細胞淋巴癌罕見的腎臟侵犯
NK/T-cell Lymphoma Invading Kidney Mimicking Adult Onset Still's Disease
江哲甫[1]　巫宏傑[1]　王偉傑[1]　丁瑞聰[1]　陳冬英[2]
[1]衛生福利部桃園醫院內科腎臟科　[2]台北馬偕紀念醫院病理科

14:36 － 14:48　4.腎臟移植受贈者之移植耐受不良症候群及切除
Graft intolerance syndrome with graft nephrectomy in a kidney transplant recipient
劉家佑　徐愷翔
亞東紀念醫院腎臟內科

14:48 － 15:00　5.血液透析治療丙戊酸中毒
Hemodialysis treatment for valproic acid poisoning
許郡珈　楊如燁　徐世平　彭渝森
亞東紀念醫院腎臟內科

15:00 － 15:12　6.於高端疫苗接種後之 A 型免疫球蛋白腎病變: 個案報告及文獻回顧
De novo IgA nephropathy following Medigen vaccination: A case report and literature review
彭梓晏[1]　董奎廷[1]　徐世平[1]　彭渝森[1]
亞東紀念醫院腎臟內科

病例報告 2

時間：14:00~15:48
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心會議室 C

15:12 – 15:24 7.腎病症候群合併雙側腎靜脈栓塞之案例報告
Nephrotic Syndrome Complicated with bilateral renal vein thrombosis: A case report
周奕伶 彭聖曾
國泰綜合醫院腎臟內科

15:24 – 15:36 8. Exon 23 c.3572C>T/c.3590 T>C 與 exon 19 c. 2808G>T 突變導致的補體介導栓塞性微血管病變
Complement-mediated thrombotic microangiopathy caused by combined exon 23 c.3572C>T/c.3590T>C, and exon 19 c. 2808G>T mutation
林煒棣[1] 簡志強[1]
[1]奇美醫學中心內科部腎臟科

15:36 – 15:48 9.一位因大腸癌而服用 Afatinib 及曾經因為急性肺栓塞使用 Edoxaban 的病人，發生急性腎衰竭、蛋白尿、血尿被診斷為 IgA 腎病變
A male with history of colon cancer with Afatinib treatment and acute pulmonary embolism under Edoxaban control, happened with acute kidney injury, proteinuria and hematuria, was diagnosed with IgA nephropathy.
李璇廷 陳銳溢
奇美醫學中心內科部腎臟科

台灣腎臟醫學會 111 年度春季學術演講會

專題演講 1

Covid-19 疫苗施打在透析腎友之可能副作用
The possible side effects of Covid-19 vaccination in dialysis population

陳冠興
林口長庚醫院 腎臟科

Covid-19 pandemic outbreak since 2019. Patients with dialysis belong to high-risk group of Covid-19 infection. Vaccination is one of most powerful manipulation to stop Covid-19 transmission and to reduce the morbidity and mortality of infected patients. However, the vaccination efficacy of Covid-19 vaccine is relatively poor in dialysis patients due to immunocompromised status. And some possible side effects related to Covid-19 vaccination have been reported in recent literatures. In this talk, we will review the latest studies about the morbidity and mortality in dialysis patients with Covid-19 infection, and the humoral or cellular immune response of different Covid-19 vaccination in dialysis patients. Furthermore, we will discuss about the possible side effects of Covid-19 vaccination in dialysis patients. A few patients with hemodialysis (HD) encountered severe complications post Covid-19 vaccination will be reported. In addition, in LinKou and Taoyuan branch of Chang Gung Memorial Hospital, we enrolled about 480 HD patients to investigate the humoral response post Covid-19 vaccination since 2021.10. when vaccination started. Preliminary data about titer of neutralizing antibody post vaccination will also be discussed. Through this talk, we hope to offer the information about pros and cons of Covid-19 vaccination in dialysis patients.

專題演講 1

Covid-19 疫苗施打在慢性疾病患者可能副作用
The possible side effect of Covid-19 vaccines in patients with chronic disease

洪思群
台北慈濟醫院 腎臟科

Patients with chronic disease have a high risk of infection with SARS-CoV-2 and worse clinical outcomes than the general population. Although vaccination is associated with a lower risk of COVID-19 infection and lower risk of hospitalization or death, vaccine response is usually attenuated in patients with chronic disease due to accelerated immunosenescence induced by chronic inflammation. In addition, a significant proportion of patients with chronic disease are hesitant about seeking COVID-19 vaccination. Willingness to get vaccinated against COVID-19 in these patients is limited by the fear of adverse effects. There have been widespread speculations of cardiovascular and other adverse events associated with COVID-19 vaccines, such as thromboembolism, neuropathy, and myocarditis following the administration of specific vaccines. The safety of COVID-19 vaccines is of great public health concern and is critical to reducing vaccine hesitancy during the pandemic. However, there is a paucity of data regarding the safety and efficacy of COVID-19 vaccines in patients with chronic disease because these patients have largely been excluded from clinical trials. Today's talk will focus on immune response to COVID-19 vaccines among patients with chronic disease and whether comorbidities impose extra risks of adverse events following COVID-19 vaccination.

台灣腎臟醫學會 111 年度春季學術演講會

專題演講 1

Covid-19 疫苗施打在移植受贈者可能副作用
The possible side effect of Covid-19 in transplant recipients

鍾牧圻
臺中榮民總醫院 腎臟科

腎臟移植受贈者因為服用免疫抑制藥物，在疫情下更容易惡化成重症，因此國內外指引均建議應該盡早施打疫苗，以建立對 COVID-19 的抵抗力。我們必須注意注射疫苗的時間點，才能期待產生安全且有效的保護力。此次演講會針對疫苗可能對於腎臟移植受贈者產生的潛在副作用探討並釐清可能的狀況。

專題演講 2

血液透析感染管制到導入先驅研究報告

盧柏樑 院長
高雄醫學大學附設醫院

臺灣每年約有6-7萬人進行血液透析，血液透析的病人因為侵入性醫療處置、免疫防禦能力下降及治療照護過程中其醫療照護人員頻繁的接觸等多重因素，特別容易成為醫療照護相關感染（healthcare-associated infections；HAI）的高危險性族群之一。所以為減少病人發生血流感染與其他感染，透析單位制訂醫療照護感染管制之相關流程及措施對維護病人健康是必要的作為。

疾病管制署期由台灣腎臟醫學會與台灣感染管制學會的合作，一同探討血液透析單位導入感染管制自我評核機制，藉由結合臨床透析、感染管制、醫療品質等專業團體，建立專業領域交流平台，提升從業人員的專業能力和整體品質，並導入自我評核制度，提升病人安全，降低醫療照護相關感染，參考國際間有關之實證與指引，建置血液透析單位有效之感染管制作業管理模式。

110 年透過參與醫療院所，調查血液透析相關醫療機構感染管制執行狀況，包含人員健康管理-工作人員、感染管制措施-環境、隔離措施、標準防護措施及其他防護措施，瞭解血液透析相關機構感染管制執行及認知狀態評估，藉由舉辦分區說明會，說明並推廣感染管制措施在血液透析醫療院所之重要性，以推動血液透析醫療院所感染管制品管活動，並經由實地輔導訪視 30 家醫療院所，發現血液透析相關醫療院所感染管制相關問題：需隔離照護病人，無法專區、專人照護；班與班之間的消毒，在少部分院所未落實執行；部分院所的各項疫苗接種措施宜再規劃改進。這些可做為未來血液透析相關醫療院所接受感染管制教育訓練、相關政策參考依據與未來規劃。經過實地訪視與分析檢討後，將使自我評核表修訂的更為實用。

台灣腎臟醫學會 111 年度春季學術演講會

專題演講 2

血液透析室如何因應 Covid 19

吳培甄
馬偕紀念醫院 腎臟科

COVID-19 is a very high transmission disease with a variable prognosis in the general population. Patients on hemodialysis (HD) therapy are particularly vulnerable to developing an infectious disease.

An outbreak in HD centers can cause staff shortages and thus puts a strain on health professionals and technicians that run these facilities. This can lead to increased waiting time for dialysis, or otherwise, patients who skip their dialysis shifts due to fear from the pandemic, putting them at risk of fluid overload and metabolic emergencies. Resource scarcity is compounded by a general shortage of medical equipment and personal protective equipment (PPE) during a pandemic. Therefore, the protection of staff working in dialysis units is very important.

The ability of a dialysis center to provide effective physical distancing for all its members is constrained by several factors including the physical layout of the unit which cannot be reconfigured without a major overhaul. Space is limited, and there is little flexibility in the scheduling to allow the following special precautions for cohorting COVID-19 infected patients. Most dialysis units operate at maximum capacity. The nature of care for these patients requires close proximity to nurses operating with the HD machine. These nurses may interact with 4 patients simultaneously. Although contacts with healthcare providers can be limited, they cannot be eliminated. Asymptomatic spread in the HD unit is another challenge. As a consequence of a relatively suppressed immune system, the proportion of asymptomatic infection in HD populations may be higher than average.

To mitigate the impact of COVID-19 in HD units, we should focus on three main areas including dialysis facilities, transportation, and patients' communities. Structural and organizational changes adopted early on and the diagnosis algorithm played a key role in minimizing the spread of the disease.

專題演講 2

腹膜透析室如何因應 Covid 19

陳怡婷
臺大醫院 綜合診療部血液淨化科

腎臟是除了肺臟以外容易受到新型冠狀病毒侵害的器官之一，國外研究顯示慢性腎臟病病患，包含透析及腎臟移植病患都是新冠病毒感染的高風險族群，更指出慢性腎臟病第四，五期及透析病患其重症風險高於糖尿病及心臟病等慢性共病。新冠疫情嚴峻當下，慢性腎臟病及透析病患照護更顯重要。

近年來腹膜透析有遠端醫療開發，在疫情時期可幫忙醫護人員透過遠端傳輸的資料了解病患在家透析情形。接種新冠疫苗也是自我保護措施之一，且因透析病患屬新冠病毒高感染風險族群，更需要接種新冠疫苗以減輕重症的風險。然而目前新冠疫情仍持續進行，對醫療照護的衝擊，我們又該如何因應？

台灣腎臟醫學會 111 年度春季學術演講會

專題演講 3

SGLT2 inhibitors for kidney protection: diabetic kidney disease and beyond

何立鈞
義大醫院 腎臟科

First disclosed in the EMPA-REG OUTCOME trial, the outstanding renal benefits of SGLT2 inhibitors for patients with diabetic kidney disease (DKD) were finally apporved in the CREDENCE trial taking renal events as the primary outcomes. Surprisingly, the renal protective effect of SGLT2 inhibitor revealed in the CREDENCE trial seemed to be unrelated to the hypoglycemic effect. The DAPA-CKD trial came to address this question by enrolling chronic kidney disease (CKD) patients with a wide spectrum of etiology, nearly 40% non-diabetic CKD. The primary (renal) composite outcome of DAPA-CKD favored SGLT2 inhibitor over placebo no matter in the whole study population or in the subgroup of patients with non-diabetic or IgA nephropathy. The great success of these trials has not only led to a paradigm shift in our approach and management of DKD and CKD, but also provoked the proposal of ‘tubule-centred model’, in which most pathophysiology of diabetic nephropathy is attributed to overactivated SGLT2 and maladaptive tubuloglomerular feedback (TGF). SGLT2 inhibitors restore the regulation of TGF in both type 1 and type 2 diabetes but the results are pre-glomerular vasoconstriction for the former and post-glomerular vasodilation for the latter. As for the renal protective effects of SGLT2 inhibitors for non-diabetic CKD, a reduced nephron number and hyperfiltration of the remaining nephrons mimicking the hyperfiltration of diabetic nephropathy may explain, but much more is still awaiting research. The last part of the speech will address some unresolved questions and future prospects about the role of SGLT2 inhibitors in CKD treatment.

