糖尿病血糖控制及併發症

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彰基體重管理及糖尿病

健康管理中心門診醫師

FB 莊武龍醫師

http://dmnote.tw

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無藥可醫？

《逆轉營養素》

讓你不藥而解！

不用藥的營養療法，
不是什麼病都需要吃藥

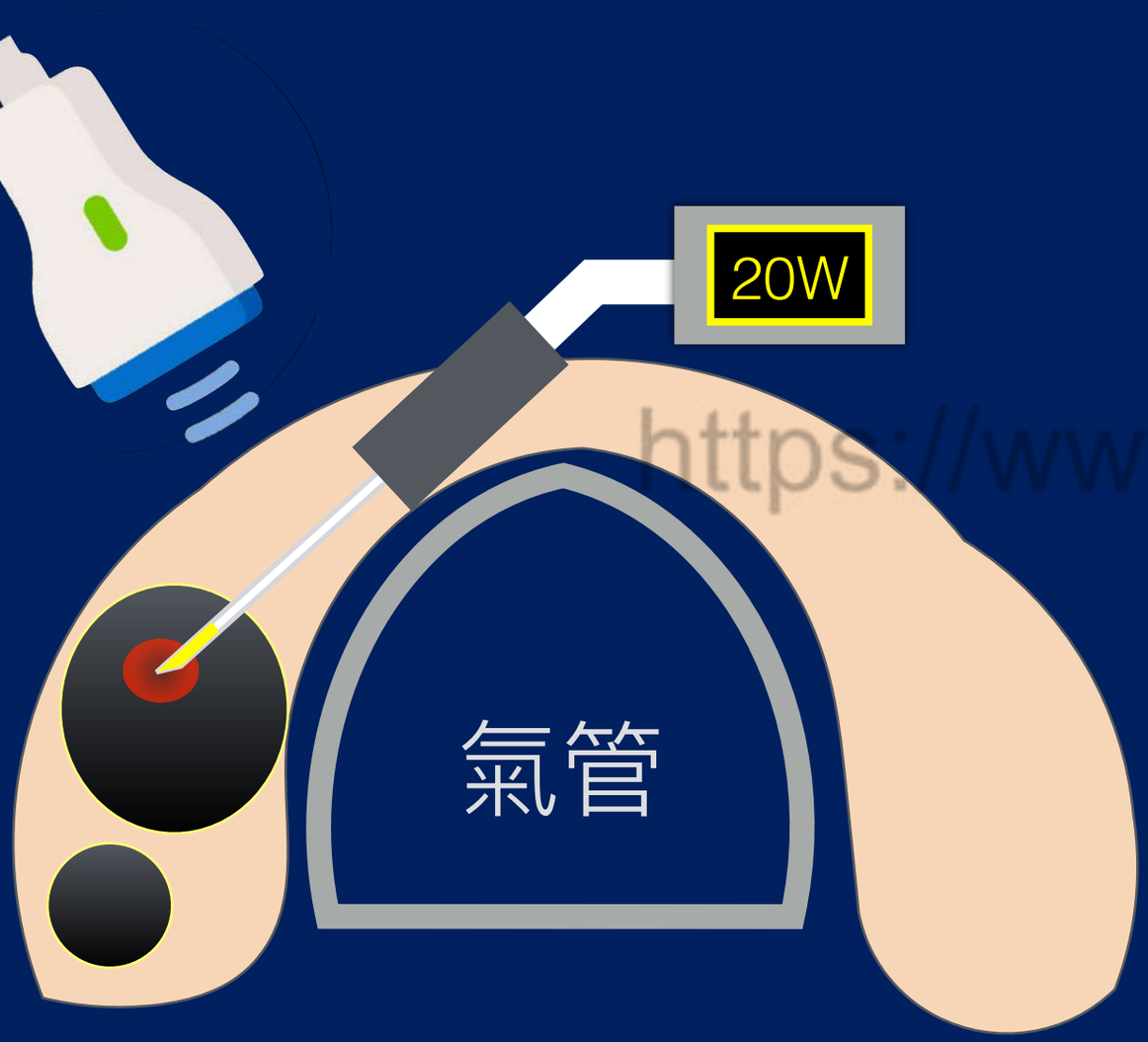
莊武龍 醫師 著

臨床照護經驗

連續 傷口 神經

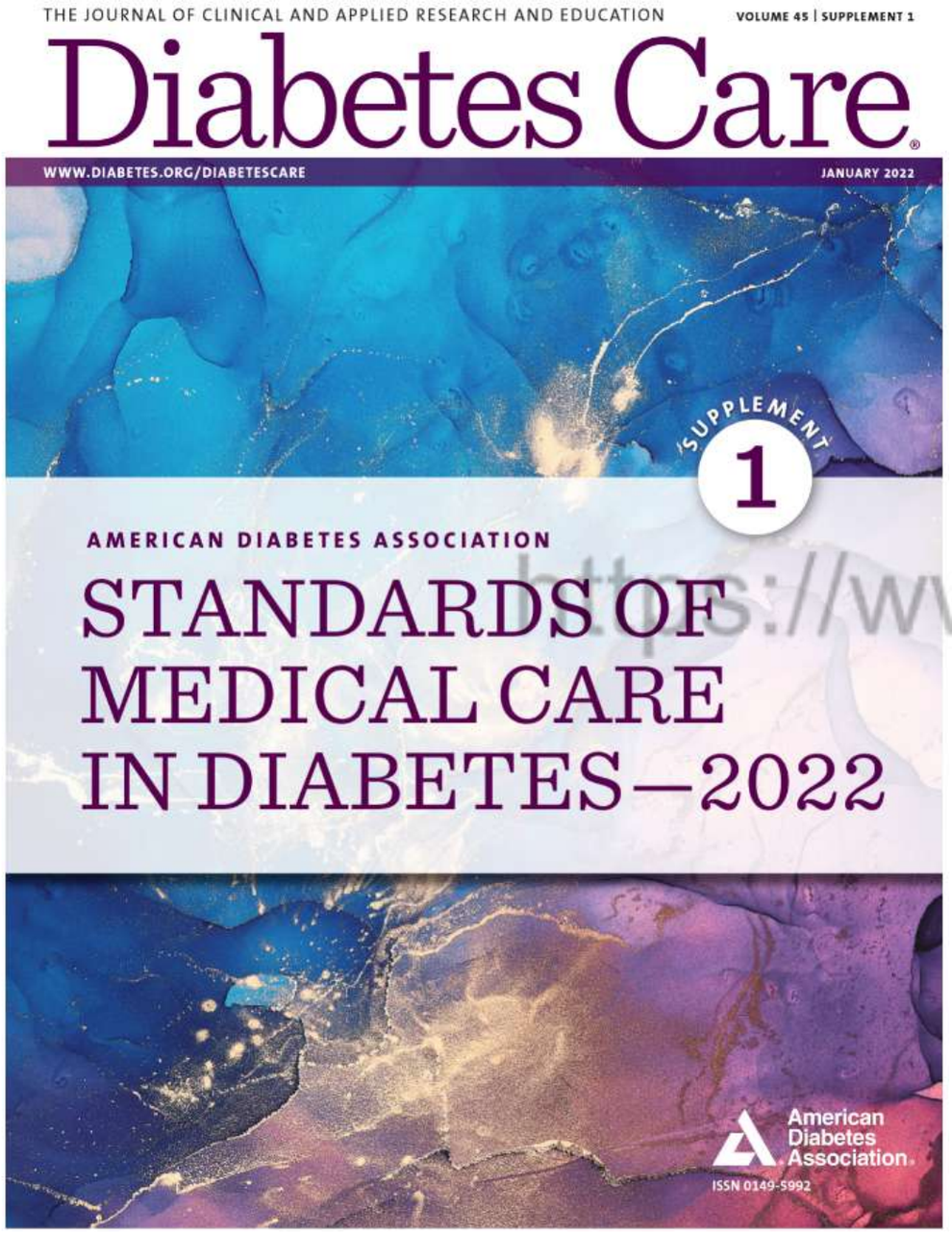
血糖 照護 病變

主治超音波介入治療



- 甲狀腺結節消融治療
  →改善結節壓迫症狀
- 超音波導引神經解套
  →改善神經麻、痛問題
- 星狀神經節阻斷治療
  →改善頭暈、耳鳴
  →改善更年期症狀
  →改善自律神經失調

2022 ADA 美國糖尿病治療指引



American Diabetes Association®

2018 糖尿病臨床照護指引

DAROC Clinical Practice Guidelines for Diabetes Care 2018

糖尿病臨床照護指引摘要

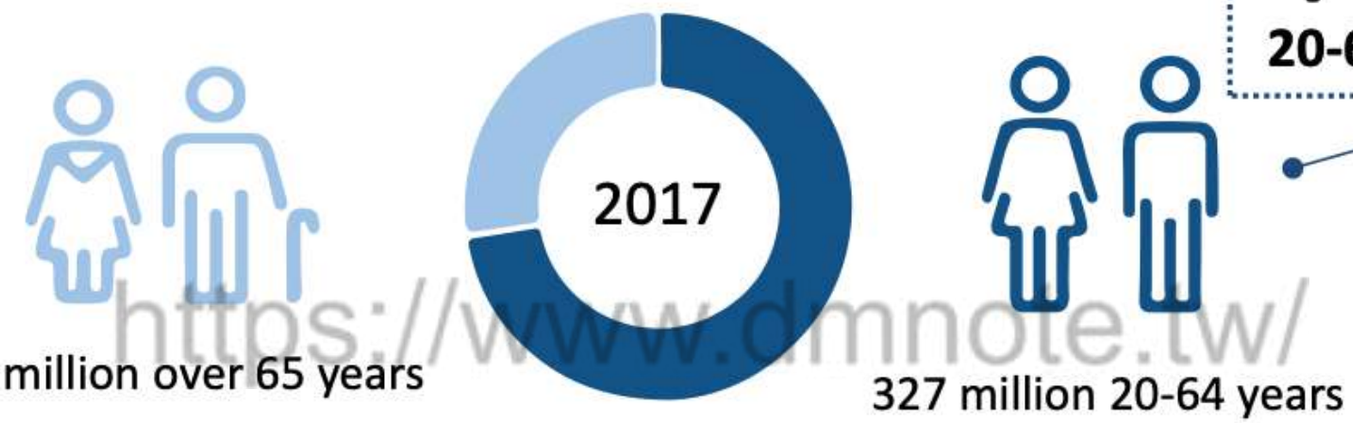
社團法人中華民國糖尿病學會 編著

糖尿病診斷與分類

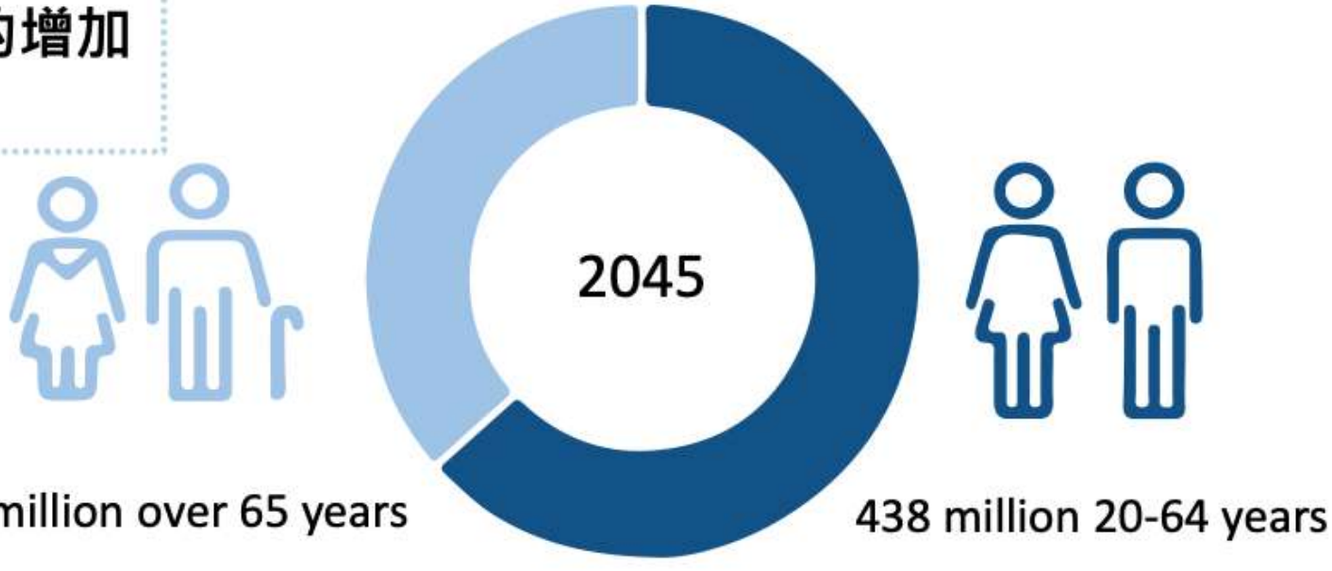
全球糖尿病患者的年齡分析與預測

Diabetes by Age

2/3 的糖尿病患者是
20-64 歲的工作人口

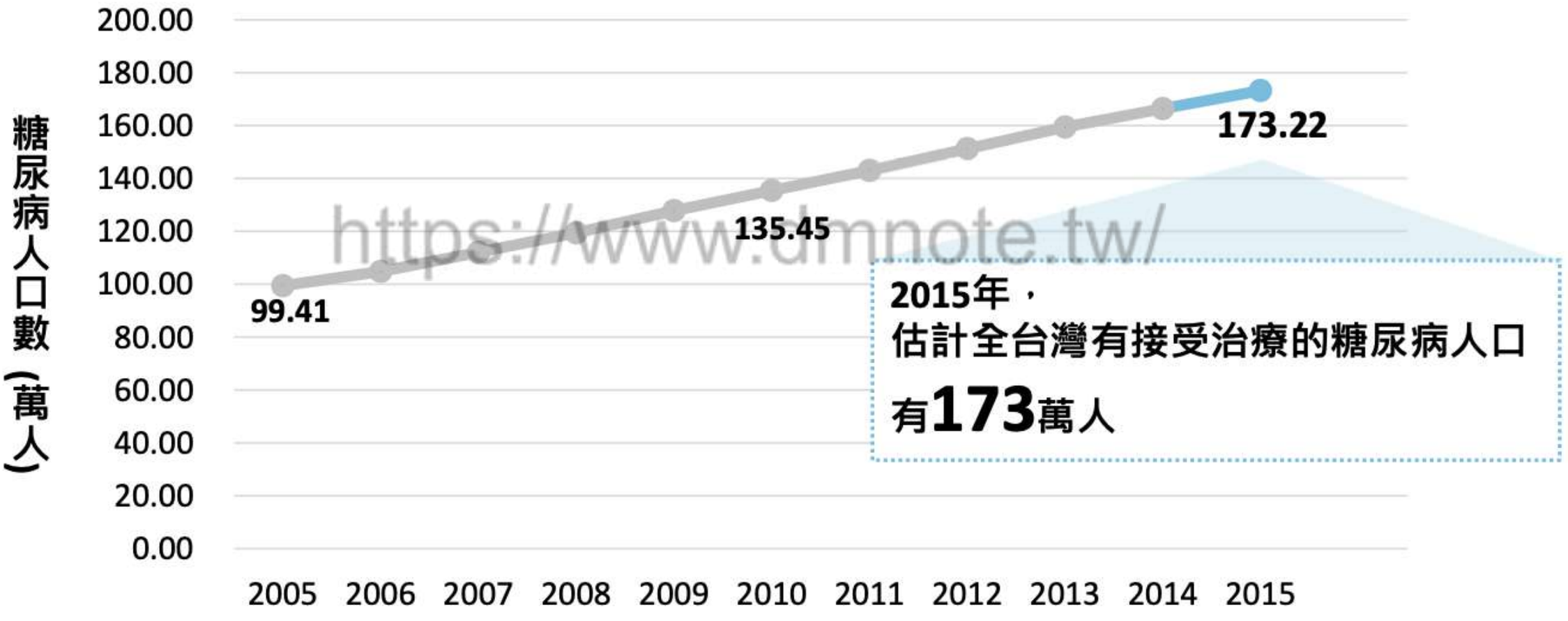


老年糖尿病患者的增加
也是重要議題



IDF Diabetes Atlas 8E ExSummary, file:///C:/Users/EN/Downloads/IDF%20Diabetes%20Atlas%208E%20ExSummary.pdf

2005-2015年，
台灣糖尿病人口數增加了 74%



*20-79歲成年人口數
資料來源:健保資料庫 (ie. 有接受治療的糖尿病人)

台灣糖尿病人口

推估 2014-2015 年時，

台灣有接受治療的糖尿病人口，

介於 173 萬到 220 萬人之間

全民健康保險研究資料庫 National Health Insurance Research Database	173 萬人
衛生福利部國民健康署 Health Promotion Administration, Ministry of Health and Welfare	220 萬人
IDF DIABETES ATLAS	178 萬人 (139~229 萬人)

105 年度十大死因死亡人數及死亡率

	死亡人數 (人)		死亡率 (每十萬人口)		標準化死亡率 (每十萬人口)	
	105 年	較上年增減 %	105 年	較上年增減 %	105 年	較上年增減 %
所有死亡原因	172,418	5.4	733.2	5.2	439.4	1.8
惡性腫瘤	47,760	2.0	203.1	1.8	126.8	-0.9
心臟疾病 (高血壓性疾病除外)	20,812	8.4	88.5	8.1	50.3	4.7
肺炎	12,212	13.5	51.9	13.2	269.0	9.3
腦血管疾病	11,846	6.1	50.4	5.8	28.6	2.4
糖尿病	9,960	4.5	42.4	4.3	24.5	0.8
事故傷害	7,206	2.5	30.6	2.2	23.1	1.2
慢性下呼吸道疾病	6				15.1	3.5
高血壓性疾病	5				13.5	2.3
腎炎、腎病症候群及腎病變	5				12.4	5.4
慢性肝病及肝硬化	4,738	1.1	20.1	0.8	13.4	-1.8

糖尿病居 105 年
國人十大死因第五名

衛生福利部105年國人死因統計結果. http://www.mohw.gov.tw/cp-16-33598-1.html.

糖尿病的診斷標準

空腹血漿葡萄糖 ≥126 mg/dL (7.0 mmol/L)
空腹的定義：至少 8 小時未攝取熱量*

或

口服葡萄糖耐受試驗
第 2 小時血漿葡萄糖 ≥200 mg/dL*

或

糖化血色素 ≥6.5%*

或

高血糖症狀 (包括多尿、頻渴和體重減輕)
且隨機血漿葡萄糖 ≥200 mg/dL (11.1 mmol/L)

*當數值在診斷標準附近時，建議進行重複測試以確認

American Diabetes Association. Classification and diagnosis of diabetes mellitus. Diabetes Care 2017; 40 (Suppl 1): S11-24.

糖尿病高風險群 (糖尿病前期) 的分類

1. **葡萄糖失耐 (IGT)：**
   口服葡萄糖耐受試驗第 2 小時血漿葡萄糖
   為 140-199 mg/dL (7.8-11.0 mmol/L)

或

2. **空腹血糖偏高 (IFG)：**
   空腹血漿葡萄糖值 100-125 mg/dL (5.6-6.9 mmol/L)

或

3. **糖化血色素：5.7-6.4%**

IGT: Impaired glucose tolerance
IFG: Impaired fasting glucose

American Diabetes Association. Classification and diagnosis of diabetes mellitus. Diabetes Care 2017; 40 (Suppl 1): S11-24.