專題演講 3

T2D caring- Move from conventional to comprehensive

李美月
高雄醫學大學附設中和紀念醫院 內分泌科

Metabolic syndrome is a cluster of conditions that occur together, increasing the risk of cardiovascular disease together with type 2 diabetes.

Cardiorenal metabolic syndrome is an umbrella term that encompasses a lot of different types of pathophysiologies. It highlights the multiple links between the kidneys and the heart as the two main organs that are involved in this process. Injury can start in one organ and transfer to the other in either direction. It's much more complicated than just hemodynamics. It also involves neurohormonal and inflammatory systems. Thus, more contemporary research and ideas surrounding the pathophysiology have started to encompass metabolic syndrome, including diabetes and lipids. They're all at interplay, causing this disease process to take place.

SGLT2 inhibitors, which the cardiovascular and renoprotective benefits evidenced with sodium–glucose cotransporter-2 (SGLT2) inhibitors make them a potential choice in the management of cardiorenal syndrome. Cardiovascular protection is mediated by a reduction in cardiac workload, blood pressure, and body weight; with improvement in lipid profile, uric acid levels, and adaptive ketogenesis process. Renoprotection is facilitated by reduction in albuminuria and hypoxic stress, and restoration of tubuloglomerular feedback. The favourable effect on cardiovascular complications and death, as well as renal complications and progression to end-stage kidney disease, has been confirmed in clinical trials. Guidelines endorse first-line use of SGLT2 inhibitors after metformin in patients with T2DM with high cardiovascular risk, chronic kidney disease or both.**

The prevalence of Cardiorenal metabolic syndrome continues to grow. We know that the various clinical conditions about here—whether it's heart failure or diabetes or kidney disease—are rising in incidence and prevalence. The overlap of these clinical conditions also continues to grow exponentially. The numbers of patients with heart failure, a quarter of them may have diabetes. If you flip it and look at it the other way, approximately 10% to 20% of patients with diabetes have heart failure. Similarly, up to a quarter of patients that are hospitalized for acute heart failure will develop acute kidney injury, or potentially cardiorenal syndrome. Let us start to care about the Cardiorenal metabolic syndrome. I believe it never too late.

台灣腎臟醫學會 111 年度春季學術演講會

專題演講 3

SGLT2 inhibitors and heart failure in cardiorenal spectrum

鍾昇穎
高雄長庚紀念醫院 心臟內科

SGLT2 inhibitor used to be anti-diabetic drugs for glucose control by blocking sodium-glucose cotransporter at proximal tubule of kidney. From a series of CVOT of SGLT2 inhibitors in diabetes patients (EMPA-REG & DECLARE & CANVUS), they can lower the incidence of hospitalization for heart failure and obvious renal protection with statistical significance. From DAPA-HF (Dapagliflozin) and EMPEROR-Reduced (Empagliflozin) trials for patients with heart failure with reduced ejection fraction with or without diabetes, the patient group with SGLT2 inhibitor has statistically significant low rates of CV death/ hospitalization for heart failure. For this reason, the international heart failure guidelines including ACC/AHA, European society of cardiology put SGLT2 inhibitor as first line fundamental guideline-directed medical therapy.

專題演講 4

Overview of the treatment guideline and unmet need for renal anemia

邱怡文
高雄醫學大學附設院 腎臟科

To maintain the hemoglobin (Hb) in CKD patients within a therapeutic range, 10-12mg/L has been recommended by international guidelines after several large RCTs reported higher thrombotic events rates when using Erthropoeitn stimulating agent (ESA) to normalize the Hb in this population. The algorithms used in KDIGO for anemia include diagnosis and evaluation, Iron therapy, ESA therapy, ESA failure, and RBC transfusion. Unfortunately, the evidence is still insufficient, and more studies are needed for optimal Hb levels in various CKD stages as well as the “overdose” or hyporesponsiveness of ESA regarding the clinical outcomes. The consensus on the ESA ceiling dose has never emerged when considering its cost, effect, and complication survey. With a more understanding of erythropoiesis and iron metabolism in CKD, we nephrologists should, considering the clinical outcomes, rethink all values used in these algorithms for decision-making. For example, when to modify ESA dose and iron supplement at which Hb level. Iron is essential for Hb synthesis and becomes more complicated in CKD. Considering its storage, uptaking, and acting together with ESA, the optimal iron status has been tested in the past decade but still without a definite conclusion. That iron or ESA, which should be the first-line treatment for anemia in CKD, is clinically determined case by case now; we need more clear individual suggestions in new guidelines. With new oral iron supplements introduced into the market, managing the iron metabolism, like Calcium-phosphorus metabolism in MBD, is crucial in renal anemia correction. Some new therapies have been incorporated into anemia correction in CKD, such as HIF stabilizers, hepcidin antagonists, and new forms of iron. Using them precisely is another concern that needs more work on it.

台灣腎臟醫學會 111 年度春季學術演講會

專題演講 4

The emerging treatment of renal anemia: HIF stabilizer.

蔡尚峰
臺中榮民總醫院 腎臟科

Renal anemia is a major problem in patients with chronic kidney disease (CKD). As the deterioration of renla function, the prevalene of anemia gets worse. Meanwhile, the anemia also causes the renal function furhter decline.

As we know, erythropoieptin (EPO) deficiency and iron dysregugulation are the two major problems of CKD related anemia. In the past, we used EPO since 1989 based on the evidence of NHCT, CHOIR, CREATE, and TREAT studies. However, more and more studies showed that increased risk of mortality and cardiovascular events with EPO at higher Hb targets in patients with CKD anemia. Similary, we used iron supplement for patients with CKD, who experienced iron dysregulation. Too much iron supplement also leads to adverse effect due to too much free radicals. We cannot treat the underlying cause of iron dysregulation (increased hepcidin) before. Nowadays, we need to face our former treatments for CKD anemia, includig EPO and iron supplement, were not good enough.

Patients with end-stage kidney disease (ESKD) living at high altitude either increase endogenous EPO production or respond better to endogenous and exogenous EPO. Altitude-induced hypoxia reduces EPO requirements in patients with ESKD with treatment refractory anemia. The above findings are assoicated with hypoxia-inducible factor (HIF). Hypoxia researchers won 2019 Nobel Prize in Physiology or Medicine. Once stabilizing HIF 2α, we will have more EPO production and less hepcidin. It can deal with two major problems of renal anemia simultaneously, which called “kill two birds with one stone”. Herein, we will review the HIF and its effect on renal anemia.

專題演講 4

Recent and emerging therapies for iron deficiency in anemia of CKD

吳家麟
彰化基督教醫院 腎臟科

慢性腎臟病是目前全球健康之重大威脅之一，多年以來，台灣的慢性腎臟病防治一直是政府與醫界努力欲改善的重點。貧血會為慢性腎臟病患者帶來許多健康上的不良影響如:降低心肺功能、加劇左心室肥大與心衰竭、降低生活品質與縮短壽命等。其中，缺鐵性貧血是腎臟病患者發生貧血發生的主因之一。慢性腎臟病患者的缺鐵性貧血會降低紅血球生成刺激劑效果，使得這些患者不易矯正貧血。本次報告主旨在回顧目前為止對於鐵劑治療在腎性貧血的最新臨床證據與提供腎性貧血治療上的建議。

台灣腎臟醫學會 111 年度春季學術演講會

專題演講 5

Delineating AKI-AKD-CKD continuum in Taiwan

吳麥斯
衛生福利部雙和醫院 腎臟科

The AKI-AKD-CKD continuum, whereby the initial acute kidney injury (AKI) subsequently become acute kidney disease (AKD) and eventually progress to chronic kidney disease (CKD), is an important global public health issue. Patients with the AKI-AKD-CKD continuum usually have increased risks of end-stage kidney disease and even death. The investigation of AKI-AKD-CKD development and spectrum is thus critical to diagnosis and prognosis of renal disease patients. Herein, our team applied an algorithm-based approach to identify patients with AKI-AKD continuum in a large AKI cohort. We found that more than half of AKI patients could be categorized as AKD patients who were characterized with different AKD stages. Further analyses revealed higher risks of major adverse kidney events, declined kidney function, dialysis and mortality in these AKD patients relative to AKI patients without AKD regardless of their baseline estimated glomerular filtration. In the study, we performed the AKI identification, followed by the AKD staging, and further indicated associations between AKD stages and adverse clinical outcomes. These findings would accelerate precision medicine and improve policy decision for the prevention of AKI-AKD-CKD development and relevant adverse clinical outcomes in the future clinical practice.

專題演講 5

強化急性腎疾病照護減少慢性腎臟病-跨團隊照護模式
Intensified AKD Care to Reduce CKD: multidisciplinary team care (MDT) model

陳佑瑋 [1,2,3], 吳美儀 [1,2], 廖家德 [1,2], 許永和 [1,2,3], 鄭彩梅 [1,2], 邱怡仁 [1,2], 洪麗玉 [1,2], 吳麥斯 [1,2,3]
[1] 臺北醫學大學雙和醫院 內科部 腎臟內科 [2] 臺北醫學大學 醫學院 醫學系腎臟內科學科
[3] 臺北醫學大學 醫學院 臨床醫學研究所

Introduction

AKI is estimated to result into a worldwide death toll more than 2 million people annually. Besides being fatal, acute kidney injury also increases the risks of chronic kidney disease (CKD) and end stage renal disease (ESRD) among survivors and thus casts large disease burden on health care system. Acute kidney disease (AKD), has been proposed as a window of intervention to prevent occurrence of CKD. However, the effective therapeutic strategies of AKD care remain to be developed.

Methods

We intended to conduct a prospective, randomized, open-label, behavioral interventional trial to validate the efficacy of in-hospital multidiscipline team (MDT) care model and special outpatient AKD clinic which aim to improve AKD care and to reduce de novo CKD incidence. The trial will enroll all adult patients with AKD. The intervention group will receive multidisciplinary team (MDT) care and will be followed at acute kidney disease (AKD) clinic. The control group will receive standard care. Primary outcome is the proportion of composite endpoint of major adverse kidney events on 90th day after AKI (MAKE: renal progression to CKD, chronic dialysis and death).

Results

First, we have formed our consensus with dietitians and developed web-based nutritional assessment tool. Second, under the support of the pharmacy department, we have formed our consensus on the recommendations of drug use for the severe AKD patients. Third, we have commenced the pilot trial to test the feasibility and efficacy of MDT care. We have conducted a temporary analysis to examine the efficacy of the intervention. Among all the 128 participants till 4th of November, 2021, mean age of the trial participants was 68-year-old and 75(59.5%) of them were male. Survival analysis has revealed a non-significant survival difference between intervention group and standard care group regarding composite endpoint of mortality and/or dialysis. However, the trend of survival curve favored the intervention group (MDT care and AKD clinic)

Conclusion

Development of effective interventions at AKD stage is a critical step to reduce the up-rising CKD incidence.

Key words: acute kidney injury; AKI; acute kidney disease; AKD; multidiscipline team; MDT; care model

台灣腎臟醫學會 111 年度春季學術演講會

專題演講 5

Epidemiological perspective of first dialysis
首次透析的流行病學觀點

廖家德
臺北醫學大學 衛福部雙和醫院腎臟科

Kidney replacement therapy, including dialysis and kidney transplantation, is an essential life-maintaining strategy to tackle the functionally failed kidneys. In the past, the outcome studies of the dialysis population have been focused on chronic kidney disease (CKD) stage 5D/end-stage kidney disease (ESKD) patients. Recently, more studies addressed the outcomes of patients with severe acute kidney injury (AKI) requiring dialysis. Here, we aim to investigate the transitional outcomes of patients receiving first dialysis treatment from 2001 to 2017 in Taiwan, utilizing National Health Research Database.

Our data has illustrated the increasing trend of first dialysis incidence during the period of 2001 to 2017 in Taiwan, with men having more risk of dialysis occurrence than women, especially men aged 35–55 years. Patients aged more than 65 years (men or women) experienced the highest incidence rate of receiving first dialysis treatment. Further comparisons between patients with the 'planned dialysis' (those who had been enrolled in the pre-ESRD program and received arteriovenous shunt creation before their first dialysis) and the 'unplanned dialysis' group have revealed that the latter has a higher death risk and mortality rate. Notably, the death risk and mortality rate are higher in the 'unplanned dialysis' group without previous kidney disease (*de novo* AKI) than those with previous kidney disease (AKI on CKD). Additionally, we have characterized the transition pathways of patients starting their first dialysis as the 'unplanned dialysis', which has disclosed that up to nearly 95% of all patients who survived after first dialysis eventually developed to continued dialysis and deaths by the end of the 5-year follow-up period. Finally, we have identified common risk factors for developing maintenance dialysis and deaths, including male gender, older age, diabetics, coronary heart disease, stroke, heart failure, and sepsis.

This is the first time that a study has comprehensively addressed the incidence and transitional outcomes of all patients initiating the first dialysis at a nationwide scale in Taiwan. The results indicate that more efforts on post-AKI care is warranted to improve the renal and overall survivals.

專題演講 5

Role of genetics and immunity in AKI-AKD-CKD continuum

吳美儀
衛生福利部雙和醫院 腎臟科

Kidney disease is an important non-communicable disease issue in global public health and has caused a heavy economic burden on the world. The literature points out that about 5-10 million people die of kidney disease worldwide every year. Among them, acute kidney injury (AKI) is an important risk factor leading to acute kidney disease (AKD), chronic kidney disease (CKD), another organ failure, and death. A multi-country, multi-center prospective observational study showed that about 47.5% of critically ill patients suffered AKID due to sepsis. Previous investigation has indicated that both genetic variation and immune status are considered as important factors for the risk of sepsis-associated AKI (SA-AKI). The genome-wide association study (GWAS) and the high-throughput sequencing (HTS)-based immune profiling analysis as powerful tools are thus capable of providing in-depth information for interrogating the connection of such disease with genetics and immunity, respectively. Accordingly, researchers could have better understanding of how genetic and immune factors are associated with SA-AKI in order to develop novel biomarkers and therapeutic strategies for diagnosis and treatment of AKI. Further application of such study approaches and findings would contribute to the prevention of AKI, AKD and CKD for healthy individuals and kidney disease patients.

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專題演講 6

腎臟穿刺

李明峰　陳信佑
高雄榮民總醫院 放射線部
高雄榮民總醫院 腎臟科

Renal parenchymal core biopsy can be done either with coaxial or noncoaxial technique. In coaxial technique, the introducing needle is placed in the target organ; then, multiple tissue samples can be performed through the same tract. After biopsy, embolizer such as blood clots, absorbable gelatin sponge or coil could be introduced via the coaxial introducer to plug the needle tract, preventing bleeding. Coaxial technique is widely used in solid organ biopsy performed by radiologists. Alternatively, in noncoaxial technique, biopsy needle is inserted repeatedly for each tissue sampling. Non-coaxial technique is usually performed by nephrologists. Through the literature review, coaxial technique has been shown to have shorter procedure time and less complication rates related to post-biopsy bleeding, especially preferable to patients with impaired coagulation tests or pathologic renal parenchyma. In this lecture, we would like to give a short review and demonstrate how to perform a quick and safe renal parenchymal biopsy through coaxial technique. Nephrologists could learn this technique and serve it as a standard of care.

專題演講 6

腹膜透析經皮植管技術執行與經驗分享

鄭本忠
高雄長庚醫院 腎臟科

Peritoneal dialysis (PD) is one of the choices of renal replacement therapy in patients with end stage renal disease (ESRD). PD is known as beneficial in suitable patients as it facilitates home therapy, encourages patient independence and the preserved residual renal function to contributes to better quality of life.

The growing number of PD patients with overall increase in comorbidities requires the development of rapid and safe PD catheterization techniques that avoid the use of general anesthesia.

Nephrologist initiated Peritoneal Dialysis Catheter Insertion (NIPD) is now as an essential/elected procedure in 15 Hospitals (11% of PD centers) in Taiwan.
However, there are many challenges ahead during the performance, including laws and regulations, qualification, privilege, facilities management, standardization, quality assurance and clinical pathway. Forty-five patients with NIPD have been successfully performed in Kaohsiung Chang Gung Memorial Hospital since Jan 2021.

Here, we would like to share the development process and experience, as well as the difficulties and solutions encountered.

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專題演講 6

上肢周邊血管超音波檢查與應用

吳重寬
新光紀念醫院 透析血管通路管理中心

演講大綱:

- 血管超音波的基本概念
- 血管的走查
- 正常上肢動脈與靜脈血管走勢、分布與特性
- 動靜脈瘻管手術前的上肢動脈與靜脈血管圖譜與評估
- 透析病人動靜脈瘻管的周邊血管超音波評估
- 評估動靜脈瘻管成熟與否與未成熟動靜脈瘻管的原因
- 無症狀病人的動靜脈瘻管的定期追蹤
- 動靜脈瘻管的併發症
- 血管超音波的臨床技術運用

專題演講 7

急性腎損傷生物標記綜論
Overview Biomarkers for AKI

黃俊德
臺中榮民總醫院 重症醫學部

Acute kidney injury (AKI) is associated with adverse cardiovascular outcome and mortality. Current international consensus adopts serum creatinine and urine output, which are functional markers of kidney, as the gold standard of AKI diagnosis. Serum creatinine and urine output are easy to use and correlates well with long term outcome. However, creatinine is confounded by fluid overload and muscle mass and delayed to rise after kidney injury. Therefore, discovery of novel stress and injury biomarkers for kidney injury become a highly expected solution for early prediction and diagnosis of AKI. In this lecture, a review of current biomarkers will be introduced in a comprehensive way to give the audience a general picture of AKI biomarkers.

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專題演講 7

肝型脂肪酸結合蛋白在急性腎衰竭的臨床應用
Clinical use of urinary liver-type fatty acid binding protein in acute kidney injury

洪啟智
高雄醫學大學附設醫院 腎臟科

Fatty acid is a major energy source of renal tubule epithelial cells. Fatty acid binding proteins (FABPs) are known as intracellular fatty acid chaperones that transport lipids to a specific component in the cell. Among them, L-FABP (or FABP1) is expressed in liver and kidney (proximal tubules) and H-FABP (or FABP3) is expressed in heart and kidney (distal tubules). However, mouse and rat do not express L-FABP. Human FABP1 transgenic mice are used to evaluate the responses of FABP1 to several AKI models.

L-FABP is effective for earlier diagnosis of acute kidney injury (AKI) in animal models and human diseases of hypoxia or proximal tubule injury. L-FABP could be upregulated by hypoxia inducible factor-1 and peroxisome proliferator- activated receptors pathways. L-FABP is then shed into urine in response to hypoxia caused by decreased peritubular capillary blood flow. In animal models of renal ischemia-reperfusion AKI or cisplatin-induced AKI, urinary L-FABP increases early before the increase of urinary N-acetyl-D-glucosaminidase and blood urea nitrogen. In patients after radiocontrast medium exposure or cardiac surgery, L-FABP is one of the best predictor that could detect AKI earlier than serum creatinine. Even in animals or patients with prerenal AKI, L-FABP is significantly elevated compared with other biomarkers.

Novel biomarkers of AKI are reflective of the molecular and cellular events that occur throughout the clinical phases of AKI. L-FABP elevation during the initial phase of AKI indicates a hypoxic insult and L-FABP elevation during the extension phase of AKI likely represents a response to a kidney insult, because of its antioxidant mechanisms.

In clinical practice, L-FABP measurement could be used to early identify high-risk patients. And strategies that increase its expression could benefit patients who undergo a renal insult.

專題演講 7

嗜中性白血球明膠酶相關運載蛋白
Neutrophil Gelatinase-Associated Lipocalin (NGAL)

張智翔
林口長庚醫院 腎臟科

Acute kidney injury (AKI) is common in hospitalized patients and associated with serious complications and high medical costs. The diagnosis was relied on serum creatinine (sCr) and urine output, to define AKI. But these markers are delayed changes after AKI and showed low sensitivity or specificity. However, AKI biomarkers were discussed for over 20 years, there is no clear rule or pathway to use them. In this session, we would like to discuss several novel biomarkers that have been shown to detect AKI earlier and are more sensitive than sCr. And discuss the way and probable prevention strategies to improve the outcome in patients.

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專題演講 7

胱蛋白 C
Cystatin C

黃道民
台大醫院 腎臟科

Acute kidney injury (AKI) and the subsequent acute kidney disease (AKD) are emerging hot topics in both fields in critical care medicine and nephrology. The health and economic burdens associated with AKD have caused a substantial loss of human lives and medical expenditure. One of the most important barriers to improve outcome is the late and inaccurate diagnosis of AKI and AKD. Traditionally, serum creatinine serves as a “static” marker for glomerular filtration rates (GFR), and one of the most easy assessment tools for renal function; however, the inaccurate estimates for renal function in acute setting make creatinine a flawed tools in AKD. Cystatin C has a low molecular weight (13.3 KD), and it is removed from the bloodstream by glomerular filtration in the kidneys. If kidney function and glomerular filtration rate decline, the blood levels of cystatin C rise. Normally, cystatin C will not be detected in urine, for renal tubules absorb almost all of the cystatin C filtered by glomerulus. Detection of cystatin C in urine may serve as an AKI marker in some circumstances. As a filtration marker, cystatin C has been validated for AKD diagnosis and as a guide for intervention. This talk will focus on the role of cystatin C on AKI prediction, outcome prediction and its possible roles as the time for intervention.

專題演講 8

Overview of therapeutic monoclonal antibodies

黃道民
台大醫院 腎臟科

Therapeutic monoclonal antibodies target pathognomic molecules, in order to abolish disease processes and achieve remissions of diseases. In kidney diseases, disease processes involving humoral immunity or cellular immunity may contribute to kidney damage. Minimal change disease (MCD) and focal segmental glomerulosclerosis (FSGS) are classified with disease processes involving cellular immunity, while IgA nephropathy and anti-GBM disease are mostly associated with humoral immunity disorder. Patients post renal transplantation, despite anti-rejection medications, also have an increased risk for de novo glomerular diseases. The roles of monoclonal antibodies to help manage these disorders are emerging and potentially change the therapeutic paradigms in the near future. This talk will give an overall view of therapeutic monoclonal antibodies in kidney diseases.

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專題演講 8

Use of monoclonal antibodies in primary glomerulonephritis

塗昆樺
林口長庚紀念醫院腎臟科系

Primary glomerular disease consists of lots of disorders with different clinical presentations, including minimal change disease, focal segmental glomerulosclerosis, membranous nephropathy, immunoglobulin A nephropathy, membranoproliferative glomerulonephritis and so on. Although the mechanisms of these disorders differ, management is similar including glucocorticoid and immunosuppressants. Pathogenic mechanisms of these disorders are not well known yet. Recently, novel immune therapy including monoclonal antibodies develop rapidly. Some of these immune therapies are applied to patients with primary glomerular disease. The successfulness of these novel treatments provides not only therapeutic benefits but also re-evaluation of diseases mechanism. This talk will introduce current use of monoclonal antibodies in primary glomerular disease.