第 1 型和第 2 型糖尿病的鑑別診斷

	第 1 型糖尿病	第 2 型糖尿病
發病年齡	通常小於 30 歲	通常大於 40 歲
發病症狀	急性 - 有明顯症狀	慢性 - 通常無症狀
臨床表現	體型瘦 體重減輕 多尿 頻渴	肥胖 有明顯的第 2 型糖尿病家族史 種族 - 高盛行率的族群 黑色棘皮症 (acanthosis nigricans) 多囊性卵巢症候群 (PCOS)
酮酸中毒	較常出現	通常沒有
空腹血清 C-胜肽濃度	低或無法偵測	低、正常或高
升糖素刺激後，血清 C-胜肽濃度	低或無法偵測	低、正常或高
自體抗體 (包括 ICA, GADA, IA-2A, IAA 及 ZnT8Ab)	較常出現	通常沒有
治療	依賴胰島素	改變生活型態、口服抗糖尿病藥或胰島素
自體免疫疾病的關聯性	多數有	無

ICA: Islet Cell Cytoplasmic Autoantibodies; **GADA:** Glutamic Acid Decarboxylase Autoantibodies; **IA-2A:** Insulinoma-Associated-2 Autoantibodies; **IAA:** Insulin Autoantibodies; **ZnT8Ab:** Zinc Transporter 8 Autoantibodies

Alberti G, Zimmet P, Shaw J, Consensus Workshop Group. Diabetes Care 2004; 27 (7): 1798-811.

第 1 型和第 2 型糖尿病的鑑別診斷

單從血糖值並無法區分第 1 型或第 2 型糖尿病。
被視為第 1 型糖尿病的典型特徵 - 酮酸血症，
有時在第 2 型糖尿病人也會出現。

酮酸血症**並不是**診斷第1型糖尿病必要條件。

血清 C-胜肽濃度的判讀要考慮病人年齡及罹患糖尿病的時間

- 一般來說，第一型糖尿病人
  - 0 min plasma C-peptide <0.5 ng/ml
  - 6 min plasma C-peptide <1.8 ng/ml
  - Change of plasma C-peptide <0.7 ng/ml
- 血清C-peptide濃度與罹病時間有關，初診斷時較高，隨著罹病時間越久，血清中測不到C-胜肽的比例就越高
- 初診斷之第一型糖尿病病童與青少年(<18歲)
  - 0 min plasma C-peptide <2.1 ng/ml
  - 6 min plasma C-peptide <3.3 ng/ml

Madsbad S et al., Acta Med Scand, 1981. 210(3): p. 153-6; Juang JH et al., Taiwan Yi Xue Hui Za Zhi 1989. 88(6): p. 579-83
Davis A et al., Diabetes Care 2015. 38:p.476-481; Szendroedi J et al., Cardiovasc Diabetol 2016. 15:59
Paschke A et al., Polskie Archiwum Medycyny Wewnetrznej 2013. 123:p401-40; Tung YC et al., Eur J Pediatr 2008. 167: p. 801-5

猛爆性第1型糖尿病
(Fulminant type 1 diabetes)

是第1型糖尿病的亞型，
特徵為：

1. β細胞破壞過程非常快
2. 發病迅速
3. 幾乎沒有 C-胜肽分泌
4. 胰臟外分泌酶升高
   (如澱粉酶，脂肪酶和彈性蛋白酶-1)
5. 無胰島相關自體抗體
6. 胰島素分泌能力
   在發病後很少恢復

多見於亞洲國家如中國、韓國及菲律賓等，
各國之推估盛行率不等，
在日本約佔急性發病
第一型糖尿病患的 19.4%

日本糖尿病学会

日本糖尿病學會建議的
診斷標準，包含三必要條件：

1. 高血糖症狀發生後七天內出現糖尿病酮酸中毒
2. 最初的糖化血色素 <8.7%
   且血糖值 ≥288mg/dL
3. 發病初始升糖素刺激試驗若空腹血清 C-胜肽濃度 <0.3 ng/ml，且升糖素注射6分鐘後，血清 C-胜肽濃度 <0.5 ng/ml

Imagawa A et al. N Engl J Med 2000; 342:301-307
Imagawa A et al. J Diabetes Investig 2012; 3:536-539

成人遲發型自體免疫糖尿病
(Latent autoimmune diabetes in adults, LADA)

發病年齡較典型第 1 型糖尿病來的晚，發病初期病人不須接受胰島素治療，故臨床上容易被誤認為第 2 型糖尿病。

隨著疾病的進展，胰島 ß 細胞的功能漸進性地喪失，患者大多在發病六年內進展成胰島素依賴的狀態。因為上述特性，這類疾病有時也被稱為 1.5 型糖尿病 (Type 1.5 diabetes)。

Immunology of Diabetes Society 提出的 LADA 臨床診斷，需符合三個要素：

- 成人時期發病 (通常三十歲以後)
- 至少帶有一種和第1型糖尿病相關的自體免疫抗體 (如：ICA 512、IAA、anti-GAD65、IA-2A)
- 診斷糖尿病後的六個月內未使用胰島素治療

Naik RG et al. J Clin Endocrinol Metab 2009; 94:4635-4644

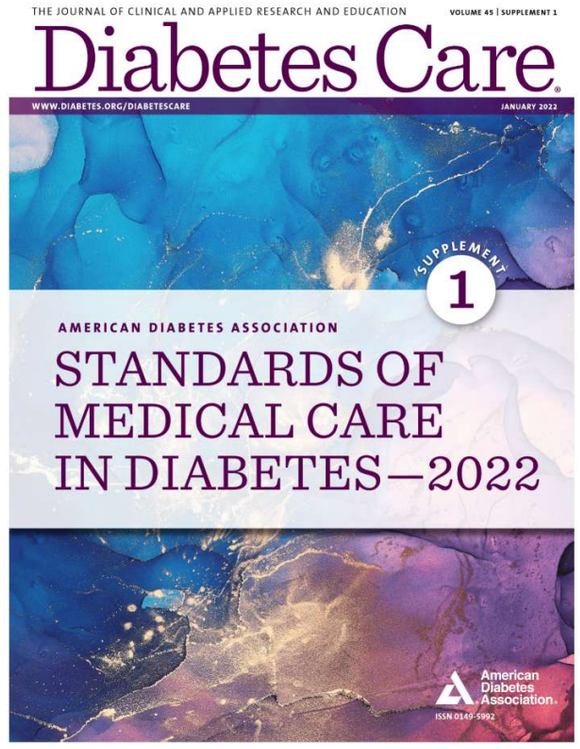
2022 ADA 美國糖尿病治療指引

CLASSIFICATION

Diabetes can be classified into the following general categories:

1. Type 1 diabetes (due to autoimmune β-cell destruction, usually leading to absolute insulin deficiency, including latent autoimmune diabetes of adulthood)
2. Type 2 diabetes (due to a progressive loss of adequate β-cell insulin secretion frequently on the background of insulin resistance)
3. Specific types of diabetes due to other causes, e.g., monogenic diabetes syndromes (such as neonatal diabetes and maturity-onset diabetes of the young), diseases of the exocrine pancreas (such as cystic fibrosis and pancreatitis), and drug- or chemical-induced diabetes (such as with glucocorticoid use, in the treatment of HIV/AIDS, or after organ transplantation)
4. Gestational diabetes mellitus (diabetes diagnosed in the second or third trimester of pregnancy that was not clearly overt diabetes prior to gestation)

This section reviews most common forms of diabetes but is not comprehensive. For additional information, see the American Diabetes Association (ADA) position statement "Diagnosis and Classification of Diabetes Mellitus" (1).



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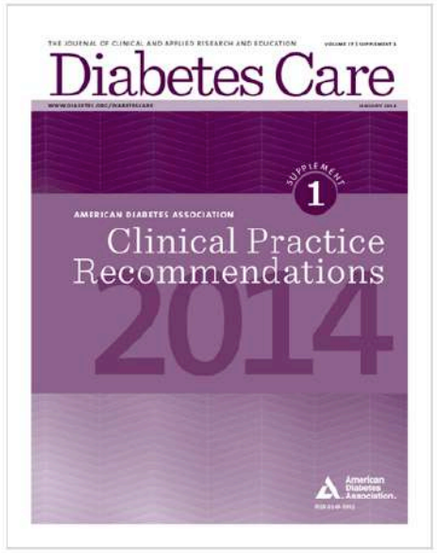
1 January 2014



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POSITION STATEMENT | DECEMBER 16 2013

Diagnosis and Classification of Diabetes Mellitus

American Diabetes Association

Diabetes Care 2014;37(Supplement_1):S81–S90

https://doi.org/10.2337/dc14-S081

Connected Content

A correction has been published: Standards of Medical Care in Diabetes—2014. Diabetes Care 2014;37(Suppl. 1):S14–S80

Diagnosis and Classification of Diabetes Mellitus. Diabetes Care 2014;37(Suppl. 1):S81–S90

Definition and Description of Diabetes Mellitus

Diabetes is a group of metabolic diseases characterized by hyperglycemia resulting from defects in insulin secretion, insulin action, or both. The chronic hyperglycemia of diabetes is associated with long-term damage, dysfunction, and failure of different organs, especially the eyes, kidneys, nerves, heart, and blood vessels.

Several pathogenic processes are involved in the development of diabetes. These range from autoimmune destruction of the pancreatic β-cells with consequent insulin deficiency to abnormalities that result in resistance to insulin action. The basis of the abnormalities in carbohydrate, fat, and protein metabolism in diabetes is deficient action of insulin on target tissues. Deficient insulin action results from inadequate insulin secretion and/or diminished tissue responses to insulin at one or more points in the complex pathways of hormone action. Impairment of insulin secretion and defects in insulin action frequently coexist in the same patient, and it is often unclear which abnormality, if either alone, is the primary cause of the hyperglycemia.

糖尿病的病因分類

Table 1—Etiologic classification of diabetes mellitus

I. Type 1 diabetes (β-cell destruction, usually leading to absolute insulin deficiency)
   A. Immune mediated
   B. Idiopathic

II. Type 2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with insulin resistance)

III. Other specific types
   A. Genetic defects of β-cell function
      1. MODY 3 (Chromosome 12, HNF-1α)
      2. MODY 1 (Chromosome 20, HNF-4α)
      3. MODY 2 (Chromosome 7, glucokinase)
      4. Other very rare forms of MODY (e.g., MODY 4: Chromosome 13, insulin promoter factor-1; MODY 6: Chromosome 2, *NeuroD1*; MODY 7: Chromosome 9, carboxyl ester lipase)
      5. Transient neonatal diabetes (most commonly ZAC/HYAMI imprinting defect on 6q24)
      6. Permanent neonatal diabetes (most commonly KCNJ11 gene encoding Kir6.2 subunit of β-cell K_{ATP} channel)
      7. Mitochondrial DNA
      8. Others
   B. Genetic defects in insulin action
      1. Type A insulin resistance
      2. Leprechaunism
      3. Rabson-Mendenhall syndrome
      4. Lipoatrophic diabetes
      5. Others
   C. Diseases of the exocrine pancreas
      1. Pancreatitis
      2. Trauma/pancreatectomy
      3. Neoplasia
      4. Cystic fibrosis
      5. Hemochromatosis
      6. Fibrocalculous pancreatopathy
      7. Others
   D. Endocrinopathies
      1. Acromegaly
      2. Cushing's syndrome
      3. Glucagonoma
      4. Pheochromocytoma
      5. Hyperthyroidism
      6. Somatostatinoma
      7. Aldosteronoma
      8. Others
   E. Drug or chemical induced
      1. Vacor
      2. Pentamidine
      3. Nicotinic acid
      4. Glucocorticoids
      5. Thyroid hormone
      6. Diazoxide
      7. β-Adrenergic agonists
      8. Thiazides
      9. Dilantin
      10. γ-Interferon
      11. Others
   F. Infections
      1. Congenital rubella
      2. Cytomegalovirus
      3. Others
   G. Uncommon forms of immune-mediated diabetes
      1. Stiff-man syndrome
      2. Anti-insulin receptor antibodies
      3. Others
   H. Other genetic syndromes sometimes associated with diabetes
      1. Down syndrome
      2. Klinefelter syndrome
      3. Turner syndrome
      4. Wolfram syndrome
      5. Friedreich ataxia
      6. Huntington chorea
      7. Laurence-Moon-Biedl syndrome
      8. Myotonic dystrophy
      9. Porphyria
      10. Prader-Willi syndrome
      11. Others

IV. Gestational diabetes mellitus

Patients with any form of diabetes may require insulin treatment at some stage of their disease. Such use of insulin does not, of itself, classify the patient.

Diabetes Care 2014;37(Supplement_1):S81–S90

糖尿病的病因分類-內分泌的影響

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Diabetes Care 2014;37(Supplement_1):S81–S90

糖尿病的病因分類-感染的影響

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      2. Anti-insulin receptor antibodies
      3. Others
   H. Other genetic syndromes sometimes associated with diabetes
      1. Down syndrome
      2. Klinefelter syndrome
      3. Turner syndrome
      4. Wolfram syndrome
      5. Friedreich ataxia
      6. Huntington chorea
      7. Laurence-Moon-Biedl syndrome
      8. Myotonic dystrophy
      9. Porphyria
      10. Prader-Willi syndrome
      11. Others

IV. Gestational diabetes mellitus

Patients with any form of diabetes may require insulin treatment at some stage of their disease. Such use of insulin does not, of itself, classify the patient.

Diabetes Care 2014;37(Supplement_1):S81–S90

Volume 43, Issue 6

June 2020

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E-LETTERS: OBSERVATIONS | MARCH 24 2020

Incidence of Type 2 Diabetes in Patients With Chronic Hepatitis C Receiving Interferon-Based Therapy FREE

Ming-Chieh Tsai ; Kai-Liang Kao; Hui-Chun Huang; Weishan Chen; Chun-Kai Fang; Fung-Chang Sung ; Shu-I Wu ; Robert Stewart

Corresponding author: Shu-I Wu, shuiwu624@gmail.com

Diabetes Care 2020;43(6):e63–e64

https://doi.org/10.2337/dc19-1704 **Article history**

Type 2 diabetes (T2D) has been associated with hepatitis C virus (HCV) infection as an independent risk factor. The increased insulin resistance induced by the HCV core protein or the treatment with interferon-based therapy (IBT) may have a role (1). It is generally agreed that IBT for patients with chronic hepatitis C (CHC) may trigger short-term insulin resistance. However, the long-term risk of developing T2D for CHC patients with IBT remains unclear. Studies suggested that if viral clearance is achieved after IBT, the risk of T2D might be reduced by two-thirds in CHC patients (2,3). An Italian study argued that a lower insulin resistance at baseline may be the reason for reduced risk of T2D (4). We therefore conducted a retrospective cohort study, using insurance claims data of Taiwan, to examine *1*) whether CHC patients are at an elevated risk of developing T2D and *2*) whether CHC patients receiving IBT are at a reduced risk of T2D, not only through viral clearance but also through possible alterations of glucose metabolism.

Volume 44, Issue 7

July 2021

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Introduction

Research Design and Methods

Results

Conclusions

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EPIDEMIOLOGY / HEALTH SERVICES RESEARCH | JULY 20 2021

Increased Incidence of Pediatric Type 1 Diabetes With Novel Association With Coxsackievirus A Species in Young Children but Declined Incidence in Adolescents in Taiwan FREE

Wei-Liang Shih; Yi-Ching Tung; Luan-Yin Chang ; Chi-Tai Fang; Wen-Yu Tsai

Corresponding author: Luan-Yin Chang, lychang@ntu.edu.tw

Diabetes Care 2021;44(7):1579–1585

https://doi.org/10.2337/dc20-1092 **Article history**

OBJECTIVE

Type 1 diabetes (T1D) has been linked to enterovirus infection in small population-based epidemiological studies. We investigated the secular relationship of T1D incidence with enterovirus infection and enterovirus species using nationwide population-based analysis.

RESEARCH DESIGN AND METHODS

We accessed the National Health Insurance Research Database of Taiwan to identify T1D and enterovirus infection cases from 2001 to 2015. Enterovirus serotype isolation rates were obtained from the nationwide laboratory surveillance systems. Negative binomial regression models assessed the incidence trend, and extended Cox proportional hazards models analyzed the association of enterovirus infection with T1D incidence. Spearman correlation coefficients evaluated the correlation between T1D incidence and circulating enterovirus species.

THE LANCET
Diabetes & Endocrinology

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ARTICLES | VOLUME 10, ISSUE 5, P311-321, MAY 01, 2022

Risks and burdens of incident diabetes in long COVID: a cohort study

Yan Xie, MPH • Ziyad Al-Aly, MD

Published: March 21, 2022 • DOI: https://doi.org/10.1016/S2213-8587(22)00044-4 •

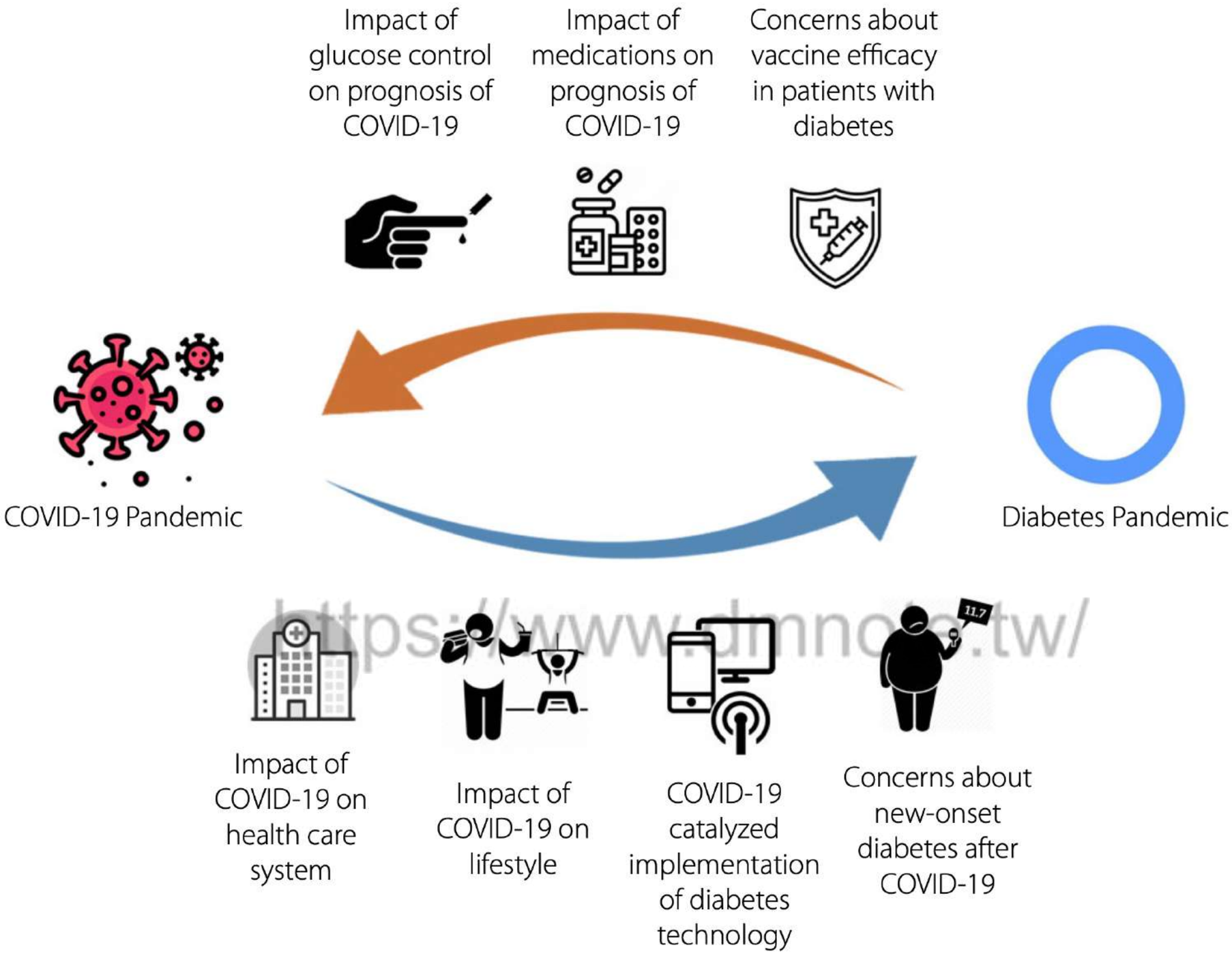

Figure 1 | The bidirectional relationship between diabetes and coronavirus disease 2019 (COVID-19). The impact of the diabetes pandemic on the COVID-19 pandemic includes: the association of glucose control and medication use with the prognosis of COVID-19; and the potential concerns about COVID-19 vaccine efficacy in patients with diabetes. The impact of the COVID-19 pandemic on the diabetes pandemic includes: the health care system, such as diabetes-related acute care; the lifestyle of patients with diabetes; catalyzing the implementation of diabetes technology; and the potential concerns about new-onset diabetes after COVID-19.