Keyword: primary glomerular disease, monoclonal antibodies, immune therapy

專題演講 8

單株抗體於腎臟領域之應用
Use of monoclonal antibodies in immune related kidney diseases

楊皇煜
林口長庚醫院腎臟科

In 2013, Science magazine choose cancer immunotherapy as breakthrough of the year. Human monoclonal antibodies (mAbs) not only changed the paradigm of tumor therapy but also became a therapeutic tool for many other diseases. Most of the application of mAbs revealed encouraging findings to treat patients with immune-mediated renal disease such as glomerular diseases and kidney transplantation rejection, for whom the standard protocols based on corticosteroids and non-specific immunosuppressants with heavy side effects have for decades been the only therapies. We will introduce human monoclonal antibodies related therapy for immune related renal disease and also for COVID-19.

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專題演講 8

Use of monoclonal antibodies in renal transplantation

游棟閔
臺中榮民總醫院 腎臟科

腎臟衰竭是國人極普遍的健康問題，對國家醫療政策造成龐大的負擔，歐、美、日等先進國家對於日益龐大的尿毒症患者，政府及醫療單位都非常鼓勵進行腎臟移植來解決這個沉重的醫療負擔。而器官移植除了對病人帶來好處外，更是評估一個國家尖端的醫療水準重要指標。

腎臟移植目前所面臨的挑戰包括: 急性抗體性排斥的處理、血型不相容(ABO incompatible)或抗人類白血球基因抗體活體腎臟移植(anti-HLA donor specific Ab)的抗體減敏化治療、非典型血溶性(atypical hemolytica uremia syndrome)活體腎臟移植的處理。

單株抗體包括 anti-CD20、anti-C5a 、anti-interleukin-6 monoclonal antibody 等等 在腎臟移植的術後處理，日益扮演重要的角色。本次演講內容將分別闡述其臨床運用與療效。

Lunch Symposium 1

Burden and challenge for heart failure patients with kidney dysfunction Nephrologists point of view for Heart Failure Treatment

姜至剛
臺大醫院 腎臟科

心臟衰竭一直以來都是心臟相關疾病中死亡率以及住院率高的疾病之一[1-4]，其中五年死亡率甚至可以高達 50%[2]，因此該如何幫助心臟衰竭病患有效並且積極的爭取更多的治療黃金期，一直是臨床治療中努力的方向。

在 CKD 進入到 ESRD 的過程中，心臟衰竭的病生理學是非常複雜的，但簡化的說當 CKD 病患因為體液過度滯留、長期壓力(血壓)過大等原因，就可能會形成心臟衰竭，進而產生一系列的惡性循環，包含 CKD 持續惡化、增加心衰竭再住院、突發心律失常死亡與 pump failure 死亡等。

傳統在這類型的心衰竭病患治療，常常使用 RASSi 作為主要治療機轉藥物之一，不過近年來在心臟衰竭有一新機轉藥物 ARNI，除了調控 RASS system 外，還可以調控 NP 系統除了幫助調控 RAAS 系統與交感神經系統以及我們熟悉幫助減少心臟重塑外，也可以幫助腎臟利尿等功能。

從 2014 年 PARADIGM-HF[5] 針對心臟衰竭 (HFrEF)病患的大規模臨床試驗結果發表以來，針對 HFrEF 的治療就有了嶄新的一頁，然而隨著 PARADIGM-HF 研究發表，後續也有許多研究證明 ARNI 對於 HFrEF 心臟治療的效果，從根本的反轉心臟功能、NT-proBNP、KCCQ 到死亡住院率的改善，都經過不同實驗的設計證實 ARNI 對於 HFrEF 病患的效果。而這樣的大型研究也促使著世界各國針對心臟衰竭治療的指引有了不同的建議，進而提升對於心衰治療的討論，而這樣的進展對於臨床醫師而言，該如何幫助病患更有效的掌握黃金治療契機，並且提供更積極的治療計畫，也是持續堅持的方向。因此，本次演講主題會跟大家分享：如何即早優化心衰竭治療，讓臨床研究的治療效果也能在實際病患治療中看到。

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台灣腎臟醫學會 111 年度春季學術演講會

Lunch Symposium 2

Revolutionize CKD Therapy - Delay Dialysis and Reduce Mortality

蔡宜純
高雄醫學大學附設醫院 腎臟科

台灣最新健保支出報告顯示2019年十大花費疾病，首位為慢性腎臟病，支出了533億點，透析人數也與日俱增，從9萬人增加到了9.2萬人，台灣透析的盛行率為世界之冠，因而被汙名為洗腎王國，是台灣政府及醫療體系急需解決的一大問題。ESRD的病因有42%為糖尿病造成，高血壓及腎絲球腎炎各佔18%，因此想挽救台灣腎臟病，糖尿病的治療及及早的腎臟保護介入非常重要。近期終於出現了一系列能夠降低腎臟硬終點(eGFR decline, ESRD, renal death)的藥物臨床試驗發表，包括已上市的SGLT-2i(Credence、DAPA-CKD試驗)和還未在台灣上市的Finerenone(FIDELIO-DKD試驗)、Atrasentan(SONAR試驗)。其中，DAPA-CKD試驗在2020年9月發表，不但發現Dapagliflozin在糖尿病合併CKD病患能降低36%腎臟硬終點，更發現在沒有糖尿病的CKD病患也能降低50%腎臟硬終點，ESRD風險顯著降低36%，全死因死亡風險更是降低31%，為人類醫療史上CKD治療的重大突破，今年8月26日Dapagliflozin已在台灣取得治療CKD及預防糖尿病腎臟病的兩個新適應症，並能持續使用到透析前。隨著這些藥物在適應症上的取得、健保的給付及臨床上使用比例的增加，相信對台灣CKD及ESRD的問題能有所幫助。

Lunch Symposium 3

如何治療及照護腎臟病 C 肝病患?

戴嘉言
高雄醫學大學附設醫院 肝膽胰內科

C 型肝炎除了造成肝硬化、肝癌以外，也會有肝外共病的產生，像是糖尿病及慢性腎臟病。腎臟病病患 C 肝的盛行率是一般人的 2 倍，治癒 C 肝後能幫助延緩腎功能的惡化且降低末期腎衰竭的發生率。C 肝是個沉默的疾病，許多 HCV 感染患者並無明顯的症狀，許多病患需要篩檢才能被發現。政府為推動 2025 年滅除 C 肝，由國健署在成人健檢中提供免費 BC 肝的篩檢，也已在 2021 年 10 月開放所有科別都可治療 C 肝。目前的全口服直接作用抗病毒藥物(DAA)已有很多有效性、安全性、方便性的實證資料，在台灣本土治療 C 型肝炎的治癒率更可達 99%以上。此次演講將會說明如何篩檢治療及後續的追蹤照護，期待腎臟科的醫師們也能一起加入滅除 C 肝的行列!

台灣腎臟醫學會 111 年度春季學術演講會

Industry Lecture

The Role of Carbamylation in Patients with Kidney Disease

林祐賢
高雄市立大同醫院 (委託高醫經營) 腎臟科

Carbamylation is a non-enzyme-reacted protein post-translational modification. When the nitrogen-containing waste in the body rises, urea dissociates into cyanate, which then reacts with proteins. This is called protein carbamylation. In basic research, carbamylated proteins have been observed altered their structure and function, contributing to adverse molecular and cellular responses. Higher concentrations of carbamylated derivatives have also been observed in pathological studies, especially in chronic kidney disease (CKD). In recent years, many studies have shown that serum carbamylated derivative concentrations are positively correlated with renal failure and cardiovascular disease, furthermore, it can be used as a predictor of mortality in patients with end stage renal disease (ESRD). When its concentration accumulates, it may promote long-term complications, including cardiovascular abnormalities, inflammatory responses, and dysregulation of immune responses.

Carbamylation affects a wide range of proteins, including albumin, lipoprotein, and may contribute to renal fibrosis, erythropoiesis-stimulating agents resistance, atherosclerosis, and vascular calcification. The degrees of carbamylated albumin are closely related to clinical outcomes in patients with kidney diseases. The intervention with dialysis can reduce the concentration of carbamylated proteins. And amino acid supplementation may be used as a scavenger to reduce carbamylation of proteins.

In conclusion, the harm of protein carbamylation to patients deserves attention, and how to measure and slow down the generation of carbamylated proteins will be an important issue in the patients with kidney diseases.

病例報告 1

1-1.

以惡性高血壓表現的 41 歲女性，最終診斷為腎素分泌瘤

A 41-year-old female patient, with initial presentation of malignant hypertension, diagnosed to be renin secreting tumor : A case report

周子巽 顏銘佐
Andrew Chou, MING-TSO YAN
國泰綜合醫院腎臟內科
Division of Nephrology, Department of Internal Medicine, Cathay General Hospital

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This 41year old female came to our emergency department for dyspnea on exertion. She has underlying uncontrolled hypertension for at least 2 years, with systolic pressure over 200mmHg. Her vital signs at triage was Body temperature = 36.6℃, Pulse rate = 122 per minute, Respiratory rate = 26 per minute, Systolic/Diastolic blood pressure = 209/159 mmHg, O2 saturation = 74%, consciousness clear. Physical examination revealed jugular vein engorgement, bilateral lung crackles, and bilateral legs pitting edema. The remainder of physical examination was unremarkable. Hemogram study showed respiratory and metabolic alkalosis (pH=7.5, pO2= 56.4 mmHg, pCO2=32.1 mmHg, HCO3=24.6 mmol/L), severe hyponatremia (Na=116 mmol/L), hypokalemia (K=2.9 mmol/L), hypoalbuminemia (2.6g/dL), A/G reverse (Serum protein = 5.4 g/dL) and impaired renal function (BUN=70 mg/dL, Creatinine =4.32 mg/dL). Urine analysis showed microscopic hematuria (RBC=30~49/HPF), and Urine Protein/Creatinine Ratio (UPCR) =3.36 g/g. Chest plain film reported bilateral pulmonary edema. Bedside renal echo showed no hydronephrosis.

She was admitted to Intensive Care Unit for pulmonary edema and respiratory failure, treated by Furosemide but was ineffective. She then received emergent hemodialysis for 2 days. After ultrafiltration, her hyponatremia and respiratory failure got much improved.

To survey her acute kidney injury, we checked auto-immune serology and relative viral infection, but were all negative. We then survey her malignant hypertension, which maybe the cause of her impaired renal function and possible glomerulonephropathy. We found high plasma aldosterone concentration (319.1 ng/dl), high plasma renin activity (>33 ng/ml/hr), and high plasma renin concentration (618.1 pg/ml). Besides, other endocrine serology data were unremarkable.

Abdominal MRI reporting an 1.7cm T1 isointense, T2 hypointense tumor over the upper pole of right kidney. Following formal renal echo showed an 1.7 cm isoechoic tumor in the cortex of the upper pole of right kidney. Thus, echo guided renal tumor biopsy was done, reporting juxtaglomerular cell tumor, with rhomboid crystals in the tumor by electron microscopy, compatible with reninoma.

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Key word: Juxtaglomerular cell tumor; Reninoma; Renin secreting tumor

台灣腎臟醫學會 111 年度春季學術演講會

病例報告 1

1-2.

引起陣發型低血鈉及低血鉀的罕見原因

A rare cause of episodic hyponatremia and hypokalemia

鄭子明 [1] 許智揚 [1]
Tzu-Ming Cheng[1], Chih-Yang Hsu[1]
[1] 高雄榮民總醫院內科部腎臟科
[1] Division of Nephrology, Department of Medicine, Kaohsiung Veterans General Hospital

Abstracts

A 21-year-old woman who was healthy before presented to the emergency department with intermittent lower abdominal pain, recurrent vomiting, and poor appetite for about six weeks. Muscular weakness was reported as well. During this period, she was ever admitted to local clinic due to two episodes of general convulsion. Laboratory analysis revealed a striking hyponatremia (119 mmol/L). Brain computed tomography, brain magnetic resonance imaging and electroencephalogram did not reveal pathologic features. She was discharged after correction of hyponatremia (129 mmol/L). However, recurrent abdominal pain was found, and she visited our hospital for evaluation.

On examination, vital signs showed blood pressure 153/112 mmHg, and heart beats 117/min. She had soft abdomen with no localized guarding and no tenderness, and no pitting edema over trunk and limbs. Abdominal plain film showed colonic stool retention. Laboratory analyses still revealed hyponatremia (122 mmol/L), hypokalemia (2.8 mmol/L), elevation of liver-enzyme (GOT/GPT 65/77 U/L) and a urine sodium level of 61 mmol/L. Under the diagnosis of SIADH, water restriction and salt supplement was administered, with recovery of sodium level. However, weakness, numbness was reported despite nerve conduction studies unremarkable. According to her clinical manifestations (recurrent abdominal pain, seizures, hyponatremia and young female), porphyria was highly suspected. A more than 10-fold increase in concentration of urine porphobinogen (PBG) and elevated urine aminolevulinic acid (ALA) lead to the diagnosis of acute intermittent porphyria.

She was then treated with intravenous hemin 3mg/kg body weight/day for 4 days, with intravenous carbohydrate. Symptoms got improved and she was then discharged with clinic follow-up.

Key words：
Hyponatremia, SIADH, Acute intermittent porphyria

病例報告 1

1-3.

腎臟移植術後一週內併發急性排斥反應

An allograft kidney recipient complicated with active antibody mediated rejection after transplantation in one week

陳怡雯[1] 周康茹[1]
I-wen Chen, Kang-Ju Chou
高雄榮民總醫院腎臟科[1]
Department of Nephrology, Kaohsiung Veterans General Hospital

Abstracts

Antibody-mediated rejection has been recognized as an important cause of graft loss after kidney transplant, and there are no standard treatments currently. We use HLA typing and crossmatching before transplantation in our hospital to choose the recipient of deceased donor kidney. Here, we report a 50-year-old woman, with history of chronic interstitial nephritis (CIN) related end stage renal disease (ESRD), who received kidney transplantation and complicated with active antibody mediated rejection soon after surgery.

In this patient, pre-operative pre-formed antibody matching test showed negative result. Mismatched HLA included HLA: 1A(3), 1B(35), 1 DR(12). Panel reactive antibodies showed class I: 95.3%, class II: 75.6%. Basiliximab, methylprednisolone, tacrolimus were given as pre-medications. After transplantation, there was no urine despite of hydration, diuretic and inotrope given to support adequate kidney perfusion. Sonography didn't detect urinoma over lower abdomen. The renal arterial resistive index (RI) was measured as 0.96~0.98 (normal range =0.5~0.7), and reversed diastolic flow in the renal artery was found, suspect acute rejection. Methylprednisolone 1 g per day was given for 3 days and renal biopsy was performed on the 7th day after transplantation, which presented acute antibody-mediated rejection, C4d(+), C4d3: >50% PTC(+), acute Banff scores grading: i3 t0 v2 g3 ptc3 C4d3. Plasmapheresis 40U/day for 10 courses, intravenous immunoglobulin 10 times, and rituximab 500mg were given. Thymoglobulin was also administrated due to highly suspected T cell rejection from v2. Donor specific antibody (DSA) identification later reported A3, B35, Cw4, DR12, DQ7 in the patient. Her urine output and renal function gradually improved with serum creatinine down to 1.55mg/dl after treatment. Although our patient had high PRA levels and complicated with active antibody mediated rejection, the treatment with plasmapheresis, IVIG, rituximab and ATG seem to be an effective regimen to treat active antibody mediated rejection.

關鍵字: 腎臟移植，抗體媒介排斥反應，群組反應性抗體，捐贈者特異性抗體
Keywords: kidney transplantation, antibody mediated rejection, panel-reactive antibody (PRA), donor specific antibody(DSA)

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病例報告 1

1-4.

產婦急性腎損傷及血小板低下症:非典型溶血性尿毒症

Pregnancy related Atypical Hemolytic Uremic Syndrome Successfully Treated by Eculizumab

羅翊中[1] 陳信佑[1]
Yi-Chung Lo[1], Hsin-Yu Chen[1]
[1] 高雄榮民總醫院內科部腎臟科
[1] Division of Nephrology, Department of Medicine, Kaohsiung Veterans General Hospital

Abstracts

Atypical hemolytic uremic syndrome (aHUS) is a rare and life-threatening disease, induced by complement alternative pathway disorder, and characterized by thrombotic microangiopathy on kidney pathology. Pregnancy associated TMA includes preeclampsia/HELLP syndrome, thrombotic thrombocytopenic purpura, and aHUS. Herein, the recognize of aHUS in pregnancy is an challenge for physician.

We describe a 34-year-old female, presented with postpartum anuria, pulmonary hemorrhage, microangiopathic hemolytic anemia and thrombocytopenia after delivery of her first-born boy. The kidney biopsy pathology demonstrated thrombotic microangiopathy with diffuse cortical necrosis. aHUS was confirmed by normal ADAMTS 13 activity and exclusion of E.coli O157, shigella and Pneumococcus pneumoniae infection. The complement gene variant of coding area of complement factor H gene and CD36 gene were detected. Resolution of hematological abnormalities, pulmonary hemorrhage and increase urine output were observed after receiving Eculizumab. In summary, we present clinical, kidney biopsy, laboratory findings and the treatment and follow-up of aHUS with cortical necrosis.

Awareness of aHUS presentations will result in accurate diagnoses and proper management, and renal cortical necrosis can be partially improved while predisposing factor is removed and treatment is focused on the cause of the aHUS as early as possible.

Key words：
Pregnancy, microangiopathic hemolytic anemia (MAHA), AKI, TMA, aHUS

病例報告 2

2-1.

陣發性夜間血紅素尿症病人：可逆之急性腎臟病

Reversable acute kidney disease in a patient with paroxysmal nocturnal hemoglobinuria

于志業[1] 林維洲[2] 陳永銘[1] 黃道民[1]
Peter Zhi-Ye Yu MD.,[1] Wei-Chou Lin MD.,[2] Yung-Ming Chen, MD.,[1] Thomas Tao-Min Huang MD.[1]
[1] 台灣大學附設醫院內科部腎臟科 [2] 台灣大學附設醫院病理部
[1] Renal Division, Department of Internal Medicine, National Taiwan University Hospital and College of Medicine, Taipei, Taiwan [2] Department of Pathology, National Taiwan University Hospital and College of Medicine, Taipei, Taiwan

Background：

Paroxysmal nocturnal hemoglobinuria (PNH) is a rare, yet life threatening disease involving multiple organs. The renal manifestations are diverse: acute tubule necrosis, acute interstitial nephritis (AIN), Fanconi's syndrome, and pigment nephropathy. Eculizumab may improve outcomes. Kidney pathology provides valuable information to guide our intervention.

Methods：

An 85-year-old man with known PNH with advanced chronic kidney disease (CKD) stage 5 (serum creatinine = 4 mg/dL) was ever on eculizumab one year ago but was judged ineffective. Uremia with extreme azotemia (urea nitrogen, UN = 204.9 mg/dL, serum creatinine 12.4 mg/dL) was found, with evidence of hemolysis (lactate dehydrogenase, LDH = 353 U/L), thrombosis (D-dimer = 4.26 mg/L), and tubular injury (euglycemic glycosuria, 2+ in urinalysis). We thus performed a kidney biopsy to guide therapy.

Results：

The histopathology reports brown pigments in the edematous tubular epithelial cells and these pigments are positive for the iron stain, which was compatible with hemosiderin deposits. Also, widening change of interstitium with mild lymphocytic infiltration (15~40%) was disclosed. For interstitial nephritis, especially in acute episode, steroid treatment may improve renal outcome. Oral prednisolone 20mg per day (0.34 mg/kg) was prescribed for interstitial nephritis. Urine amount increased and hemodialysis was discontinued on the 56th day after biopsy. The serum creatinine was 6.0 mg/dL on the 25th day of dialysis free.

Conclusions：

Kidney pathology is diverse in PNH patients, and could serve a guide for therapy to improve outcomes

Key words
paroxysmal nocturnal hemoglobinuria, acute kidney disease, eculizumab, kidney biopsy

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病例報告 2

2-2.

Everolimus 所致肺炎在腎移植患者

A Rapidly developed Everolimus-induced Pneumonitis in Renal Transplant Recipient

邵月珠 甄沛勤 陳錫賢
Yueh-Chu Sio, Pei-Chin Chen, Hsi-Hsien Chen
台北醫學大學附設醫院內科部腎臟內科
Division of Nephrology, Department of Internal Medicine, Taipei Medical University Hospital

Inhibitors of mTOR (mammalian target of rapamycin) are currently widely used as an immunosuppressive and anti-malignancy medication. However, they are also known for their adverse effects on pulmonary system that could be life-threatening. Many studies have implicated that everolimus caused pneumonitis frequently, and to our knowledge, would happened as soon as 5 days after drug initiation. Here, we presented a rapidly deteriorating case of drug-induced pneumonitis within 3-4 days upon starting everolimus that spontaneously regressed after drug discontinuation in a renal transplant recipient.

病例報告 2

2-3.

NK/T 細胞淋巴癌罕見的腎臟侵犯

NK/T-cell Lymphoma Invading Kidney Mimicking Adult Onset Still's Disease

江哲甫[1] 巫宏傑[1] 王偉傑[1] 丁瑞聰[1] 陳冬英[2]
Che-Fu Chiang[1], Hung-Chieh Wu[1], Wei-Jie Wang[1], Jui-Tsung Ting[1], Tung-Ying Chen[2]
[1] 衛生福利部桃園醫院內科 腎臟科
[2] 台北馬偕紀念醫院 病理科
[1]Division of Nephrology, Department of Internal Medicine, Taoyuan General Hospital, Ministry of Health and Welfare, Taoyuan, Taiwan
[2]Department of Pathology, Taipei Branch, MacKay Memorial Hospital, Taipei, Taiwan

Lymphoma involvement in kidney is less reported in past literature. We report a case of 53 year-old male patient initially presented with fever, jaundice, favor biliary tract infection complicated with chronic active hepatitis. Gastrointestinal bleeding episode was also noted over admission course. Panendoscopy biopsy of stomach and liver biopsy had exclude IgG4 disease and autoimmune hepatitis, primary biliary cirrhosis. As salmon-pink maculopapular rash was noted and further laboratory data revealed elevation in serum ferritin, tentative diagnosis of adult onset Still's disease was made. Empiric antibiotics was given first and steroid was prescribed afterward. He was then discharged to outpatient department for following up and titration of immunosuppressants. However, on second visit of our emergent department, the patient presented with persistent fever and proteinuria. Thus renal biopsy was performed. The pathology revealed NK/T-cell lymphoma involvement in kidney. Further bone marrow biopsy also confirmed the diagnosis. To our knowledge, there has been cases of NK/T-cell lymphoma involvement in upper airway, Waldeyer's ring, gastrointestinal tract, skin, testis, lung, eye, or soft tissue, but kidney involvement was seldom reported. NK/T-cell lymphoma was divided to nasal type and extra-nasal type. Extra-nasal type NK/T-cell lymphoma has slightly different immunophenotype distribution from nasal type, and generally presented with clinically more B symptoms or more advanced stage, as correlated with our patient. The treatment of late stage extra-nasal NK/T-cell lymphoma consists of asparaginase-based combination chemotherapy. Immunotherapy such as Pembrolizumab or Nivolumab is also indicated in relapse/refractory disease. Otherwise clinical trial or best supportive care are options for such patients as prognosis is poor in relapse/refractory disease. We hope to raise consideration of malignancy in presentation of proteinuria through this case registration.