Front. Clin. Diabetes Healthc. 3:812134.

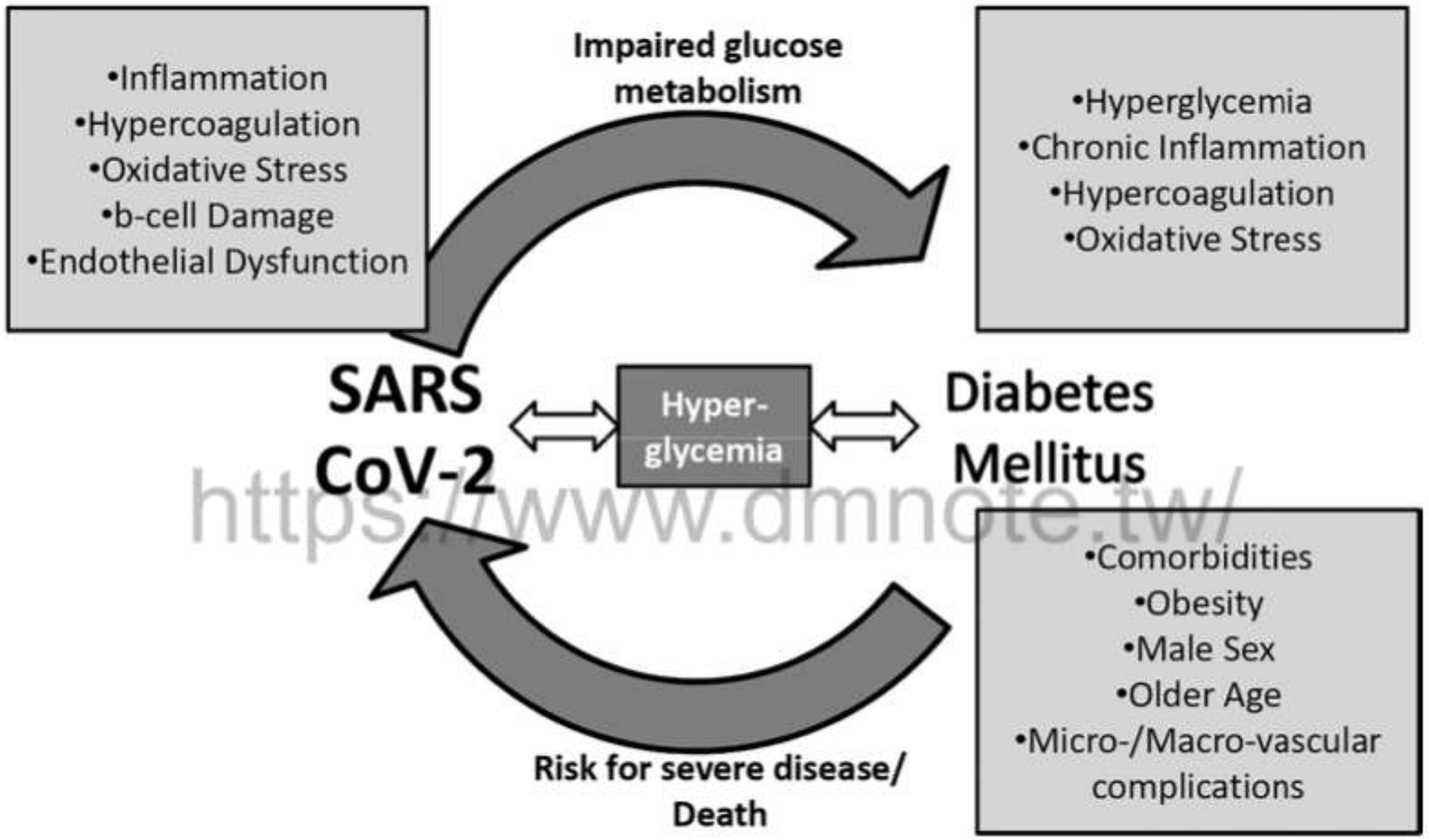

FIGURE 1 | The vicious cycle between diabetes mellitus, hyperglycemia, and severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2).

糖尿病的病因分類-藥物的影響

Table 1—Etiologic classification of diabetes mellitus

I. Type 1 diabetes (β-cell destruction, usually leading to absolute insulin deficiency)
   A. Immune mediated
   B. Idiopathic

II. Type 2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly secretory defect with insulin resistance)

III. Other specific types
   A. Genetic defects of β-cell function
      1. MODY 3 (Chromosome 12, HNF-1α)
      2. MODY 1 (Chromosome 20, HNF-4α)
      3. MODY 2 (Chromosome 7, glucokinase)
      4. Other very rare forms of MODY (e.g., MODY 4: Chromosome 13, insulin promoter factor-1; MODY 6: Chromosome 2, *NeuroD1*; MODY 7: Chromosome 9, carboxyl ester lipase)
      5. Transient neonatal diabetes (most commonly ZAC/HYAMI imprinting defect on 6q24)
      6. Permanent neonatal diabetes (most commonly KCNJ11 gene encoding Kir6.2 subunit of β-cell K_{ATP} channel)
      7. Mitochondrial DNA
      8. Others
   B. Genetic defects in insulin action
      1. Type A insulin resistance
      2. Leprechaunism
      3. Rabson-Mendenhall syndrome
      4. Lipoatrophic diabetes
      5. Others
   C. Diseases of the exocrine pancreas
      1. Pancreatitis
      2. Trauma/pancreatectomy
      3. Neoplasia
      4. Cystic fibrosis
      5. Hemochromatosis
      6. Fibrocalculous pancreatopathy
      7. Others
   D. Endocrinopathies
      1. Acromegaly
      2. Cushing's syndrome
      3. Glucagonoma
      4. Pheochromocytoma
      5. Hyperthyroidism
      6. Somatostatinoma
      7. Aldosteronoma
      8. Others
   E. Drug or chemical induced
      1. Vacor
      2. Pentamidine
      3. Nicotinic acid
      4. Glucocorticoids
      5. Thyroid hormone
      6. Diazoxide
      7. β-Adrenergic agonists
      8. Thiazides
      9. Dilantin
      10. γ-Interferon
      11. Others
   F. Infections
      1. Congenital rubella
      2. Cytomegalovirus
      3. Others
   G. Uncommon forms of immune-mediated diabetes
      1. Stiff-man syndrome
      2. Anti-insulin receptor antibodies
      3. Others
   H. Other genetic syndromes sometimes associated with diabetes
      1. Down syndrome
      2. Klinefelter syndrome
      3. Turner syndrome
      4. Wolfram syndrome
      5. Friedreich ataxia
      6. Huntington chorea
      7. Laurence-Moon-Biedl syndrome
      8. Myotonic dystrophy
      9. Porphyria
      10. Prader-Willi syndrome
      11. Others

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Patients with any form of diabetes may require insulin treatment at some stage of their disease. Such use of insulin does not, of itself, classify the patient.

Diabetes Care 2014;37(Supplement_1):S81–S90

JDI Journal of Diabetes Investigation
Official Journal of the Asian Association for the Study of Diabetes

Case Report | Open Access

Development of type 1 diabetes in a patient treated with anti-interleukin-6 receptor antibody for rheumatoid arthritis

Eiji Kawasaki, Takahiro Fukuyama, Aira Uchida, Yoko Sagara, Yuko Nakano, Hidekazu Tamai, Masayuki Tojikubo, Yuji Hiromatsu, Nobuhiko Koga

First published: 06 November 2021 | https://doi.org/10.1111/jdi.13706 | Citations: 1

Abstract

Interleukin-6 is a pleiotropic cytokine that plays a pathogenic role in type 1 diabetes. Therefore, anti-interleukin-6 receptor antibody, tocilizumab, used for the treatment of rheumatoid arthritis, is considered a candidate for immune intervention in type 1 diabetes. Here, we report the case of a 73-year-old woman (HLA-DR9-DQ3 homozygote) with well-controlled rheumatoid arthritis who developed type 1 diabetes while receiving tocilizumab treatment. At 57 years-of-age, the patient was diagnosed with rheumatoid arthritis, for which she underwent tocilizumab therapy that enabled complete suppression of her joint inflammation. A total of 17 months after starting tocilizumab therapy, she noticed polydipsia, polyuria, general fatigue and weight reduction (−2 kg/month), and was diagnosed with type 1 diabetes with diabetic ketoacidosis based on an arterial pH of 7.26, serum ketone body of 7,437 μmol/L, blood glucose level of 925 mg/dL, glycated hemoglobin of 13.2% and the presence of anti-islet autoantibodies. This case report shows valuable insight regarding the effect of anti-interleukin-6 receptor antibody therapy on type 1 diabetes prevention.

無症狀成人的糖尿病篩檢建議

①

利用國民健康署所提供的成人健康檢查：

≥ 40 歲以上民眾

每 **3** 年篩檢 1 次

≥ 65 歲以上民眾

每 **1** 年篩檢 1 次

②

台灣糖尿病風險評估公式顯示為：

中或**高**風險者

每 **3** 年篩檢 1 次

極高風險者

每 **1** 年篩檢 1 次

③

符合下列 A 或 B 情況者，也建議篩檢

A. 符合 ≥ 2 個危險因子者，建議篩檢：

- 身體質量指數 ≥24 kg/m^2 或腰圍男/女 ≥90/80 cm
- 一等親人罹患糖尿病
- 曾罹患心血管疾病
- 高血壓 (≥140/90 mmHg) 或正接受高血壓治療
- 高密度脂蛋白膽固醇 <35 mg/dL 或三酸甘油酯 >250 mg/dL
- 多囊性卵巢症候群的婦女
- 曾診斷為妊娠性糖尿病的婦女
- 缺乏運動
- 臨床上有胰島素阻抗的症狀 (例如：重度肥胖，黑色棘皮症)

篩檢結果未達糖尿病診斷標準者，建議至少每 3 年再檢測一次

B. 曾檢查為葡萄糖失耐、空腹血糖偏高、或 HbA_{1C} ≥5.7% 者，建議每年篩檢

台灣糖尿病風險評估公式

♂ 男性	X = - 8.3805 + 年齡 (歲)× 0.0325 + 腰圍 (cm)× 0.0423 + 如果有使用抗高血壓藥物加 0.5866 + 如果有糖尿病家族史加 0.2429
♀ 女性	X = - 9.523 + 年齡 (歲)× 0.0446 + 腰圍 (cm)× 0.0468 + 如果有使用抗高血壓藥物加 0.4264 + 如果有糖尿病家族史加 0.5060
⚠ 罹患糖尿病的風險 (%)	$1 / (1 + e^{-X})$

風險等級	潛藏糖尿病機率
極高	> 20 %
高	10 ~ 20%
中	5 ~ 10 %
低	< 5 %

糖尿病家族史指的是父母、祖父母或兄弟姊妹罹患糖尿病

連結網頁以獲得更多資訊：http://www.diabetes.org.tw

Lin JW et al., Diabetes Care 2009; 32 (12): 2294-6
Li HY et al., Diabetes Care 2010; 33 (2): e26

建議篩檢流程

敏感性：91.9%
特異性：100%
OGTT：14.7%

測定空腹血糖+糖化血色素

空腹血糖 <100 mg/dL
且糖化血色素 <5.7%

空腹血糖 <100 mg/dL
且 5.7% ≤ 糖化血色素 <6.1%

其他情況*

空腹血糖 ≥126 mg/dL
或糖化血色素 ≥6.5%

執行口服葡萄糖耐受試驗

第 2 小時血漿
葡萄糖 <200 mg/dL

第 2 小時血漿
葡萄糖 ≥200 mg/dL

正常

糖尿病前期

糖尿病

*包括空腹血糖 <100 mg/dL 且 6.1% ≤ 糖化血色素 <6.5%，以及 100 ≤ 空腹血糖 <126 mg/dL 且糖化血色素 <6.5%

Modified from Li HY et al., *J Diabetes Investigation* 2012; 3 (3): 259-265

2022 ADA 美國糖尿病治療指引

American Diabetes Association.
Connected for Life

Are you at risk for type 2 diabetes?

Diabetes Risk Test:

WRITE YOUR SCORE IN THE BOX.

1. How old are you? .....
Less than 40 years (0 points)
40–49 years (1 point)
50–59 years (2 points)
60 years or older (3 points)

2. Are you a man or a woman? .....
Man (1 point) Woman (0 points)

3. If you are a woman, have you ever been diagnosed with gestational diabetes?.....
Yes (1 point) No (0 points)

4. Do you have a mother, father, sister or brother with diabetes? .....
Yes (1 point) No (0 points)

5. Have you ever been diagnosed with high blood pressure? .....
Yes (1 point) No (0 points)

6. Are you physically active? .....
Yes (0 points) No (1 point)

7. What is your weight category? .....
See chart at right.

Height	Weight (lbs.)		
4' 10"	119–142	143–190	191+
4' 11"	124–147	148–197	198+
5' 0"	128–152	153–203	204+
5' 1"	132–157	158–210	211+
5' 2"	136–163	164–217	218+
5' 3"	141–168	169–224	225+
5' 4"	145–173	174–231	232+
5' 5"	150–179	180–239	240+
5' 6"	155–185	186–246	247+
5' 7"	159–190	191–254	255+
5' 8"	164–196	197–261	262+
5' 9"	169–202	203–269	270+
5' 10"	174–208	209–277	278+
5' 11"	179–214	215–285	286+
6' 0"	184–220	221–293	294+
6' 1"	189–226	227–301	302+
6' 2"	194–232	233–310	311+
6' 3"	200–239	240–318	319+
6' 4"	205–245	246–327	328+
	1 point	2 points	3 points

If you weigh less than the amount in the left column: 0 points

Adapted from Bang et al., Ann Intern Med 151:775–783, 2009 • Original algorithm was validated without gestational diabetes as part of the model.

ADD UP YOUR SCORE.

If you scored 5 or higher:

You are at increased risk for having type 2 diabetes. However, only your doctor can tell for sure if you do have type 2 diabetes or prediabetes, a condition in which blood glucose levels are higher than normal but not yet high enough to be diagnosed as diabetes. Talk to your doctor to see if additional testing is needed.

Type 2 diabetes is more common in African Americans, Hispanics/Latinos, Native Americans, Asian Americans, and Native Hawaiians and Pacific Islanders.

Higher body weight increases diabetes risk for everyone. Asian Americans are at increased diabetes risk at lower body weight than the rest of the general public (about 15 pounds lower).

Lower Your Risk

The good news is you can manage your risk for type 2 diabetes. Small steps make a big difference in helping you live a longer, healthier life.

If you are at high risk, your first step is to visit your doctor to see if additional testing is needed.

Visit diabetes.org or call 1-800-DIABETES (800-342-2383) for information, tips on getting started, and ideas for simple, small steps you can take to help lower your risk.

Learn more at diabetes.org/risktest | 1-800-DIABETES (800-342-2383)

Diabetes Risk Test | American Diabetes Association®

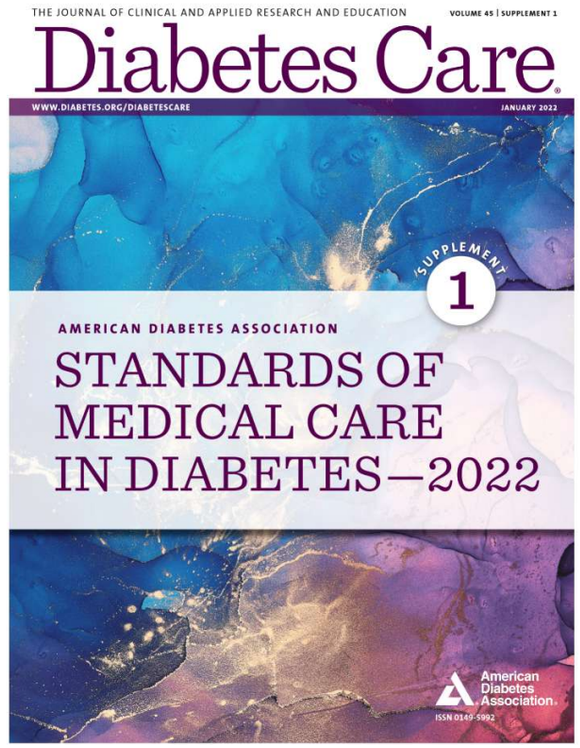


Figure 2.1—ADA risk test (diabetes.org/socrisktest).