Key words: NK/T-cell lymphoma, Extra-nasal type, Proteinuria, Kidney biopsy, Unknown fever, Adult onset Still's disease

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病例報告 2

2-4.

腎臟移植受贈者之移植耐受不良症候群及切除

Graft intolerance syndrome with graft nephrectomy in a kidney transplant recipient

劉家佑、徐愷翔
Chia-Yu Liu, Kai-Hsiang Hsu
亞東紀念醫院腎臟內科
Division of Nephrology, Department of Internal Medicine, Far Eastern Memorial Hospital

We present a 67-year-old male with underlying disease of type 2 diabetes mellitus, hypertension, and end stage kidney disease, status post hemodialysis since April 2015. He received deceased donor kidney transplant in December 2015. He had regular follow-up at out-patient department and received tacrolimus, prednisolone and mycophenolate mofetil as immunosuppressants with stationary graft function. His blood creatinine level was steady around 1.5 mg/dL. However, blood creatinine elevated gradually to 2.0 mg/dL in February 2020. Graft renal biopsy revealed diabetic glomerulosclerosis with moderate tubular atrophy and interstitial fibrosis. In December 2020, acute kidney injury with blood creatinine level up to 5.62 mg/dL was noted. Graft renal biopsy was arranged again which revealed diabetic glomerulosclerosis with crescent formation, superimposed on marked arteriolosclerosis. Malignant nephrosclerosis was favored and his creatinine level improved to 3.47 mg/dL after optimal supportive care. In April 2021, he was admitted due to creatinine 10.33 mg/dL with fluid overload, uremia and oliguria, and hemodialysis was initiated. Fever with right lower abdominal pain, gross hematuria, and graft hydronephrosis were also found. Tentative impression of graft acute pyelonephritis was suspected but the symptoms persisted despite graft percutaneous nephrostomy drainage and antibiotics treatment course. Furthermore, persisted elevated CRP level 10.8 mg/dL and resistant anemia 6-7 g/dL under high dose erythropoietin-stimulating agent were noted. Graft intolerance syndrome was impressed and graft nephrectomy was arranged. His general condition much improved after graft nephrectomy.

Based on literature review, benefit and risk should be weighed before graft nephrectomy and graft intolerance syndrome is the main indication for graft nephrectomy after late kidney graft failure. In summary, we present a case of kidney transplant recipient with graft nephrectomy due to graft intolerance syndrome.

關鍵字:移植腎切除、移植耐受不良症候群、腎臟移植
Key word: Graft nephrectomy, graft intolerance syndrome, renal transplant

病例報告 2

2-5.

血液透析治療丙戊酸中毒

Hemodialysis treatment for valproic acid poisoning

許郡珈、楊如燁、徐世平、彭渝森
Chun-Chia Hsu, Ju-Yeh Yang, Shih-Ping Hsu, Yu-Sen Peng
亞東紀念醫院腎臟內科
Division of Nephrology, Department of Internal Medicine, Far Eastern Memorial Hospital

This 42-year-old woman with a past medical history of bipolar disorder and presented to the emergency department with consciousness change. She had history of suicide attempt several times. Empty prescription bottles of lorazepam, estazolam, quetiapine and valproic acid were brought by the emergency medical service. At the triage, her vital signs on presentation included a blood pressure of 94/53 mmHg, heart rate of 89 beats per minutes, respiratory rate of 18 cycles per minutes, body temperature of 35.7 Celsius degree and oxygen saturation of 100% on room air. Her consciousness was comatose, E1V1M1. The non-contrast computed tomography of brain reported no obvious abnormal lesion. Her initial laboratory workup revealed Na 137 mmol/L, K 4.5 mmol/L, Ca 7.3 mg/dL, Glucose 125 mg/dL, BUN 13 mg/dL, Creatinine 0.48 mg/dL, Ethyl alcohol 151 mg/dL, a valproic acid level of 524.2 ug/mL, ammonia level of 196 ug/dL. The liver function tests were within normal range. The initial arterial blood gas analysis pH 7.439, PCO_2 29.9 mmHg, HCO_3^- 19.8 mmol/L. The urine toxicology screen was positive for benzodiazepine. The electrocardiography was normal sinus rhythm with QTc of 547 milliseconds. After received Flumazenil, her consciousness became E2V2M4. Intubation was done because of poor consciousness and airway protection. The nephrologist was consulted for hemodialysis. She received the hemodialysis approximately 10 hours after ingestion. The hemodialysis was initiated with a high-flux-dialyzer [Fresenius FX 100 Dialyzer], dialysate flow 500 milliliter per minute, blood flow 300 milliliter per minute and continued for 4 hours. 2 hours after the dialysis, the valproic acid level was 170.6 ug/mL. The next day, her consciousness was E2VeM4. The valproic acid level was 204.8 ug/mL. She received the second time hemodialysis. The formula was the same as before. The follow-up valproic acid level was 93.7 ug/mL. Her consciousness became E4VeM6 after receiving the second time hemodialysis. The patient was extubated. She remained stable and was transferred for psychiatric hospitalization.

Based on literature review, plasma protein binding of valproate is nonlinear and concentration dependent. In high valproic acid level, the hemodialysis could be an alternative treatment.

關鍵字：丙戊酸，血液透析
Keyword: Valproic acid, hemodialysis

台灣腎臟醫學會 111 年度春季學術演講會

病例報告 2

2-6.

於高端疫苗接種後之 A 型免疫球蛋白腎病變：個案報告及文獻回顧

De novo IgA nephropathy following Medigen vaccination: A case report and literature review

彭梓晏[1] 董奎廷[1] 徐世平[1] 彭渝森[1]
Tzu Yen Peng[1], Kuei Ting Tung[1], Shih Ping Hsu[1], Yu Sen Peng[1]
亞東紀念醫院腎臟內科
Division of nephrology, department of internal medicine, Far Eastern memorial hospital

Following the coronavirus disease 2019 (COVID-19) pandemic, there has be a global COVID-19 vaccine effort. Increasingly, there are reports of de novo and reactivation of glomerulonephritis soon after vaccination, with a predominance of Immunoglobulin A(IgA) nephropathy and minimal change disease.

We present a 35-year-old Taiwanese woman who developed painless gross hematuria 5 weeks after the second dose of Medigen COVID-19 vaccination. Her urine analysis revealed hematuria as well as proteinuria 2027 mg/g (Spot urine protein creatinine ratio, UPCR). Imaging studies showed no evidence of structural lesions. Her plasma creatinine level was normal(0.65 mg/dL), serologic examination for anti-nuclear antibody (ANA), Hepatitis B/C, Human immunodeficiency virus were negative, and complement levels were within normal limits. Immunoglobulin A levels were elevated (494.8 mg/dL, normal range 66-433). She had no other presentations such as arthralgia, skin rash or anemia.

Kidney biopsy showed mild to moderate increase of mesangial cellularity and matrix, mild tubular atrophy and interstitial fibrosis (30%) with focal mononuclear cell infiltration. Immunofluorescence study showed study shows granular deposition of IgG(-), IgA(++), IgM(-), C3(++), and C1q(-) in the mesangial areas. IgA nephropathy was confirmed. Irbesartan without steroid was used as intial therapy after discussing with the patient. Her daily proteinuria-to-creatinine ratio decreased from 2027 mg/g to 1257 mg/g after being treated for 3 months.

In summary, we report a case of de novo IgA nephropathy following Medigen vaccine injection, adding to the abundant literature of COVID-19 vaccine associated glomerulonephritis.

關鍵字: A 型免疫球蛋白腎病變，高端，蛋白尿
Key words: Immunoglobulin A nephropathy, Medigen, proteinuria, de novo, glomerulonephritis

病例報告 2

2-7.

腎病症候群合併雙側腎靜脈栓塞之案例報告

Nephrotic Syndrome Complicated with bilateral renal vein thrombosis: A case report

周奕伶，彭聖曾
YI-LING CHOU, SHENG-JENG PENG
國泰綜合醫院腎臟內科
Division of Nephrology, Department of Internal Medicine, Cathay General Hospital

The 69-year-old female came to our emergency department with chief complaints of general weakness for 3 days. She had hypertension and hyperlipidemia in the past few years with regular medication. Five weeks before this approach, she had been admitted in other hospital due to nephrotic syndrome with legs edema for six months. Renal biopsy revealed membranous nephropathy one month before this admission. She was given steroid pulse therapy followed by prednisolone 60 mg/day.

After admission, physical examination revealed blood pressure 105/86 mm Hg, pulse rate 95 beats/min with a regular rhythm, and temperature 37.3°C. Bilateral massive symmetric legs edema was observed. Blood analysis was remarkable for hemoglobin 10.4 g/dL, white blood cell 12790/μL, platelet count 71000/μL, albumin 1.8 g/dL, creatinine 0.93 mg/dL, and C-reative protein 24.45 mg/dL. Urine analysis revealed 10-19 red blood cells per high power field, 20-29 white blood cells per high power field, and urine protein 1000 mg/dL. Urinary protein and creatinine ratio was 12.1. Blood culture and urine culture yielded group B β-hemolytic streptococcus. Piperacillin/Tazobactam was given. Abdominal and pelvic computed tomography (CT) were performed for infection source survey, revealed bilateral renal vein thrombosis and inferior vena cava thrombosis with massive ascites. D-dimer elevated significantly to 4.725 g/mL, and serum antithrombin III activity decreased to 64.8%. Duplex ultrasound of peripheral vein in lower limbs revealed bilateral deep vein thrombosis. Then she underwent intravenous heparin infusion followed by apixaban. The following abdominal CT before discharge revealed slowly resolution of bilateral renal vein and inferior vena cava thrombi.

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病例報告 2

2-8.

Exon 23 c.3572C>T/c.3590 T>C 與 exon 19 c. 2808G>T 突變導致的補體介導栓塞性微血管病變

Complement-mediated thrombotic microangiopathy caused by combined exon 23 c.3572C>T/c.3590T>C, and exon 19 c. 2808G>T mutation

林煒棣 [1] 簡志強 [1]
Wei-Jian Lim[1], Chih-Chiang Chien [1]
[1] 奇美醫學中心內科部腎臟科
[1] Department of Nephrology, Chi-Mei Medical Center, Tainan, Taiwan

Complement-mediated thrombotic microangiopathy (CM-TMA) is a rare variant of thrombotic microangiopathy (TMA). It is characterized by hemolytic anemia, thrombocytopenia, and acute kidney injury (AKI). The disease arises from genetic and acquired abnormalities that result in uncontrolled alternative-pathway complement activation. Mutations causing CM-TMA can be subdivided into two groups: (1) loss-of-function mutations (affecting factor H, factor H-related proteins, membrane co-factor protein, and factor I) and (2) gain-of-function mutations (affecting factor B and C3). Herein, we present a case of CM-TMA with alternation of complement factor H (CFH) function, caused by combined exon 23 c.3572C>T/c.3590T>C, and exon 19 c. 2808G>T mutations.

A 55-year-old man was brought to our emergency department on October 25, 2021. The patient had suffered from malaise for a few days and was initially diagnosed with *Staphylococcus aureus* bacteremia. After antibiotic treatment, the patient`s clinical condition and infection improved. However, hemoglobinuria combined with AKI and oliguria developed on November 15. Moreover, thrombocytopenia and microangiopathic hemolytic anemia (MAHA) were noted. Blood smear consistent with MAHA include schistocyte and reduced platelets. Blood examination revealed low haptoglobin (<4 mg/dL), elevated lactate dehydrogenase, negative Coombs test (non-autoimmune hemolytic anemia), unconjugated hyperbilirubinemia, which are characteristic of with MAHA.

Under the impression of TMA, we initially excluded other causes of TMA, including systemic lupus erythematosus, disseminated intravascular coagulation, cobalamin deficiency, cancer, Shiga-toxin-associated hemolytic uremic syndrome …et al. Thereafter, ADAMTS13 activity test sent and therapeutic plasma exchange arranged due to consideration of thrombotic thrombocytopenic purpura.

However, the patient had ADAMTS13 activity > 10 percent. Subsequently, genetic analysis was performed due to consideration of CM-TMA. The gene report showed exhibited CFH mutation (exon 23 c.3572C>T, S1191L / c.3590 T>C, V1197A and exon 19 c. 2808G>T, E936D). We considered eculizumab, an anti-complement 5 monoclonal antibody, for the treatment of CM-TMA. The patient underwent hemodialysis therapy for AKI and continued receiving TPE before eculizumab availability. Anemia and thrombocytopenia remained inactive under the current treatment.

病例報告 2

2-9.

一位因大腸癌而服用 Afatinib 及曾經因為急性肺栓塞使用 Edoxaban 的病人，發生急性腎衰竭、蛋白尿、血尿被診斷為 IgA 腎病變

A male with history of colon cancer with Afatinib treatment and acute pulmonary embolism under Edoxaban control, happened with acute kidney injury, proteinuria and hematuria, was diagnosed with IgA nephropathy.

李璥廷* 陳銳溢
Ching-Ting Lee* Jui-Yi Chen
奇美醫學中心內科部腎臟科
Nephrology Division, Internal Medicine Department, Chi Mei Medical Center

This 48-year-old man had past history of ascending colon cancer, pT3N2bM1a with liver metastases status post laparoscopic right hemicolectomy, cholecystectomy, hepatectomy, status post completed chemotherapy FOLFIRI + capecitabine several times and maintaining single regimen of afatinib three months ago. Besides, he had acute pulmonary embolism and Edoxaban was also prescribed three months ago. On top of that, poorly controlled hypertension(SBP around 170-190 mmHg) was found for half year. This time, he suffered from persistent hematuria, foamy urine and bilateral lower limbs edema for three months with progressive higher blood pressure. The lab data showed the kidney function declining gradually with proteinuria (serum creatinine, sCr, 1.08 to 3.31 mg/dL; estimated glomerular filtration rate, eGFR, 73 to 20 mL/min/1.73m^2. Urine protein/creatinine ratio UPCR, 2827.3 mg/g) found at oncology and nephrology out-patient department from July to September, 2021. However, he denied fever, cough, dyspnea, abdominal pain, flank pain, dysuria, nor petechiae. Therefore, he was admitted for kidney biopsy of unknown cause of acute kidney injury.

At admission on October,7th, 2021, the body-mass index with the weight in kilograms divided by the square of the height in meters was 30.5 kg/m². The vital signs were as following: body temperature 36.4°C, blood pressure 174/115 mmHg, pulse rate 99 beats per minute, respiratory rate 17 breaths per minute, and the oxygen saturation 96-100%, room air. The blood examination showed normocytic anemia (Hb, 11.3 g/dL; MCV, 82.4 fL), impairment of renal function (sCr, 2.87 mg/dL; eGFR, 23.6 mL/min/1.73m^2); Anti-HBc T, Positive; IgE, 906.9 IU/mL; Antinuclear Ab, Positive. In addition, other markers were normal, such as electrolyte; liver function; coagulation time. Anti-ds DNA; CEA; CA19-9; Anti-GBM, 1.1 U/mL; p-ANCA(MPO), <0.2 IU/mL; c-ANCA(PR3), 0.3 IU/mL.

The urine analysis showed proteinuria and microscopic hematuria with dysmorphic RBC. (Blood, 3+; Sediment-RBC, >100 /HPF; Dysmorphic RBC ratio,50%; UPCR, 2827.3 mg/g; Sediment-WBC,0-5 /HPF) The renal echography in August, 26th,2021 showed no evidence of hydronephrosis with bilateral normal kidney size (right kidney, 12.7cm; left kidney, 10.8cm, longitudinally).

Finally, the kidney biopsy pathological report revealed IgA neprhopathy with focal segmental sclerosis, endocapillary proliferation, and crescent formation found at six over eleven glomeruluses (M1S1E1T1C2, by Oxford Classification System); acute tubular injury and interstitial nephritis with eosinophils; hypertensive renovascular disease. Due to IgA nephropathy with proteinuria and acute kidney injury progressively, we prescribed Prednisolone 15mg TID and gradually tapered every two weeks till 5mg TID now. Besides, Nifedipine 30mg BID, Hydralazine 25mg QD, Valsartan 80mg QD and Carvedilol 25mg QD were given for blood pressure control. The following lab data showed improving renal function, (SCr, 2.87 to 1.94 mg/dL), decreased proteinuria (UPCR, 2827 to 392 mg/g) and improved blood pressure (SBP 110 mmHg) since September, 2021 to November, 2022.

Under stable condition, he continued his chemotherapy of Regorafenib 80mg QD for colon cancer and kept warfarin 2.5mg QD for pulmonary embolism.

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ଌ 感謝上列廠商之支持 ଌ

交通資訊

日期：111 年 4 月 17 日(星期日)
地點：高雄醫學大學國際學術研究大樓 B2 國際會議中心
(高雄市三民區十全一路 100 號)

- 因應疫情，進入學校須掃實名制登記。
- 請由同盟路正門進入，依指標往國際學術研究大樓 B2 前進。

★ 會議室 A 廳、B 廳及 C 廳—位於國際學術研究大樓 B2 國際會議中心
A1、A2 講堂—位於勵學大樓 (Lunch Symposium 1 及 2)

交通資訊

- 火車高雄站後站出口距本校約兩公里，車程約 5 分鐘。
- 小港機場轉搭計程車約 30 分鐘。
- 開車沿中山高速公路南下：
  1.下「鼎金交流道」於民族路左轉，再於同盟路右轉，即達本校。
  2.下「九如交流道」沿九如路往火車站方向，於自由路右轉，遇同盟路右轉，即達本校。
- 高鐵車站轉搭計程車約 17 分鐘，沿大中路於自由路右轉，再於同盟路左轉即達本校 。
- 高捷車站轉搭捷運接駁公車，由後驛站出入口 2 搭乘紅 29 接駁車，即達本校。