糖尿病風險評估計算機

By 莊武龍醫師 晚上11:23 糖尿病 Edit

血糖控制

治療目標

成年人血糖控制目標

血糖目標		
	糖化血色素 (HbA_{1C})	< 7.0 % (需個別化考量)
	空腹 (餐前) 血糖	80-130 mg/dL
	餐後 1-2 小時血糖	**80**-160 mg/dL

- 餐後血糖控制目標加上**下限**

- 餐後血糖控制目標建議改為 **160 mg/dL**，以與本學會 "2012 餐後高血糖指引"、國際糖尿病聯盟 (IDF) " 2011 Guideline for Management of PostMeal Glucose in Diabetes" 之建議目標一致。

成年人血糖控制目標

個人化的血糖控制目標與 個別化考量的內容	較嚴格目標 (如 HbA_{1C} <6.5 %)	較寬鬆目標 (如 HbA_{1C} <8.5 %)
低血糖或其他治療相關副作用的風險	低	高
糖尿病罹病時間	短 (例如 <5 年)	長
預期壽命	長	短
相關共病	無	嚴重
糖尿病大小血管併發症	無或輕微	嚴重
病人與家屬態度與配合度	積極	消極
醫療資源與支持系統	佳	有限

兒童及青少年第 1 型糖尿病的血糖控制目標

血糖目標

空腹血糖	90-130 mg/dL
睡前血糖	90-150 mg/dL
A1C	<7.5 %

- 治療目標要依照病人實際情況而定 (individualized)。若低血糖的情況在可接受的範圍內，可考慮較嚴格的目標 (ex. HbA_{1C} <7%)。
- 如經常發生低血糖、無預知性的低血糖或血糖波動太大，則目標可適度放寬，並可考慮**連續血糖監測 (CGM)**。
- 在接受 basal-bolus 胰島素治療的孩童，當空腹血糖與 HbA_{1C} 的結果不一致時，要考慮測定餐後血糖值。

血壓控制目標

血壓目標

一般建議	< 140/90 mmHg
腎病變患者	< 130/80 mmHg

- 嚴格控制血壓至 130/80 mmHg 以下，對減少腎病變有好處，也可能對減少中風有好處，但對其他心血管疾病與死亡率的好處並不顯著。
- 目前對於積極的控制血壓，仍有正反不同的意見。
- 各學會建議之治療目標並不一致。

成年人糖尿病的治療目標

血糖	糖化血色素 (HbA1c)	<7.0% (需個別化考量)
	空腹 (餐前) 血糖	80-130 mg/dL
	餐後 2 小時血糖	80-160 mg/dL
血壓	一般建議	<140/90 mmHg
	腎病變患者	<130/80 mmHg
血脂肪 (首要目標)	低密度脂蛋白膽固醇 (LDL-C)	<100 mg/dL <70 mg/dL (如有心血管疾病)
血脂肪 (次要目標)	總膽固醇 (TCH)	<160 mg/dL
	非高密度脂蛋白膽固醇 (non-HDL-C)	<130 mg/dL <100 mg/dL (如有心血管疾病)
	高密度脂蛋白膽固醇 (HDL-C)	>40 mg/dL (男)；> 50 mg/dL (女)
	三酸甘油酯 (Triglyceride)	<150 mg/dL
生活型態改變	戒菸	強烈建議
	運動	中等強度有氧運動，建議每週 >150 分鐘; 較中等強度稍強的體能活動，建議每週至少 3 日，每日至少 20 分鐘。
	身體質量指數 (BMI)	18.5-24 kg/m^2
	腰圍	<90 cm (男)；<80 cm (女)

2022 ADA 美國糖尿病治療指引

Table 6.1—Estimated average glucose (eAG)

A1C (%)	mg/dL*	mmol/L
5	97 (76–120)	5.4 (4.2–6.7)
6	126 (100–152)	7.0 (5.5–8.5)
7	154 (123–185)	8.6 (6.8–10.3)
8	183 (147–217)	10.2 (8.1–12.1)
9	212 (170–249)	11.8 (9.4–13.9)
10	240 (193–282)	13.4 (10.7–15.7)
11	269 (217–314)	14.9 (12.0–17.5)
12	298 (240–347)	16.5 (13.3–19.3)

Data in parentheses are 95% CI. A calculator for converting A1C results into eAG, in either mg/dL or mmol/L, is available at professional.diabetes.org/eAG. *These estimates are based on ADAG data of ~2,700 glucose measurements over 3 months per A1C measurement in 507 adults with type 1, type 2, or no diabetes. The correlation between A1C and average glucose was 0.92 (12,13). Adapted from Nathan et al. (12).

Table 6.2—Standardized CGM metrics for clinical care

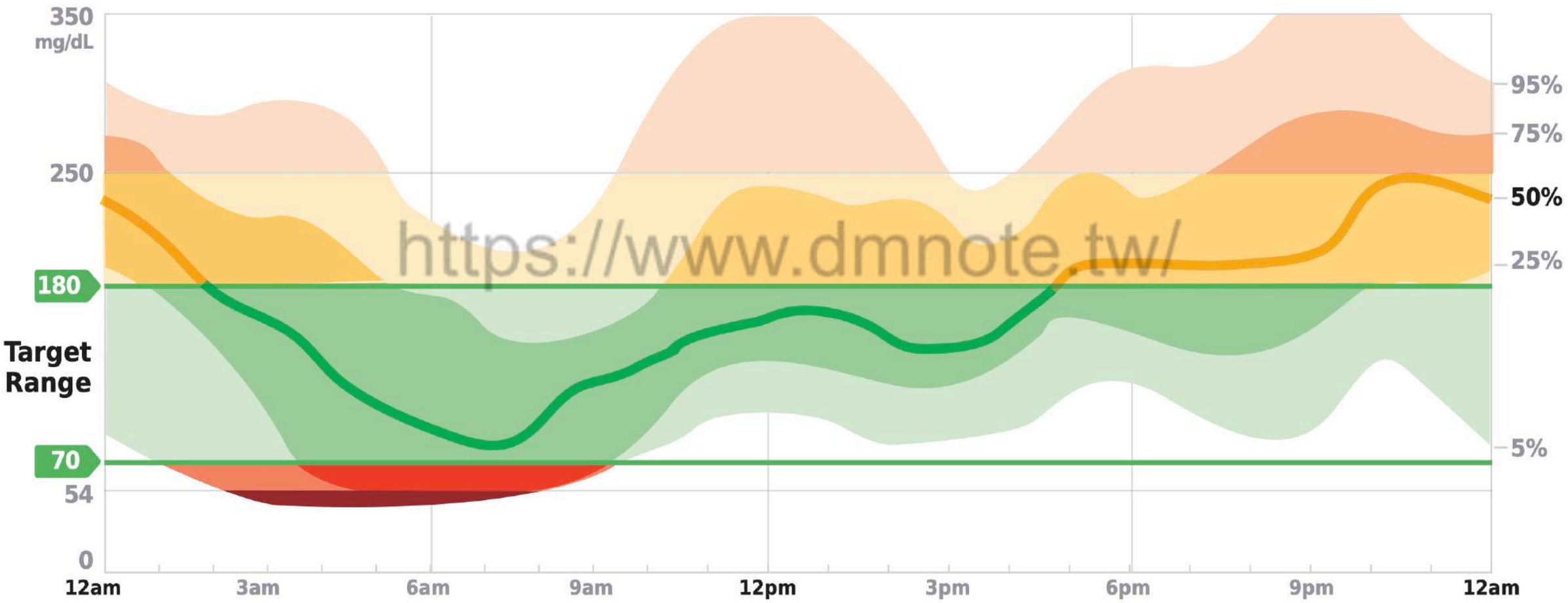
1. Number of days CGM device is worn (recommend 14 days)	
2. Percentage of time CGM device is active (recommend 70% of data from 14 days)	
3. Mean glucose	
4. Glucose management indicator	
5. Glycemic variability (%CV) target ≤36%*	
6. TAR: % of readings and time >250 mg/dL (>13.9 mmol/L)	Level 2 hyperglycemia
7. TAR: % of readings and time 181–250 mg/dL (10.1–13.9 mmol/L)	Level 1 hyperglycemia
8. TIR: % of readings and time 70–180 mg/dL (3.9–10.0 mmol/L)	In range
9. TBR: % of readings and time 54–69 mg/dL (3.0–3.8 mmol/L)	Level 1 hypoglycemia
10. TBR: % of readings and time <54 mg/dL (<3.0 mmol/L)	Level 2 hypoglycemia

CGM, continuous glucose monitoring; CV, coefficient of variation; TAR, time above range; TBR, time below range; TIR, time in range. *Some studies suggest that lower %CV targets (<33%) provide additional protection against hypoglycemia for those receiving insulin or sulfonylureas. Adapted from Battelino et al. (34).

2022 ADA 美國糖尿病治療指引

Ambulatory Glucose Profile (AGP)

AGP is a summary of glucose values from the report period, with median (50%) and other percentiles shown as if they occurred in a single day.



2022 ADA 美國糖尿病治療指引

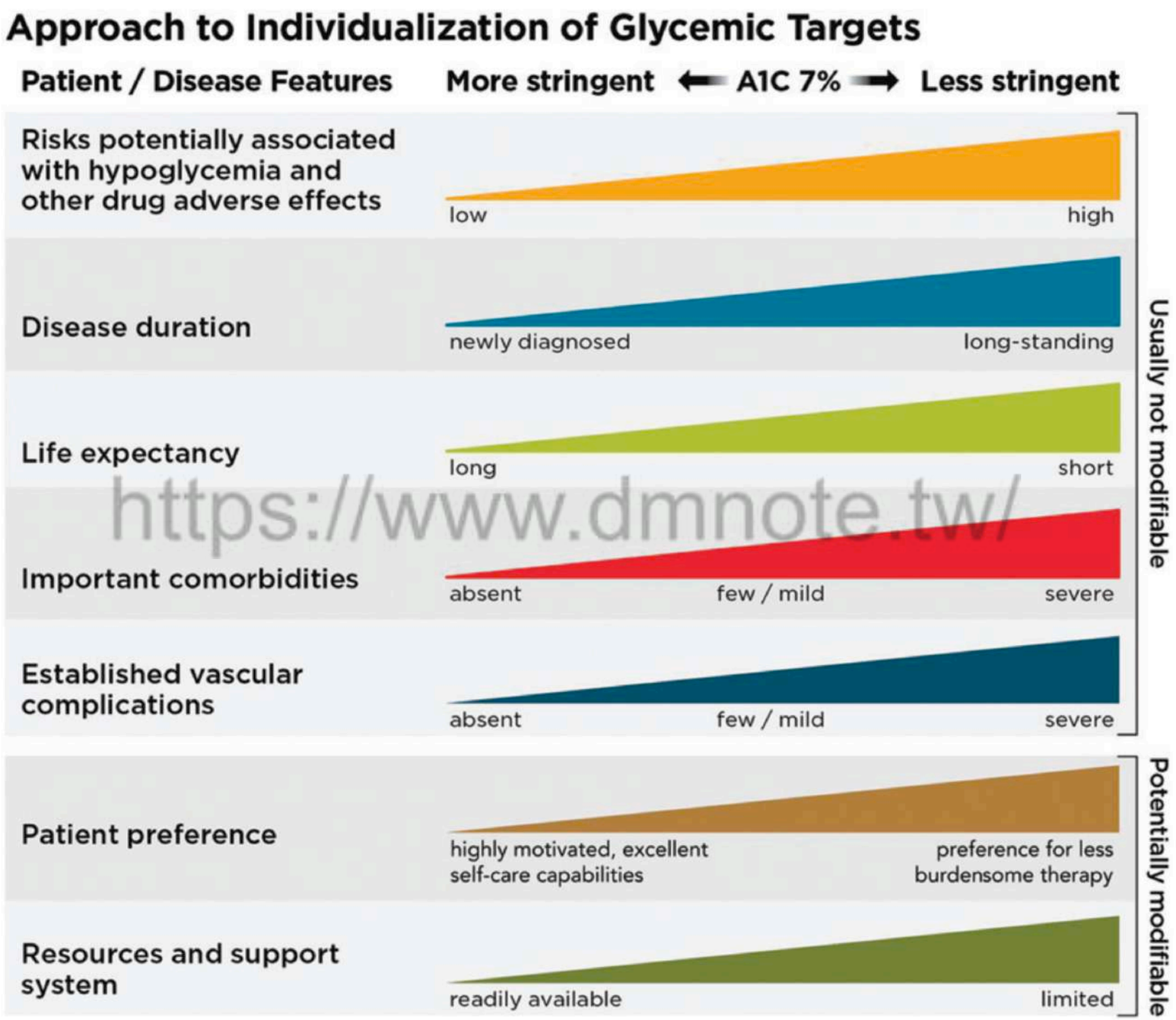


Figure 6.2—Patient and disease factors used to determine optimal glycemic targets. Characteristics and predicaments toward the left justify more stringent efforts to lower A1C; those toward the right suggest less stringent efforts. A1C 7% = 53 mmol/mol. Adapted with permission from Inzucchi et al. (68).

2022 ADA 美國糖尿病治療指引

Table 6.3—Summary of glycemic recommendations for many nonpregnant adults with diabetes

A1C	<7.0% (53 mmol/mol)*#
Preprandial capillary plasma glucose	80–130 mg/dL* (4.4–7.2 mmol/L)
Peak postprandial capillary plasma glucose†	<180 mg/dL* (10.0 mmol/L)

*More or less stringent glycemic goals may be appropriate for individual patients. #CGM may be used to assess glycemic target as noted in Recommendation 6.5b and **Fig. 6.1**. Goals should be individualized based on duration of diabetes, age/life expectancy, comorbid conditions, known CVD or advanced microvascular complications, hypoglycemia unawareness, and individual patient considerations (as per **Fig.6.2**). †Postprandial glucose may be targeted if A1C goals are not met despite reaching preprandial glucose goals. Postprandial glucose measurements should be made 1–2 h after the beginning of the meal, generally peak levels in patients with diabetes.

2022 ADA 美國糖尿病治療指引

Table 6.4—Classification of hypoglycemia

	Glycemic criteria/description
Level 1	Glucose <70 mg/dL (3.9 mmol/L) and ≥54 mg/dL (3.0 mmol/L)
Level 2	Glucose <54 mg/dL (3.0 mmol/L)
Level 3	A severe event characterized by altered mental and/or physical status requiring assistance for treatment of hypoglycemia

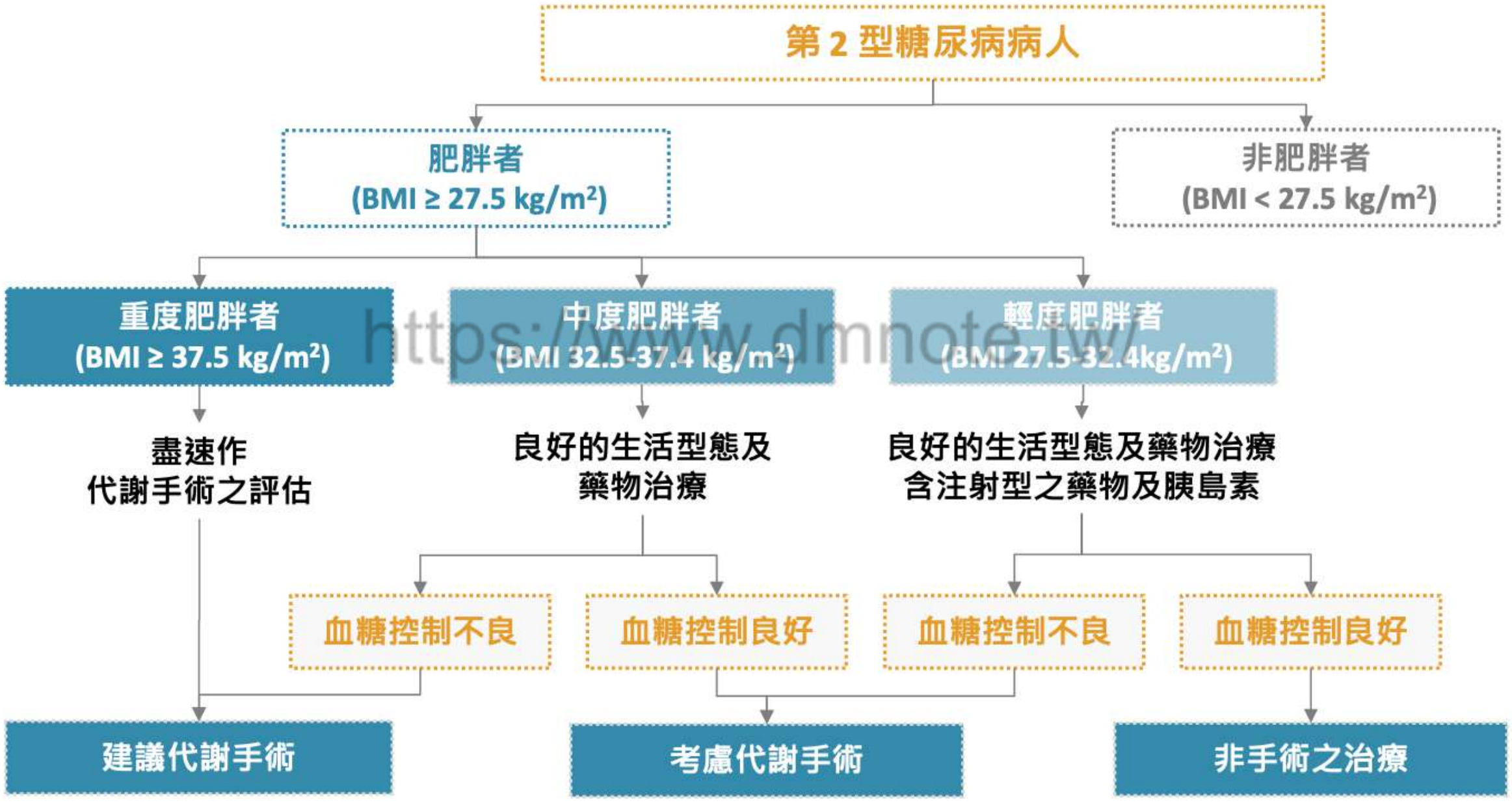
Reprinted from Agiostratidou et al. (72).

糖尿病人臨床監測建議表

測試項目及建議測試頻率	
糖化血色素及靜脈血漿糖[註1]	3個月
糖尿病衛教	3個月
血脂肪：低密度、高密度與總膽固醇/三酸甘油酯 *(血脂異常需追蹤者)*	1年 *(3-6 個月)*
腎臟：肌酸酐/eGFR/尿液常規/白蛋白尿[註2] *(上述檢查異常需追蹤者)*	1年 *(3-6 個月)*
眼睛：視力、眼底檢查[註3]	1年

測試項目及建議測試頻率	
足部：脈博、踝臂動脈收縮壓比值[註4]	1年
神經病變：單股纖維壓覺、頻率128Hz音叉震動感、肌腱反射	1年
口腔檢查	1年
癌症篩檢	配合國健署癌篩政策
糖尿病人自我管理：體重、血壓、血糖、足部	經常
焦慮與憂鬱之評估	高風險病患或有臨床症狀時

糖尿病代謝手術的適應症



第二次糖尿病手術高峰會對第2型糖尿病代謝手術的治療建議

血糖控制

藥物治療

第 2 型糖尿病人高血糖的處理流程圖
(2020年修訂版)

健康生活型態的飲食和運動及醫病共享決策

糖化血色素<7.5%

建議使用一種抗糖尿病藥

初診斷建議首選：Metformin
- 效果：佳
- 低血糖：低
- 體重：稍下降
- 副作用：腸胃道/乳酸血症

合併高血糖症狀

建議先以 Insulin 治療

糖化血色素 ≥ 7.5%或高於個別化目標值1.5%以上

建議使用兩種抗糖尿病藥

建議 Metformin +
以下不同機轉的抗糖尿病

合併高血糖症狀

建議先以 Insulin 治療

若單一治療未達控制目標時，宜評估心腎風險及共病，加上以下不同機轉的抗糖尿病藥

SGLT2i	GLP1-RA	TZD	DPP4i	AGI	SU/Glinide	Insulin
心血管實證：有(建議使用) 心衰竭實證：強(建議使用) 腎病變實證：強(建議使用) 控制血糖效果：中等 體重：下降 低血糖：低 副作用：糖尿病酮酸中毒、生殖泌尿道感染、骨折、截肢、脫水	心血管實證：部分有(建議使用) 心衰竭實證：中立 腎病變實證：有(蛋白尿) 控制血糖效果：佳 體重：下降 低血糖：低 副作用：腸胃道	心血管實證：有 心衰竭實證：不建議使用 腎病變實證：缺 控制血糖效果：佳 體重：增加 低血糖：低 副作用：水腫、心衰竭、骨折	心血管實證：中立 心衰竭實證：部分中立 腎病變實證：有(蛋白尿) 控制血糖效果：中等 體重：無影響 低血糖：低 副作用：少見	心血管實證：中立 心衰竭實證：缺 腎病變實證：缺 控制血糖效果：中等 體重：稍下降 低血糖：低 副作用：腸胃道	心血管實證：缺 心衰竭實證：缺 腎病變實證：缺 控制血糖效果：佳 體重：增加 低血糖：中 副作用：低血糖	心血管實證：中立 心衰竭實證：中立 腎病變實證：中立 控制血糖效果：最佳 體重：增加 低血糖：高 副作用：低血糖