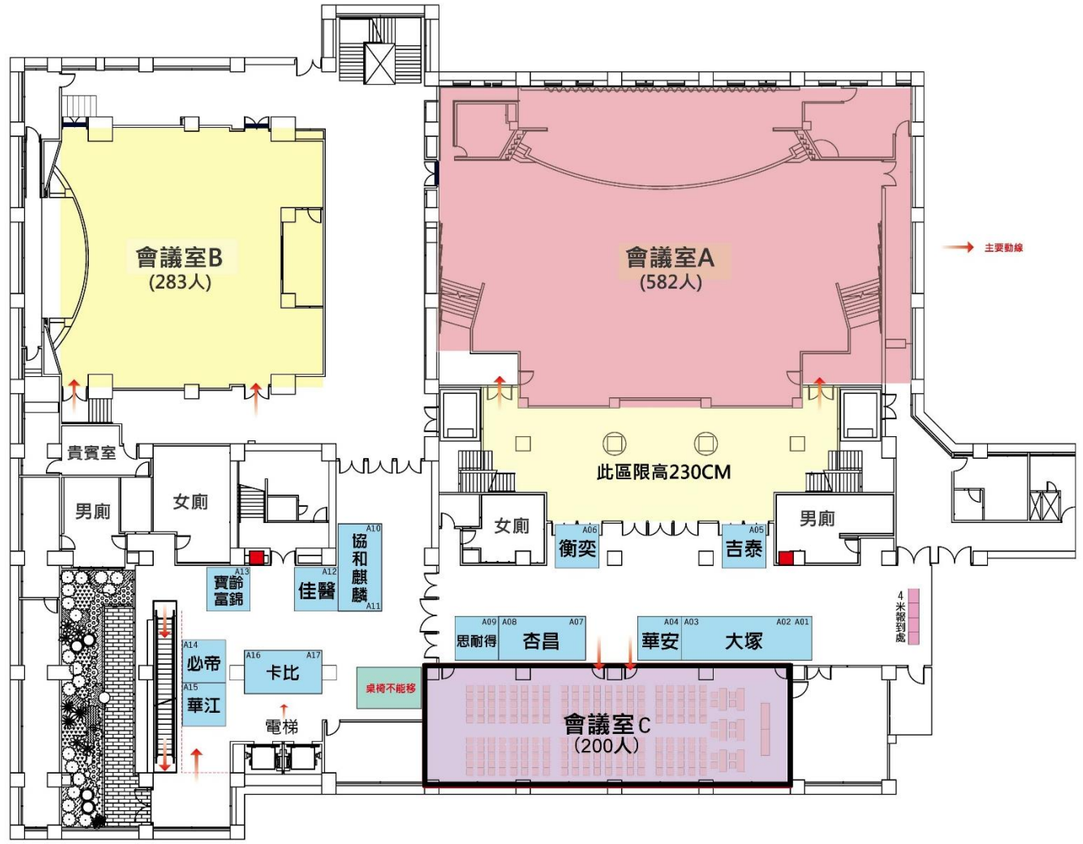
停車資訊

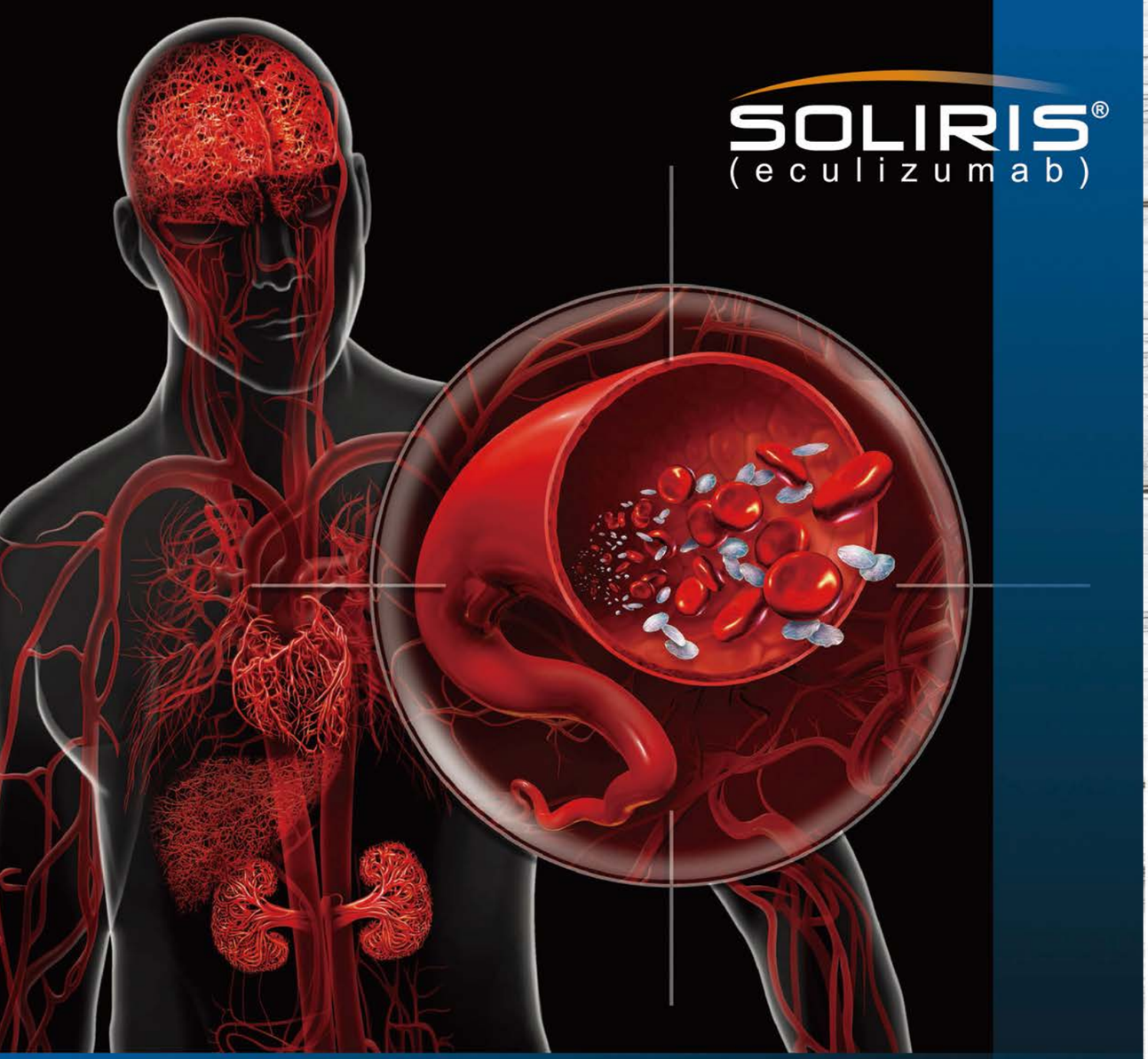
- 運動場地下停車場：在校園內，由同盟路進入。
- 同盟路上路邊收費停車格。

台灣腎臟醫學會 111 年度春季學術演講會

會場平面圖

台灣腎臟醫學會 111 年度春季學術演講會
高雄醫學大學國際學術研究大樓 B2 國際會議中心





【適應症】

治療對血漿治療反應不佳之非典型溶血性尿毒症候群(aHUS)病人。

【說明】

過去一星期內接受至少4次血漿治療後血小板計數低於正常值的病人。

【使用限制】

舒立瑞不可以用於治療Shiga toxin E. coli相關的溶血性尿毒症候群的病人。

警告：嚴重腦膜炎球菌感染

進行舒立瑞治療的病人，曾有過發生危及性命與致死性腦膜炎球菌感染症的案例。腦膜炎球菌感染若未被察覺及早治療，可能迅速發展到危及性命或致死的程度。

- 應遵守傳染病防治諮詢委員會預防接種組(ACIP)對腦膜炎預防注射的最新建議給予補體缺乏病人接種腦膜炎球菌疫苗。
- 除非延後給予舒立瑞療法的危險性遠大於出現腦膜炎球菌感染的危險性，否則應安排病人接種腦膜炎球菌疫苗2週後，再施打第一劑舒立瑞。
- 監測病人是否有腦膜炎球菌感染的早期病徵，若懷疑受到感染應立即進行評估。

舒立瑞Soliris® (eculizumab)簡易仿單

適應症：治療陣發性夜間血紅素尿症(PNH)病人。說明：舒立瑞治療需要輸血或曾有血栓併發症的病人。治療對血漿治療反應不佳之非典型溶血性尿毒症候群(aHUS)病人。說明：過去一星期內接受至少4次血漿治療後血小板計數低於正常值的病人。使用限制：舒立瑞不可以用於治療Shiga toxin E. coli相關的溶血性尿毒症候群的病人。**禁忌**：奈瑟氏菌腦膜炎感染症尚未痊癒者。除非延後給予舒立瑞療法的危險性遠大於出現腦膜炎球菌感染的危險性，否則禁用於最近未施打奈瑟氏菌腦膜炎疫苗者。**警告及注意事項**：使用舒立瑞會增加腦膜炎球菌感染的風險；所有病人應接種腦膜炎球菌疫苗，兒童應依據傳染病防治諮詢會預防接種組(ACIP)準則給予預防肺炎鏈球菌與B型流行性感冒嗜血桿菌(Hib)感染的疫苗接種。接種疫苗可降低腦膜炎球菌感染的風險，但未能完全消除感染的風險。應密切監測病人是否有腦膜炎球菌感染的早期病徵與症狀，若懷疑受到感染應立即進行評估。任何有全身性感染的病人給予舒立瑞時，應特別小心。**特定族群用藥**：孕婦關於孕婦使用Soliris後懷孕結果的數據有限，尚未發現有特定不良發育結果的憂慮。授乳在治療過程中不建議餵母乳。**兒科用藥**舒立瑞治療PNH兒科病人的安全性和有效性尚未建立。舒立瑞治療aHUS兒科病人的安全性和有效性已確立。**不良反應**：嚴重腦膜炎球菌感染、其他感染，曾有包含播散性淋球菌感染之嚴重感染奈瑟氏球菌（腦膜炎奈瑟球菌以外）感染的報導、及輸注反應。在臨床試驗中出現的不良反應包括頭痛、鼻咽炎、背痛、嘔心、疲累、咳嗽、感染、腹瀉、嘔吐、肌肉/四肢/關節痛、皮疹、白血球減少、貧血、泌尿道感染、發燒、上呼吸道感染、類流感症狀等(詳細內容請參見仿單說明)。**給藥劑量**：PNH舒立瑞療法如下：頭4週每週給予600 mg，第五週後給予第5次劑量900 mg，然後每2週給予900 mg。aHUS對於18歲以及18歲以上病人，舒立瑞療法如下: 頭4週每週給予900 mg，接著第五週後給予第5次劑量1200 mg，然後每2週給予　1200　mg。18歲以下病人，舒立瑞劑量依據體重給予，在大於40kg的18歲以下病人，給藥劑量與18歲以上病人相同。**劑量調整**：　舒立瑞應按照建議劑量療法的時間點或在這些時間點前後兩天內給藥。同時進行血漿分離術-plasmapheresis，或血漿置換或輸注新鮮冷凍血漿(PE/PI)的aHUS病人需給予舒立瑞補充劑量。**備藥及給藥**：將適當量(藥量與稀釋液同體積)的0.9%氯化鈉注射用溶液、0.45%氯化鈉注射用溶液、5%葡萄糖水注射液或林格氏注射用溶液加入輸注袋中，使舒立瑞稀釋成最終濃度5 mg/mL。以超過35分鐘的時間靜脈輸注投與舒立瑞稀釋液(成年病人的總輸注時間不可超過2小時，兒科病人最長不超過為4小時)。輸注完畢後，病人應留觀至少1小時，監測是否有輸注反應的徵兆或症狀。修訂版本TW04 (US PI 07/2018)。

使用前請詳閱說明書警語及注意事項

ALEXION
衛部罕菌疫輸字第 000016 號
北市衛藥廣字第 109070139 號

Alexion-Soliris-202006005 (2020 Jun)

僅供專業醫護人員參考

forxiga™
(dapagliflozin)
福適佳

*針對第二型糖尿病

福適佳 膜衣錠 5 毫克、10 毫克Forxiga (Dapagliflozin) Film-coated Tablets 5 mg , 10 mg 衛部藥輸字第026475號, 衛部藥輸字第026476號

【**適應症**】1. 第二型糖尿病：血糖控制：配合飲食和運動，改善第二型糖尿病成人病人的血糖控制。預防心血管事件：用於具第二型糖尿病且已有心血管疾病(CVD)或多重心血管風險因子的成人病人時，可降低心衰竭住院的風險。預防腎臟病：降低慢性腎臟病(CKD)新發生或惡化的風險。2.心衰竭：用於心衰竭(NYHA分類第二至四級) 且心室射出分率降低(≤ 40%)的成人病人時，可降低心血管死亡和心衰竭住院的風險。3.慢性腎臟病：用於治療有惡化風險之慢性腎臟病的成人病人時，可降低持續性腎絲球過濾率(eGFR)下降、末期腎病(ESKD)、心衰竭住院和心血管死亡的風險。【**使用限制**】不建議Forxiga　用於第一型糖尿病病人，其可能增加遠群病人糖尿病酮酸中毒之風險。不建議使用Forxiga作為eGFR低於45　mL/min/1.73　m²的第二型糖尿病病人的血糖控制，Forxiga基於其作用機制，在此情況下可能無效。不建議使用Forxiga用於治療多囊性腎臟病病人，或慢性腎臟病病人其病情需要或近期曾接受免疫抑制療法治療。Forxiga預計不會對這些病人族群有效。【**用法用量**】在開始Forxiga治療前應評估腎功能，之後依臨床需要進行評估。對於血容量不足的病人，應於在開始Forxiga治療前應評估血容量狀態，必要時應矯正血容量不足的情形。建議劑量：1. eGFR 45或以上：為改善血糖控制，Forxiga的建議起始劑量是5毫克每天口服1次。在耐受Forxiga 5毫克每天1次的病人，需要對於額外血糖控制時，劑量可增至10毫克每天口服1次。對於所有其他適應症的建議起始劑量是每天口服1次10毫克。2. eGFR 25至小於45：每天口服1次10毫克。3. eGFR小於25：針對此類病人不建議開始治療，然而Forxiga治療後，eGFR降低至小於25的病人，可持續使用以降低eGFR下降、ESKD、心血管死亡和心衰竭住院的風險。4.　透析病人，禁用Forxiga。【**禁忌**】對Forxiga嚴重過敏反應病史，如過敏反應或血管性水腫。透析病人。【**警語和注意事項**】於糖尿病病人的酮酸中毒：使用Forxiga治療的病人，若出現嚴重代謝性酸中毒的症狀，無論血糖值為何，皆應評估是否發生酮酸中毒。當懷疑是酮酸中毒時，應停用Forxiga，並評估病人狀況立即採取適當的治療。在開始使用Forxiga前，應考慮病人病史中可能容易產生酮酸中毒的因素，包括任何原因所導致的胰島素不足、熱量限制及酗酒。預計接受非緊急、選擇性手術的病人，應考慮至少3天前暫時中斷Forxiga。血容量不足：Forxiga可導致血管內容積減少，有時可能引起症狀性低血壓或肌酸酐急性短暫變化。腎功能不全的病人、老年病人或使用環利尿劑的病人，發生血容量不足或低血壓的風險可能增加。尿路敗血症和腎盂腎炎：接受SGLT2抑制劑(包括Forxiga)的病人，有發生因嚴重泌尿道感染，包括尿路敗血症和腎盂腎炎，而需要住院的通報案例。與胰島素和胰島素分泌促進劑同時使用伴隨的低血糖：當與胰島素或胰島素分泌促進劑併用，Forxiga可能會增加低血糖風險。會陰部壞疽性筋膜炎　(弗尼爾氏壞疽)：對於接受Forxiga治療如有出現生殖器或會陰區域疼痛或壓痛、紅斑或腫脹並伴隨發燒或身體不適的病人，皆應評估是否發生壞死性筋膜炎。生殖器黴菌感染：Forxiga會增加生殖器黴菌感染風險。【不良反應】於糖尿病病人的酮酸中毒、血容量不足、尿路敗血症和腎盂腎炎、與胰島素和胰島素分泌促進劑同時使用伴隨的低血糖、會陰部壞疽性筋膜炎（弗尼爾氏壞疽）、生殖器黴菌感染。

【使用前詳閱說明書簡語及注意事項，詳細仿單資料備索。】
【僅限醫藥專業人員參考，處方藥物請參考衛生福利部核准仿單說明書。】
北市衛藥廣字110090086 號

AstraZeneca
臺灣阿斯特捷利康股份有限公司 台北市敦化南路二段207號21樓
電話：(02) 2378-2390 傳真：(02) 2377-0914 http://www.astrazeneca.com.tw

Reference: FORXIGA仿單(2021.08)
TW-16624_FOX_31/08/2021