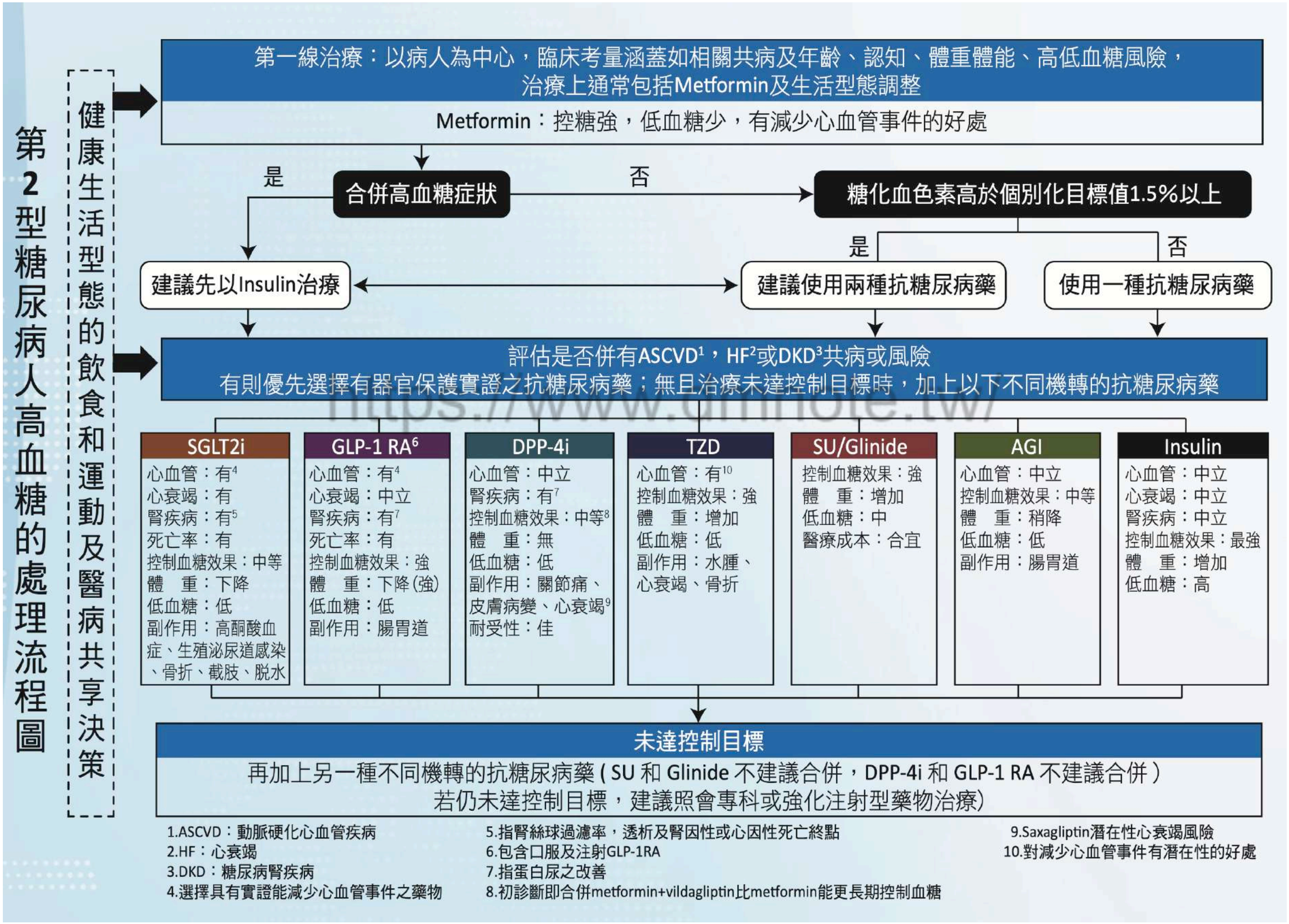
未達控制目標

再加上另一種不同機轉的抗糖尿病藥 (SU 和 Glinide 不建議合併，DPP4i 和 GLP1-RA 不建議合併)

未達控制目標

建議照會專科或強化注射型藥物治療

2022糖尿病學會指引:第 2 型糖尿病人高血糖的處理流程



2022糖尿病學會指引:

第2型糖尿病人注射型藥物的治療流程圖

若糖化血色素未達目標值，建議強化注射型藥物

動脈硬化心血管疾病(ASCVD[1])

GLP-1 RA[2]

高血糖且無動脈硬化心血管疾病(Hyperglycemia without ASCVD)

基礎胰島素（可由0.1-0.2U/kg/day開始） 或 GLP-1 RA 或 GLP-1 RA +基礎胰島素 FRC[3] 速效胰島素+基礎胰島素 FRC[4]

嚴重高血糖(HbA1c > 10%)

胰島素強化治療

若糖化血色素未達目標值，胰島素強化治療

基礎胰島素 + 一次速效胰島素

可考慮FRC[4]QD

GLP-1 RA + 基礎胰島素

可考慮FRC[3]

基礎胰島素 + 二次速效胰島素

可考慮FRC[4]BID

預混型胰島素

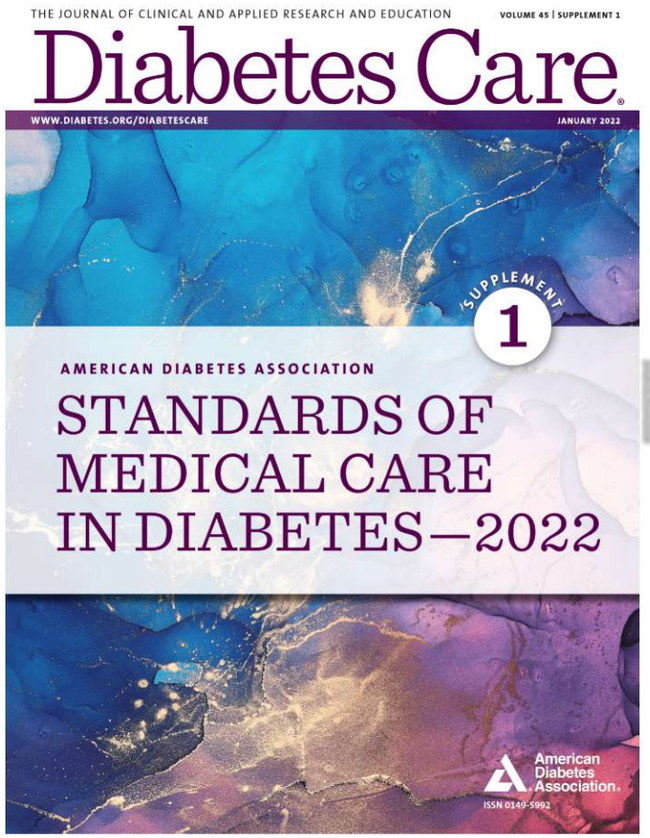
視臨床需求一天一次或二次或三次

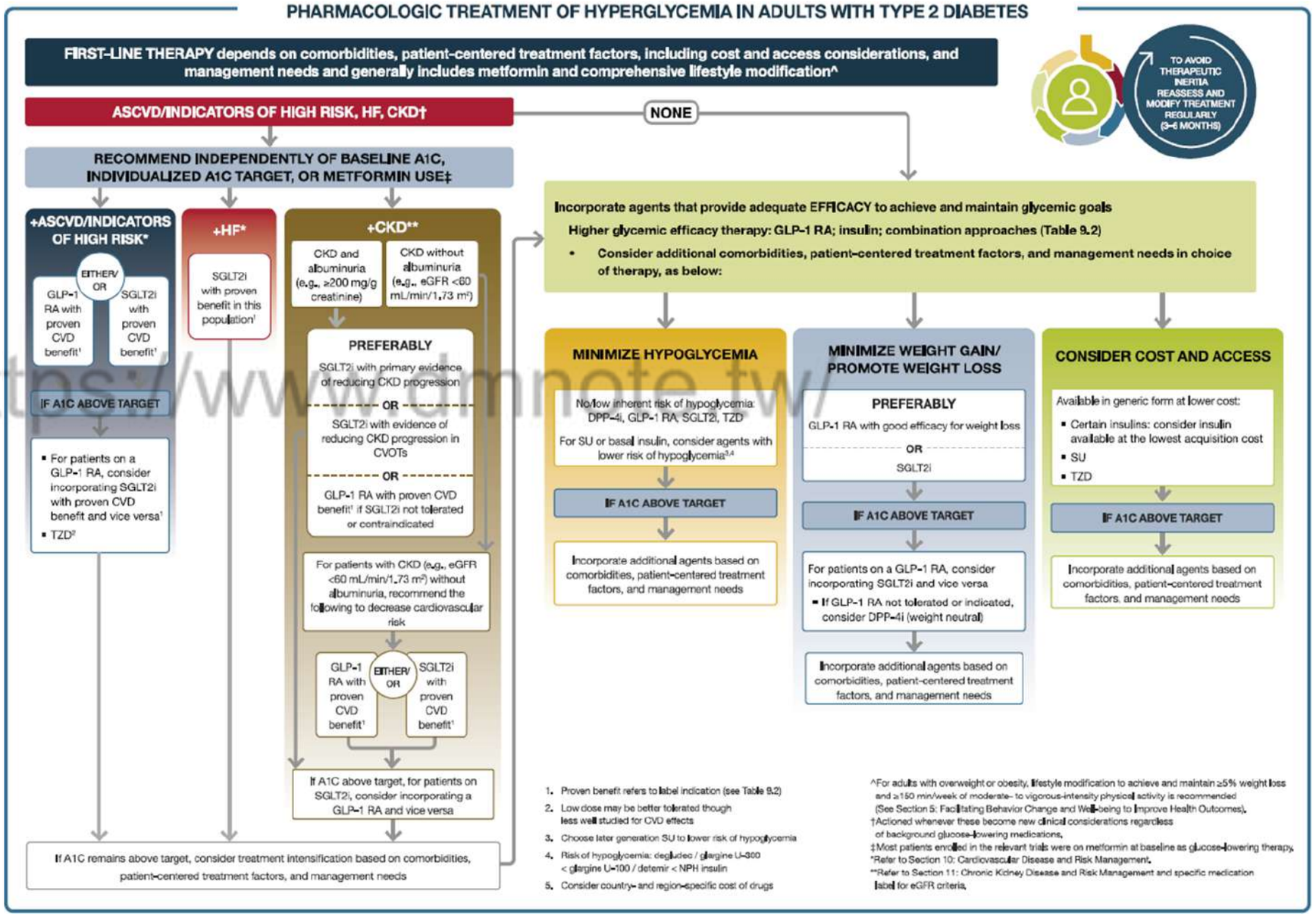
基礎胰島素 + 三次速效胰島素

可考慮FRC[4]BID + 一次速效胰島素

1.ASCVD：動脈硬化心血管疾病
2.選擇具有實證能減少心血管事件之藥物
3.GLP-1 RA + 基礎胰島素FRC：GLP-1 RA + 基礎胰島素之fixed-ratio combination（定例複方）
4.速效胰島素 + 基礎胰島素FRC：速效胰島素 + 基礎胰島素之fixed-ratio combination（定例複方）

2022 ADA guidelines 的治療建議





American Diabetes Association.
Connected for Life EM-TW-101320 _202201

糖尿病併發症

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Long-Term Complications in Youth-Onset Type 2 Diabetes

TODAY Study Group*

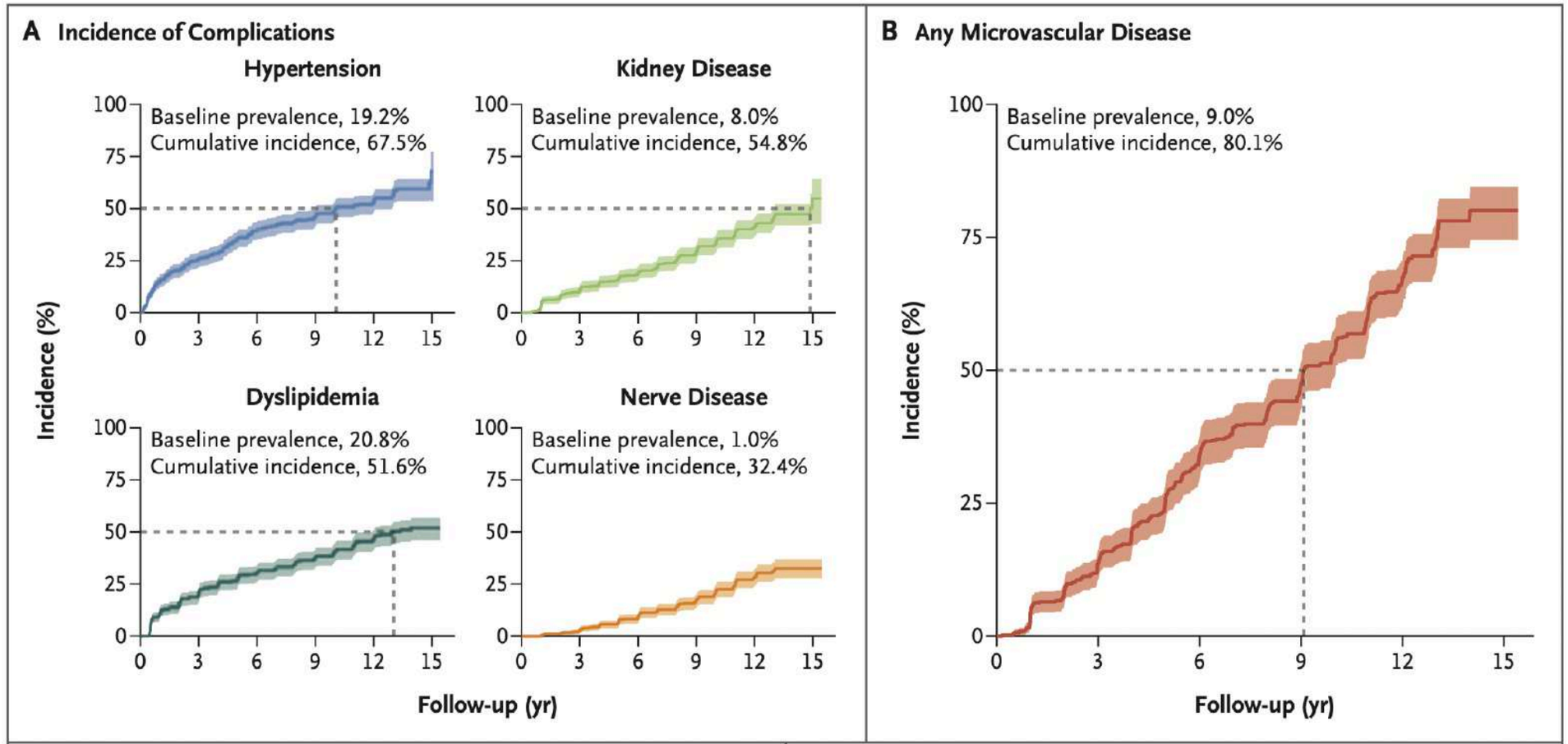


Figure 2. Diabetes-Related Complications That Occurred during the Study.

Panel A shows the baseline prevalences and cumulative incidences of hypertension, dyslipidemia (low-density-lipoprotein or triglyceride dyslipidemia), kidney disease, and nerve disease. Panel B shows the baseline prevalence and cumulative incidence of any microvascular complication. The dashed lines in Panels A and B indicate the time when the cumulative incidence reached 50%, and the shaded bands represent 95% confidence intervals. Baseline refers to the time of enrollment in the clinical trial. Panel C shows the numbers of patients with kidney, nerve, and retinal complications. Panel D shows the years to onset of the first and subsequent (if applicable) microvascular complications among participants with one, two, or three complications. The bottoms and tops of the boxes represent the first and the third quartiles, respectively, and the horizontal lines inside the boxes indicate medians. The symbols inside the boxes indicate means, and the symbols outside the boxes outliers. I bars indicate the minimum and maximum range, excluding outliers.

神經病變

困難傷口

Medicina (Kaunas, Lithuania), 57(10), 1072

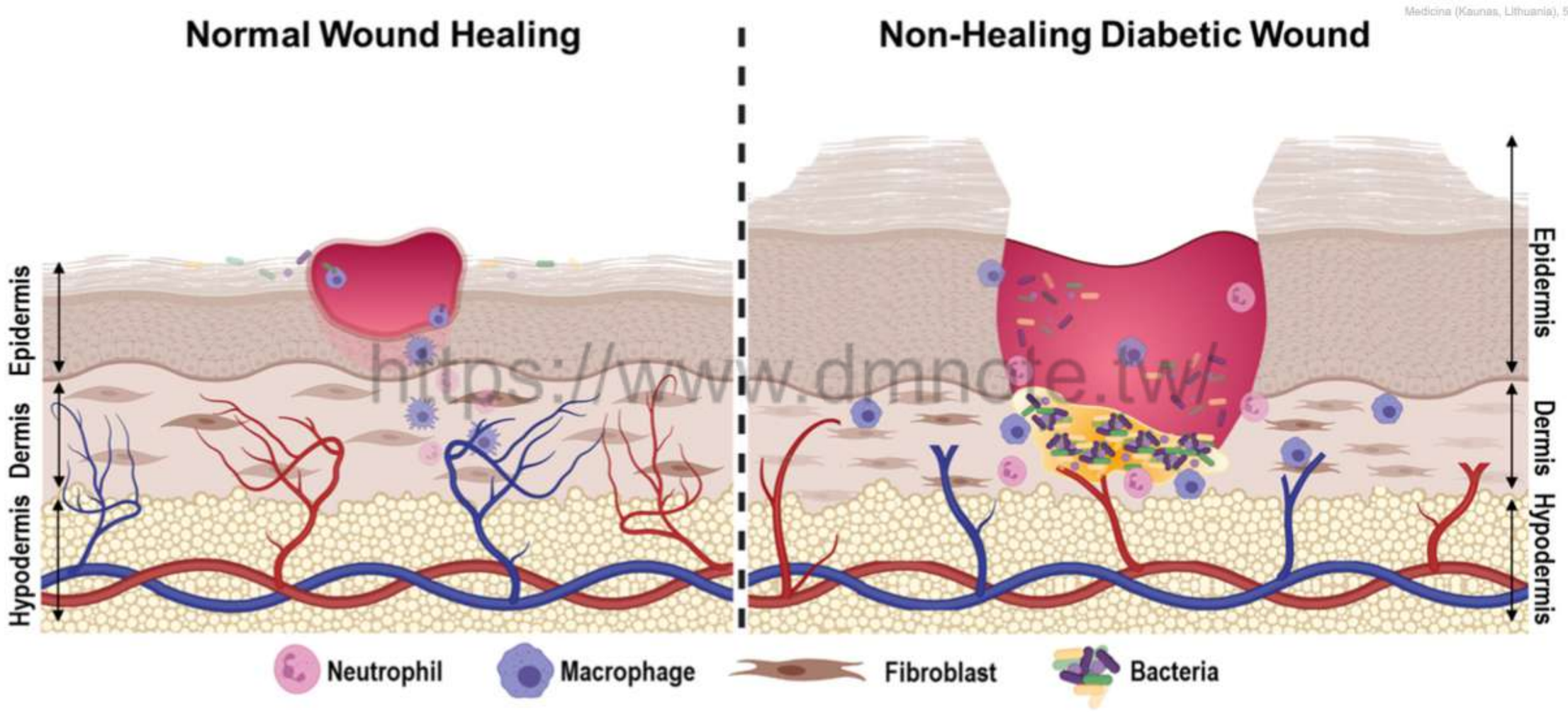


Figure 1. Pathophysiology of diabetic wounds. Diabetic wounds exhibit deregulated angiogenesis, chronically sustained sub-optimal inflammatory response, increased levels of reactive oxygen species, and persistent bacterial colonization that often develops into a hard-to-treat biofilm. Created with BioRender.com, 29 July 2021.

個案分享

傷口於其它醫院或門診照顧...

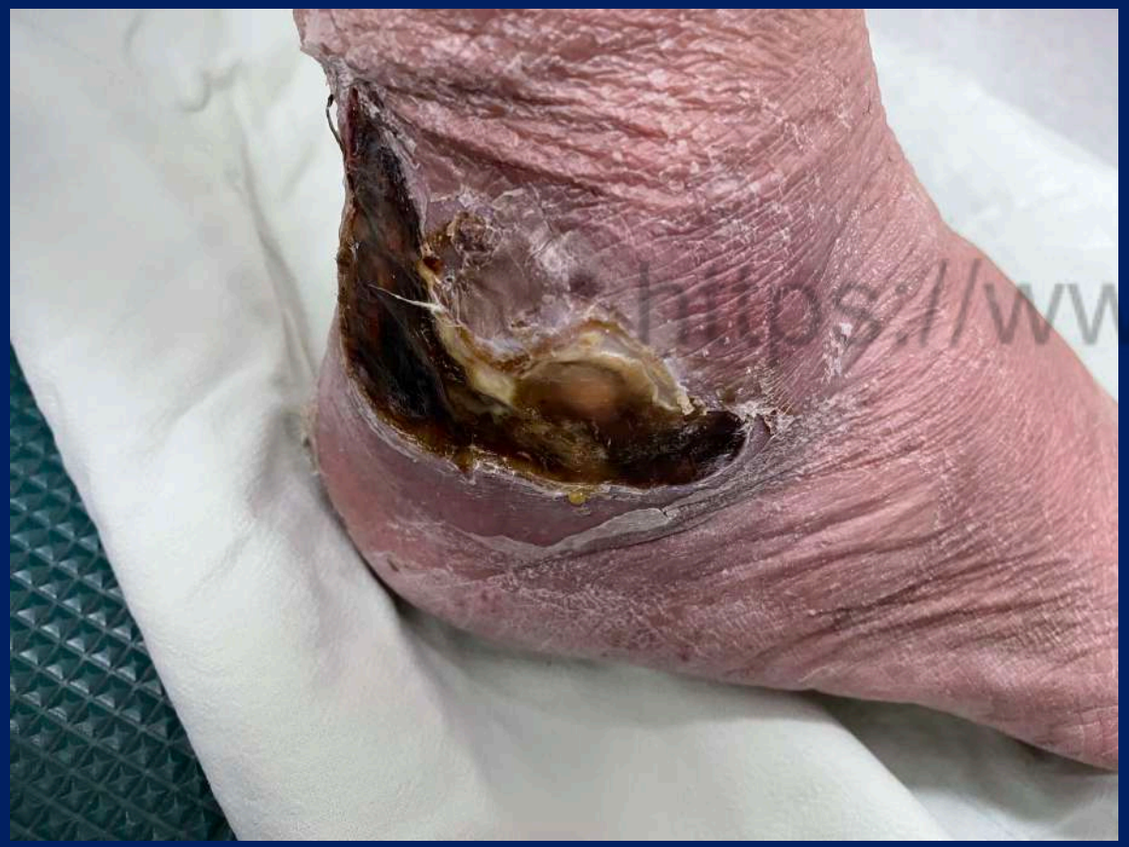
但...超個4個月以上的治療

傷口越來越大而且變焦黑

而且腳痛到睡不覺

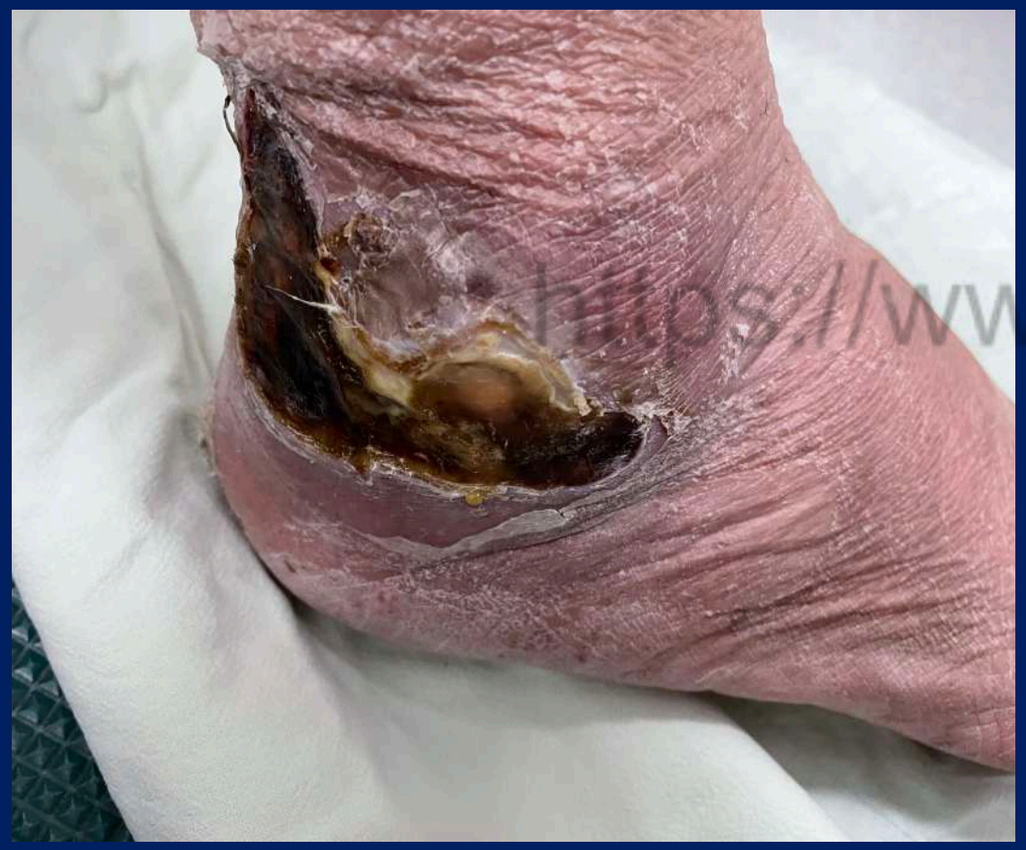
全方位傷口照護

2021-11-03

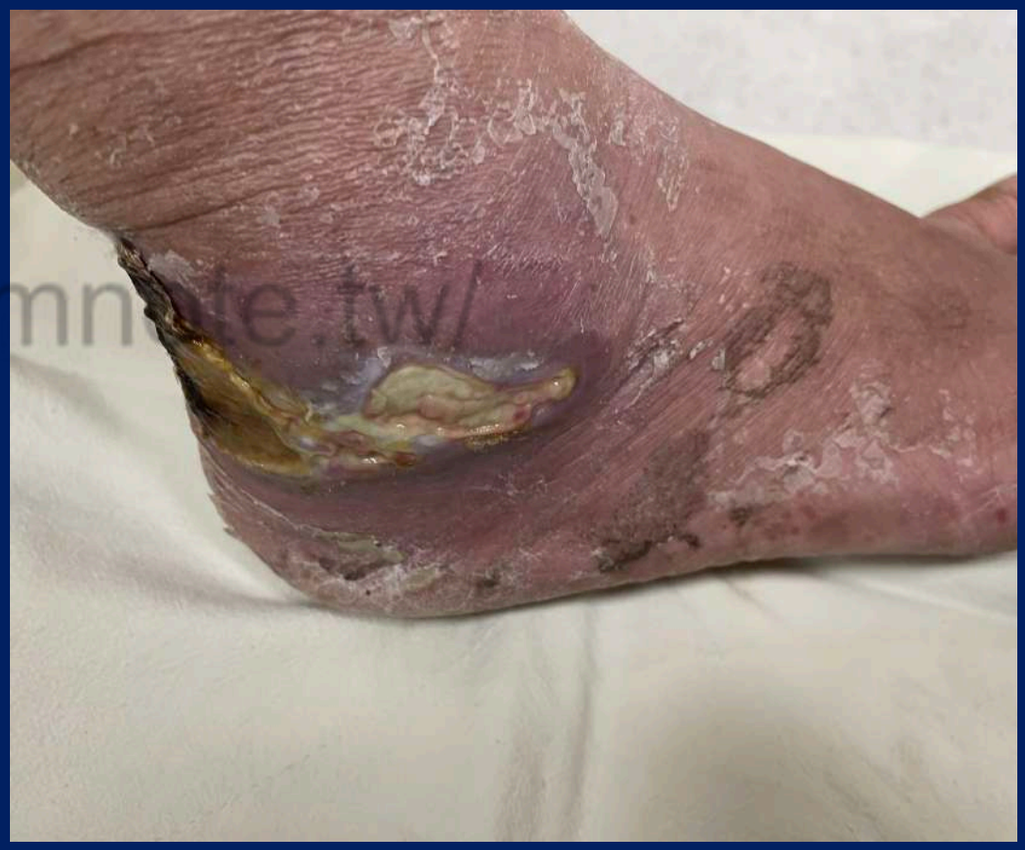


全方位傷口照護

2021-11-03

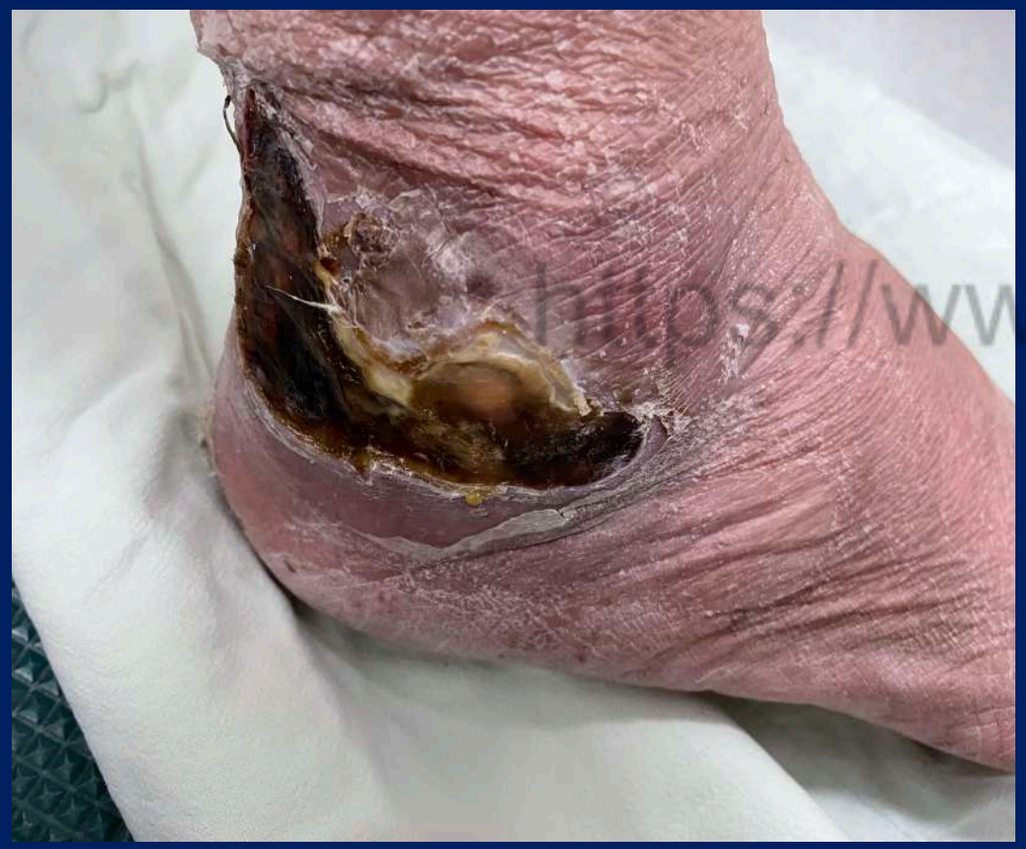


2021-12-08

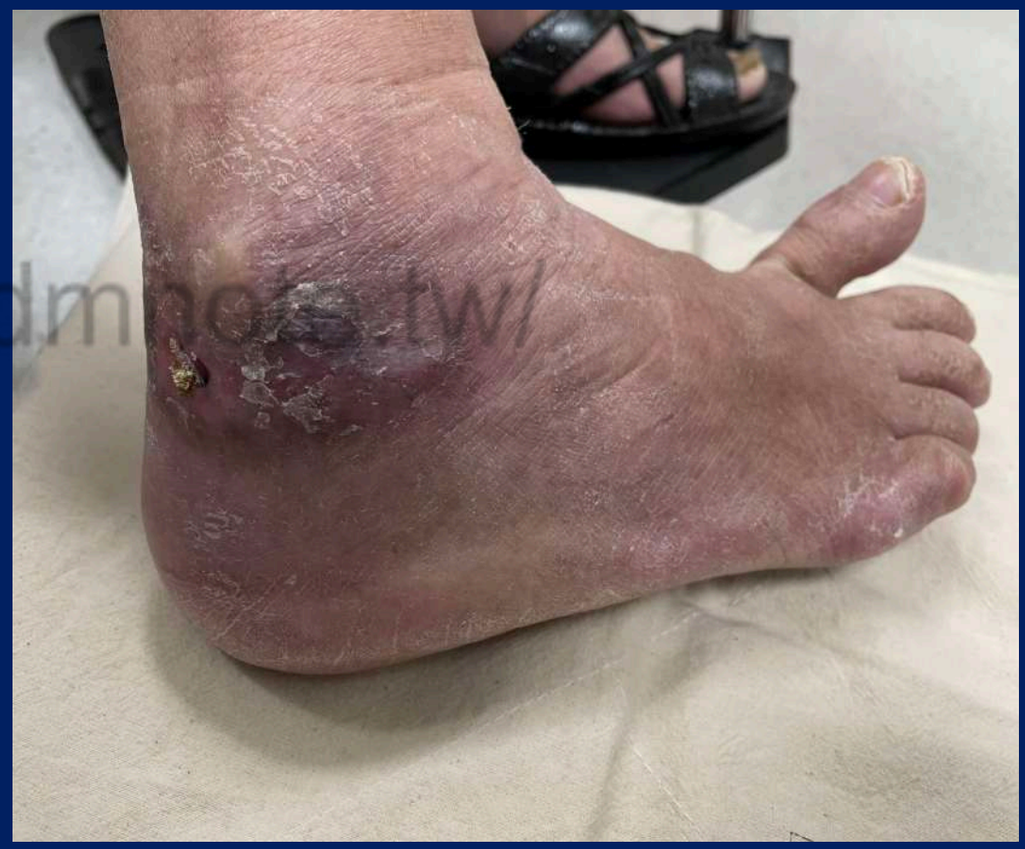


全方位傷口照護

2021-11-03

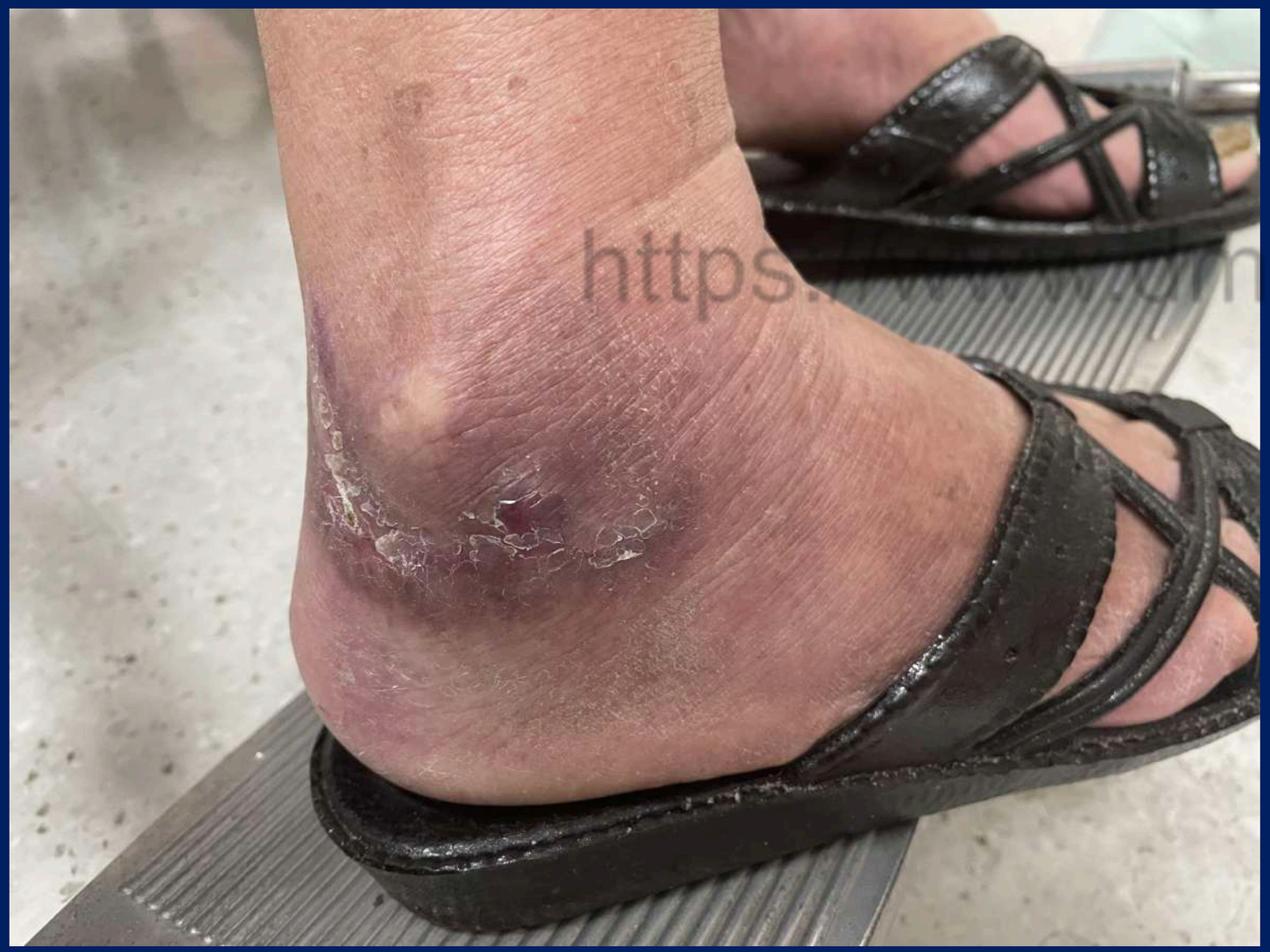


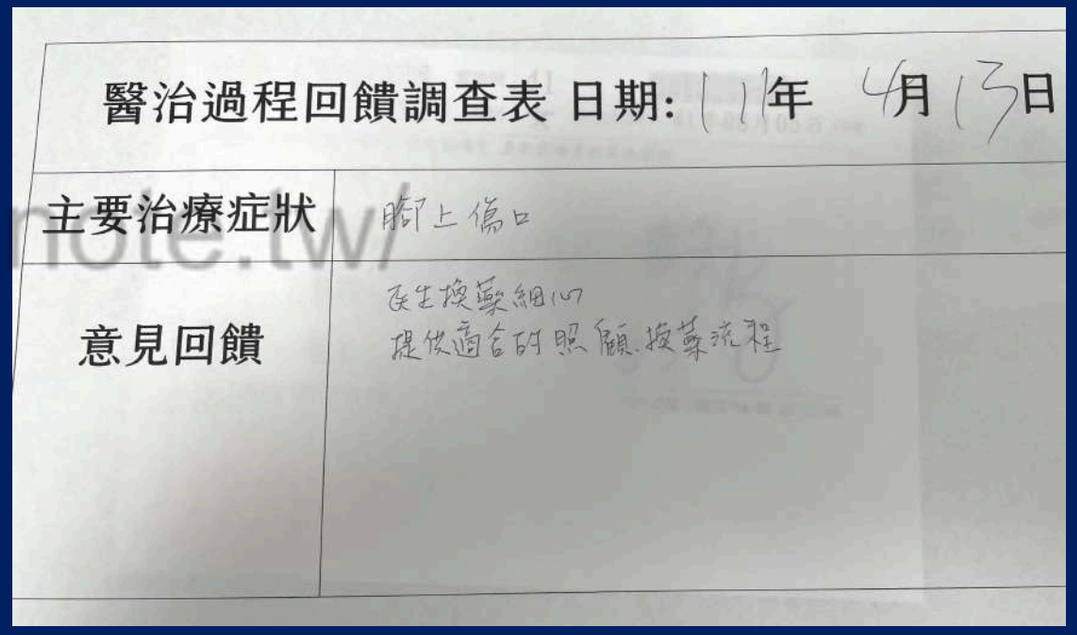
2022-3-16



全方位傷口照護

2022-04-13





醫治過程回饋調查表 日期: 111年 4月 13日

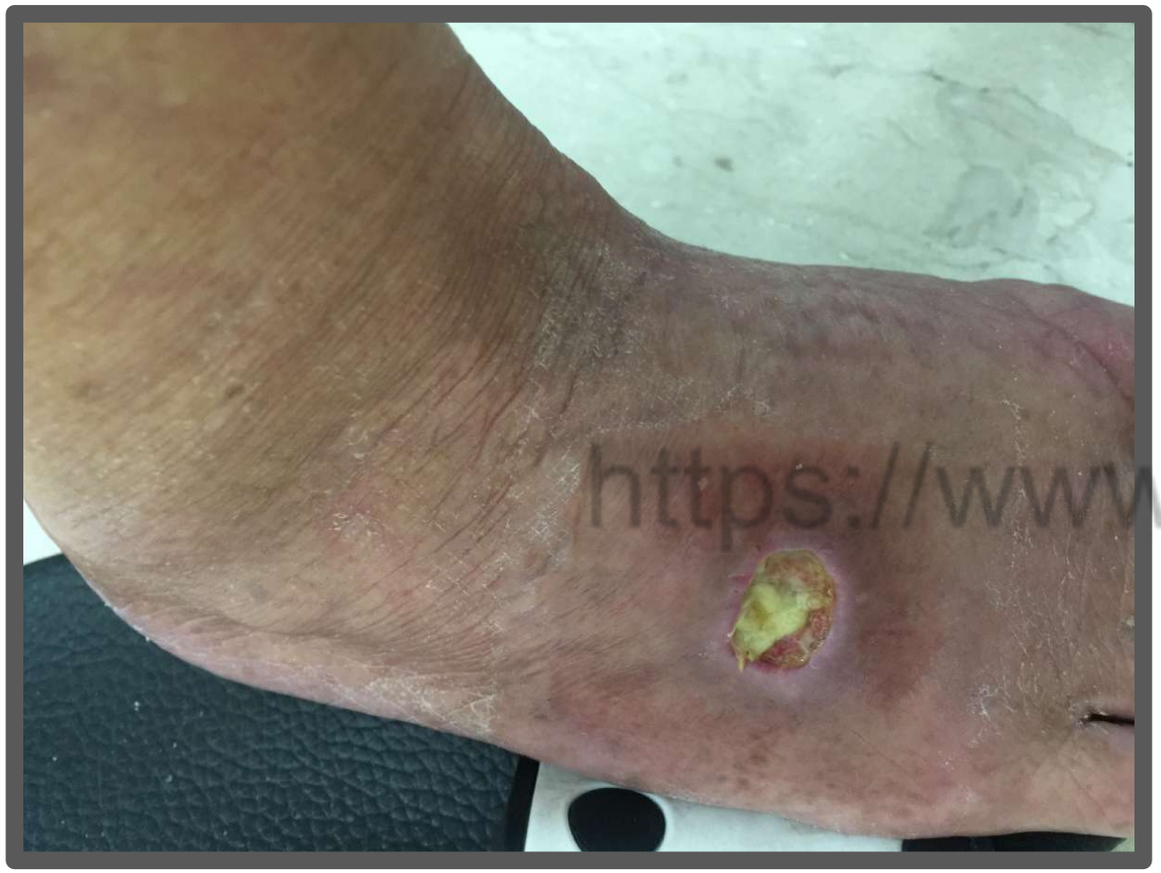
主要治療症狀	腳上傷口
意見回饋	医生換藥細心 提供適合的照顧換藥流程

個案分享

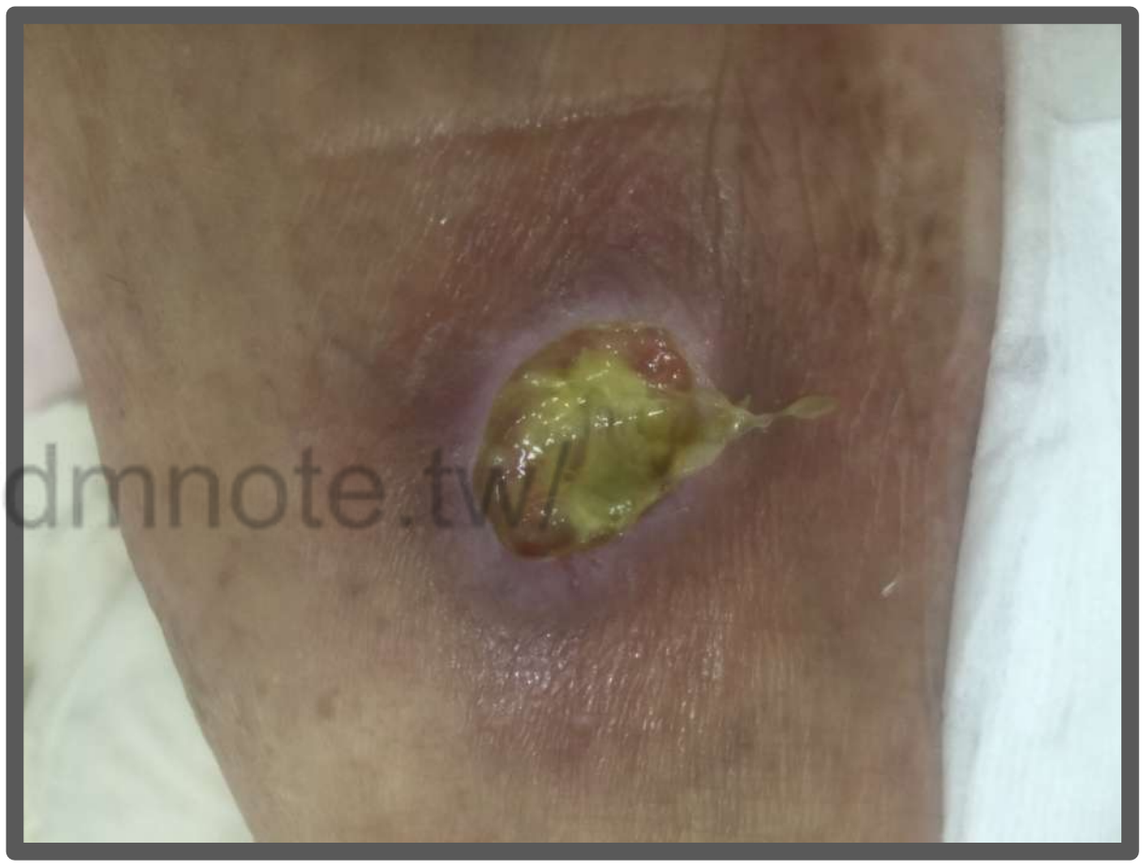
傷口於醫學中心照顧...

但...超過３個月沒癒合

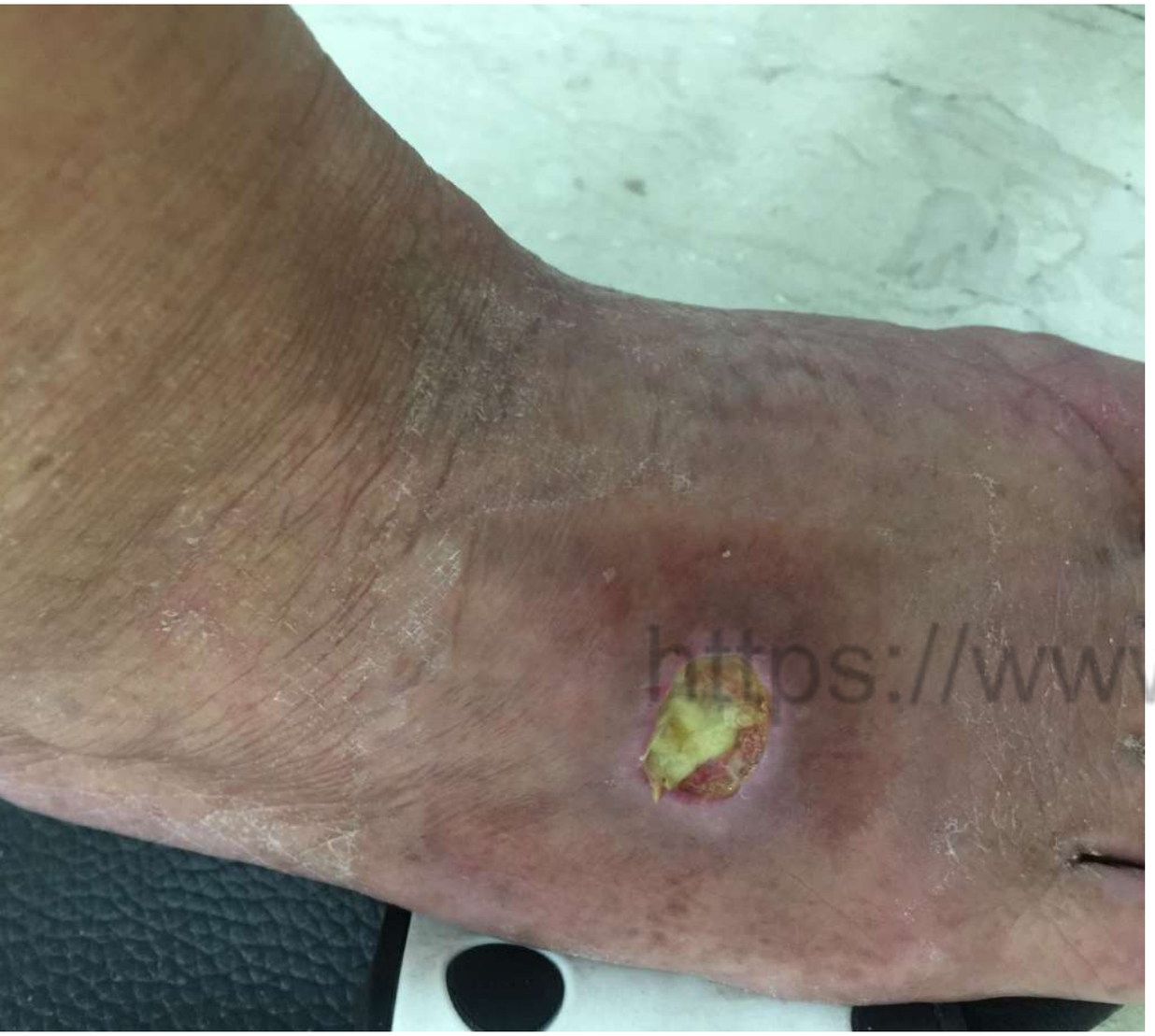
糖尿病傷口照護

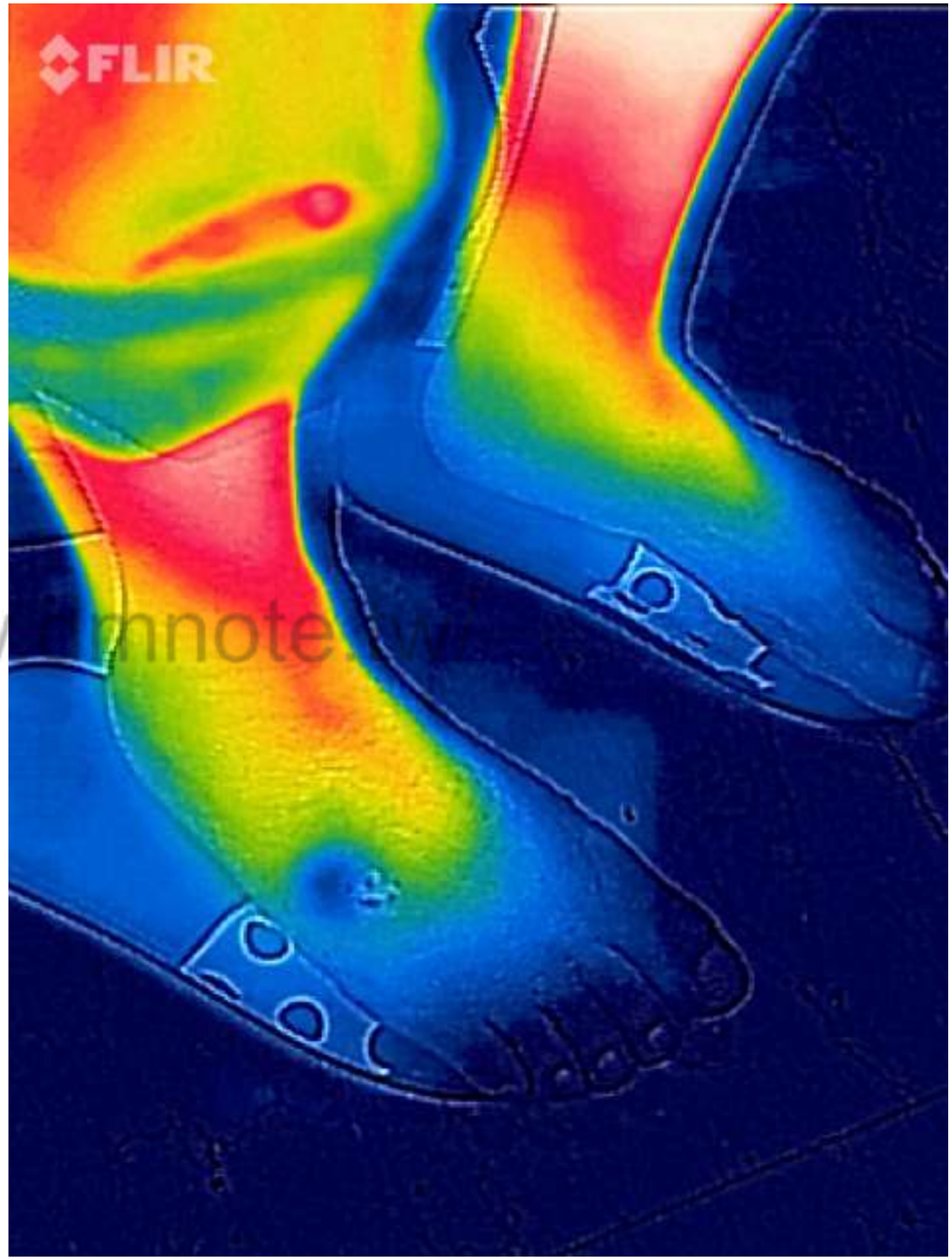


2018/2/13

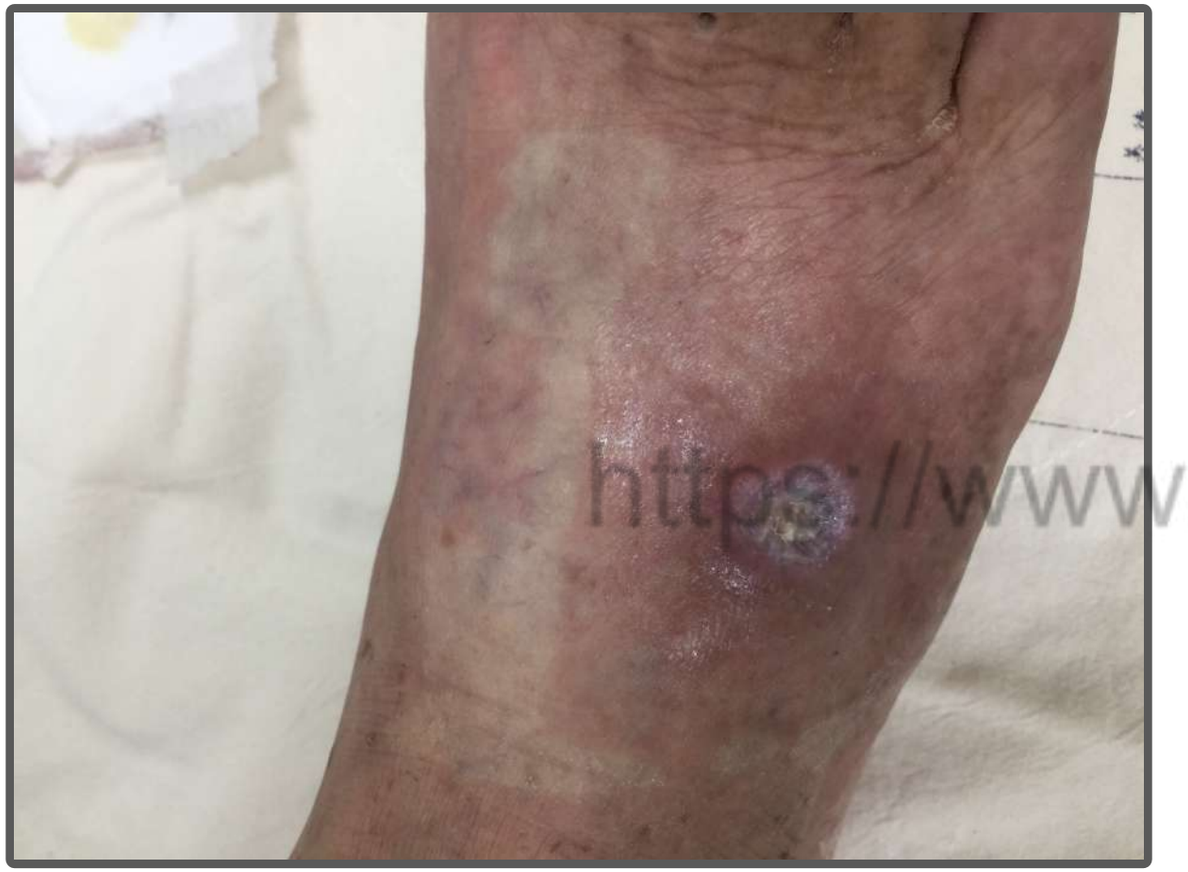


2018/2/20

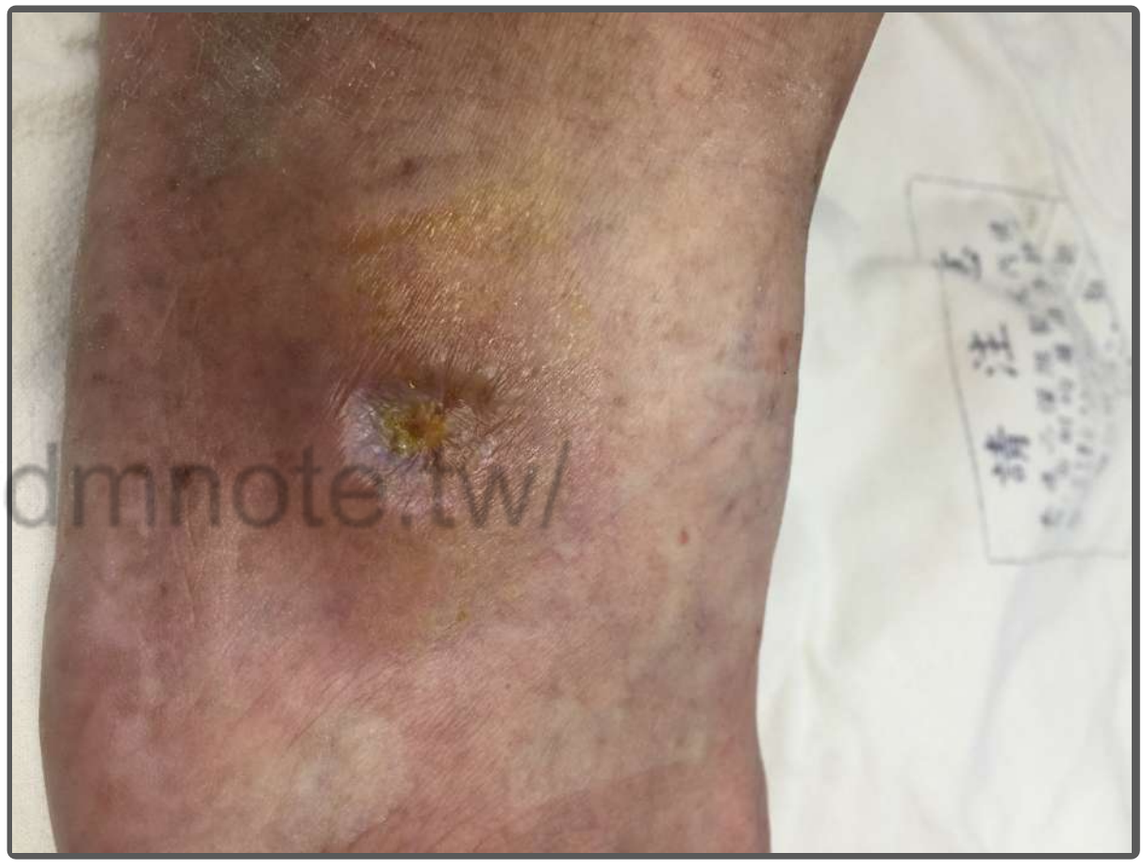




糖尿病傷口照護



2018/4/29



2018/5/08

個案分享

傷口於其它醫院照顧...

但...超過2年沒癒合

病歷號碼：[redacted]

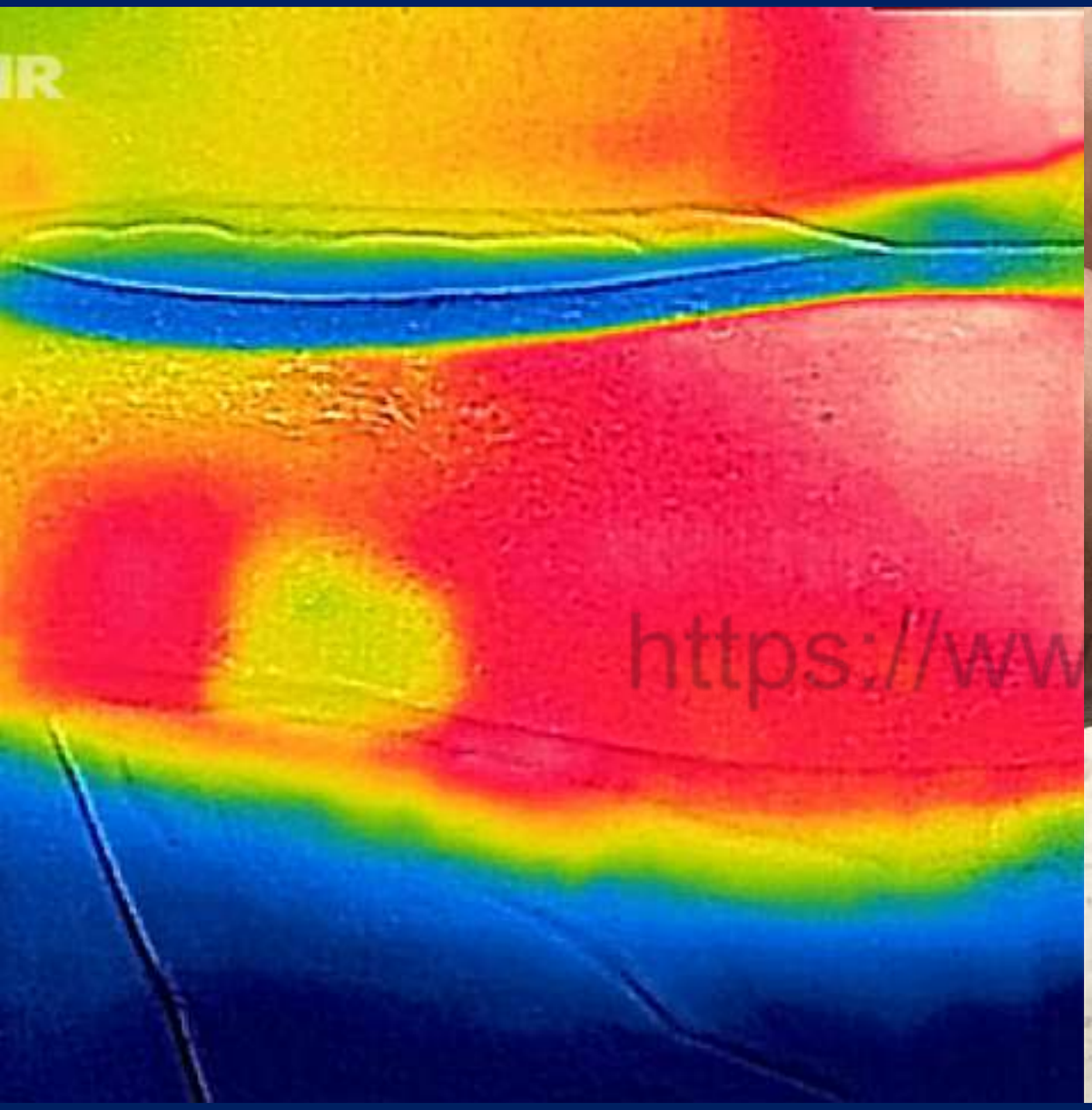
看診號：30

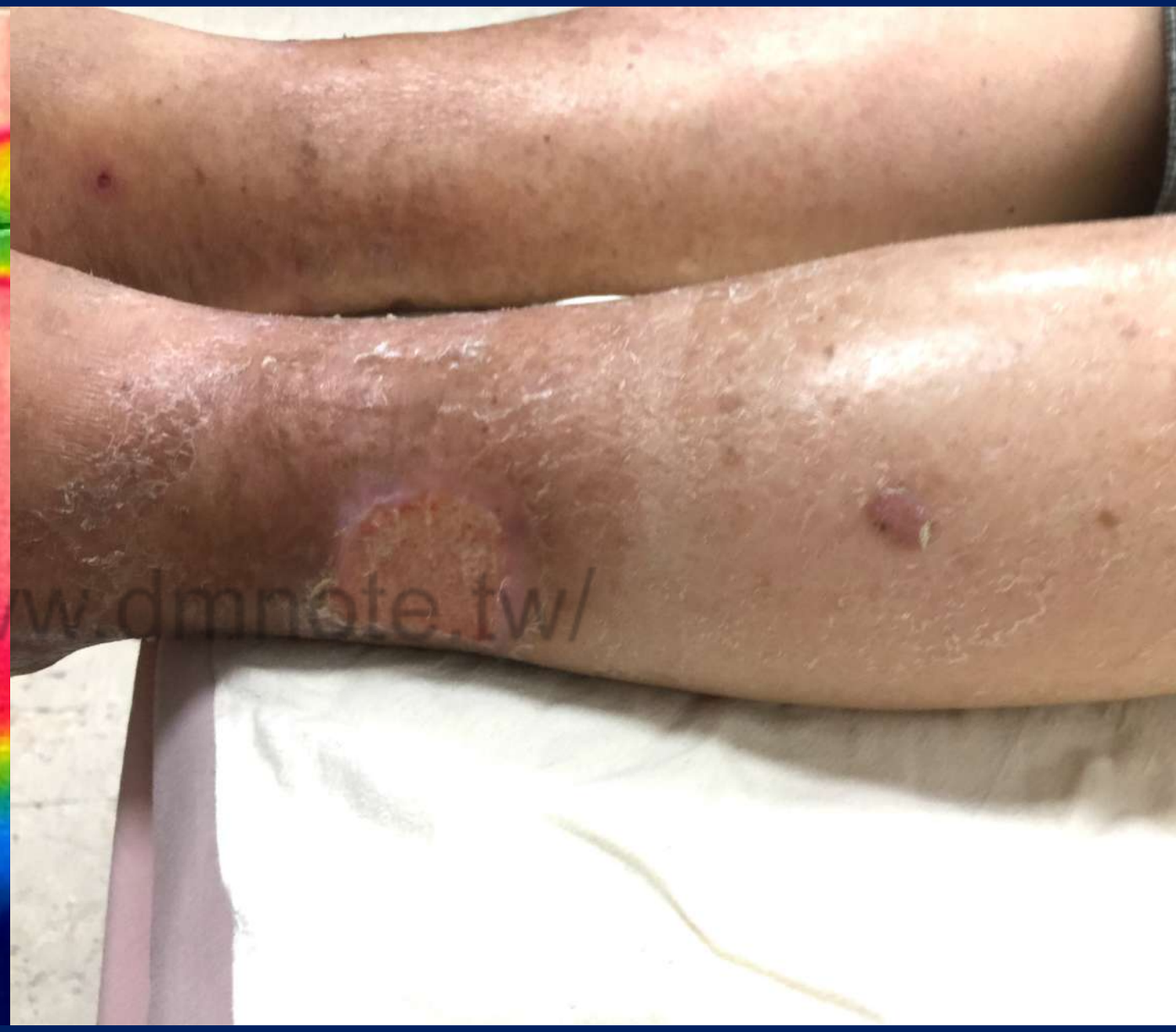
患者姓名：

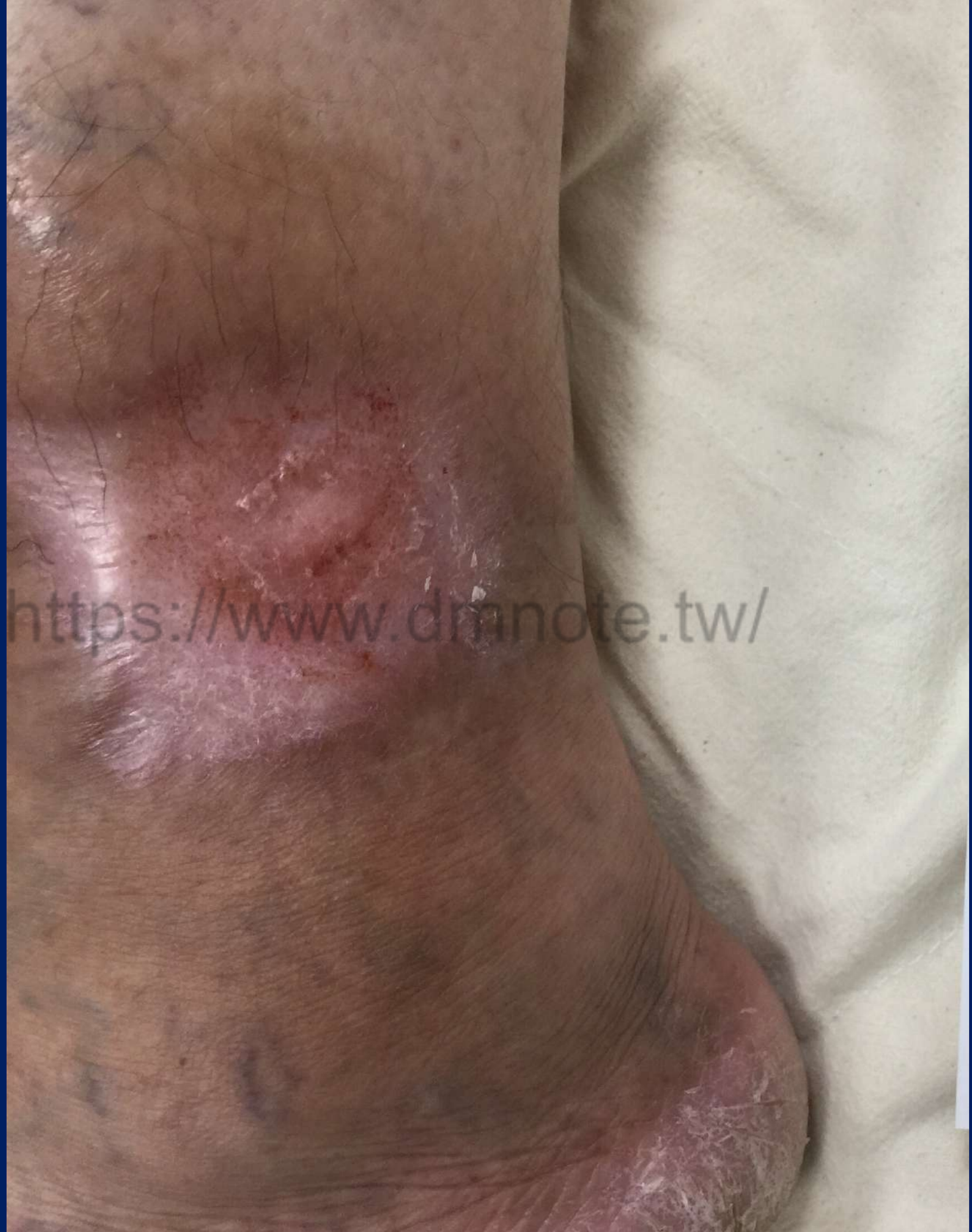
性別：女　出生日期：53年

10時 58分

ㄇㄇkg







個案分享

傷口於其它醫院照顧...

但...醫師說要截肢

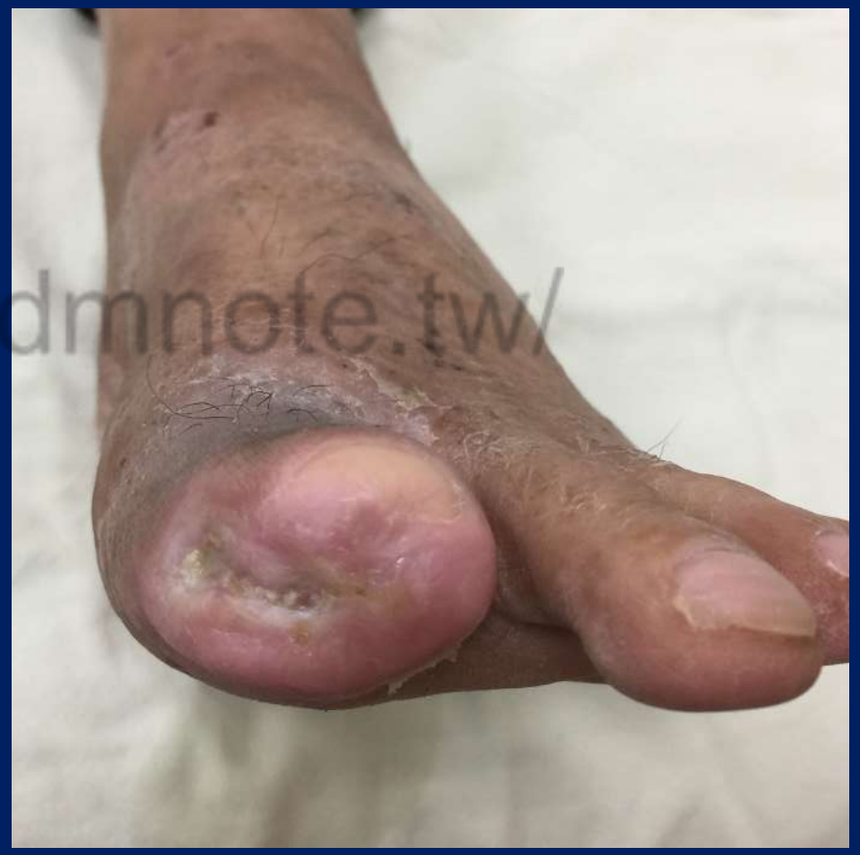
全方位傷口照護

2019-07-15

全方位傷口照護

2019-07-15

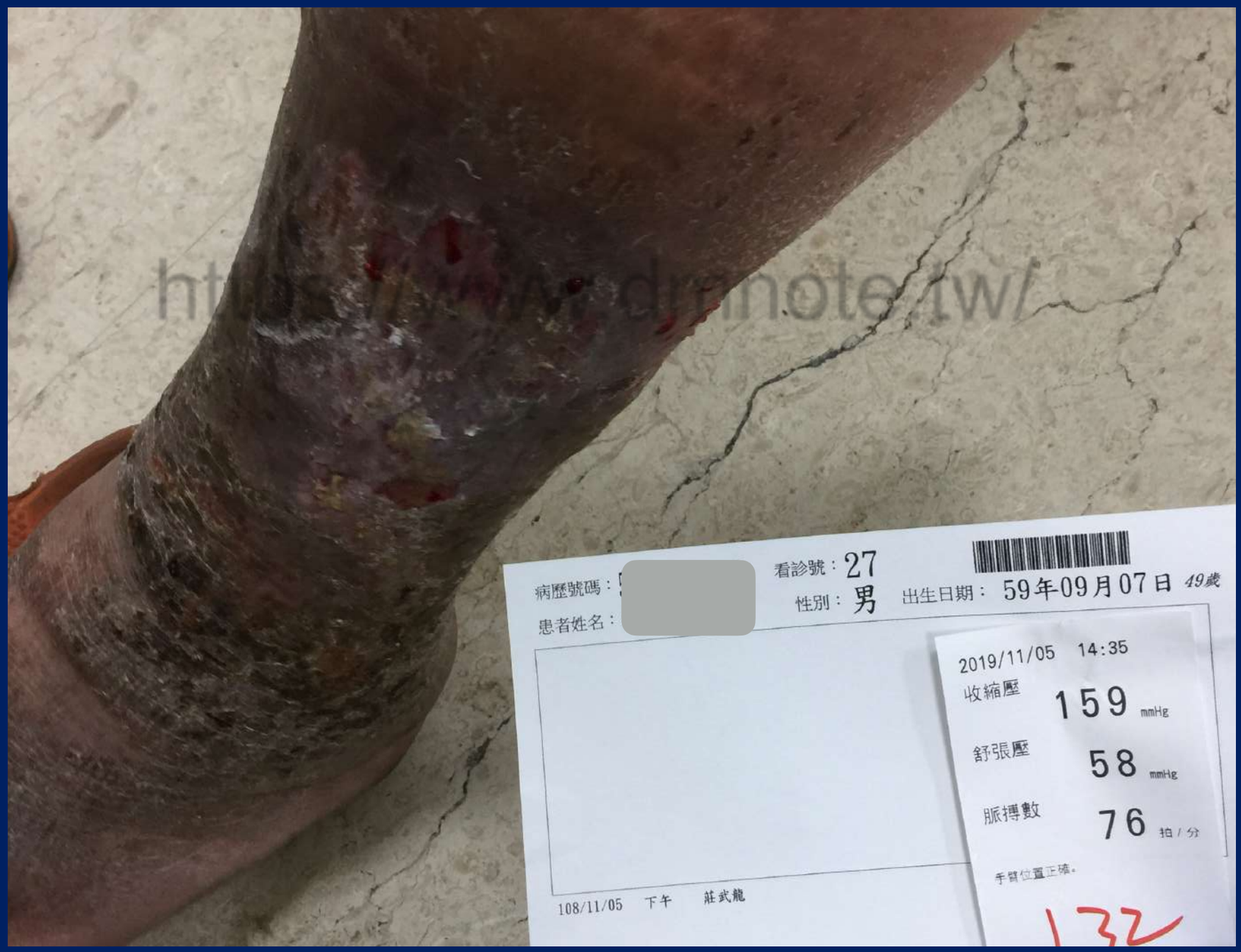
2019-09-26



個案分享
傷口一直沒有癒合...
但...已治療幾個月了

全方位傷口照護

2019-11-05



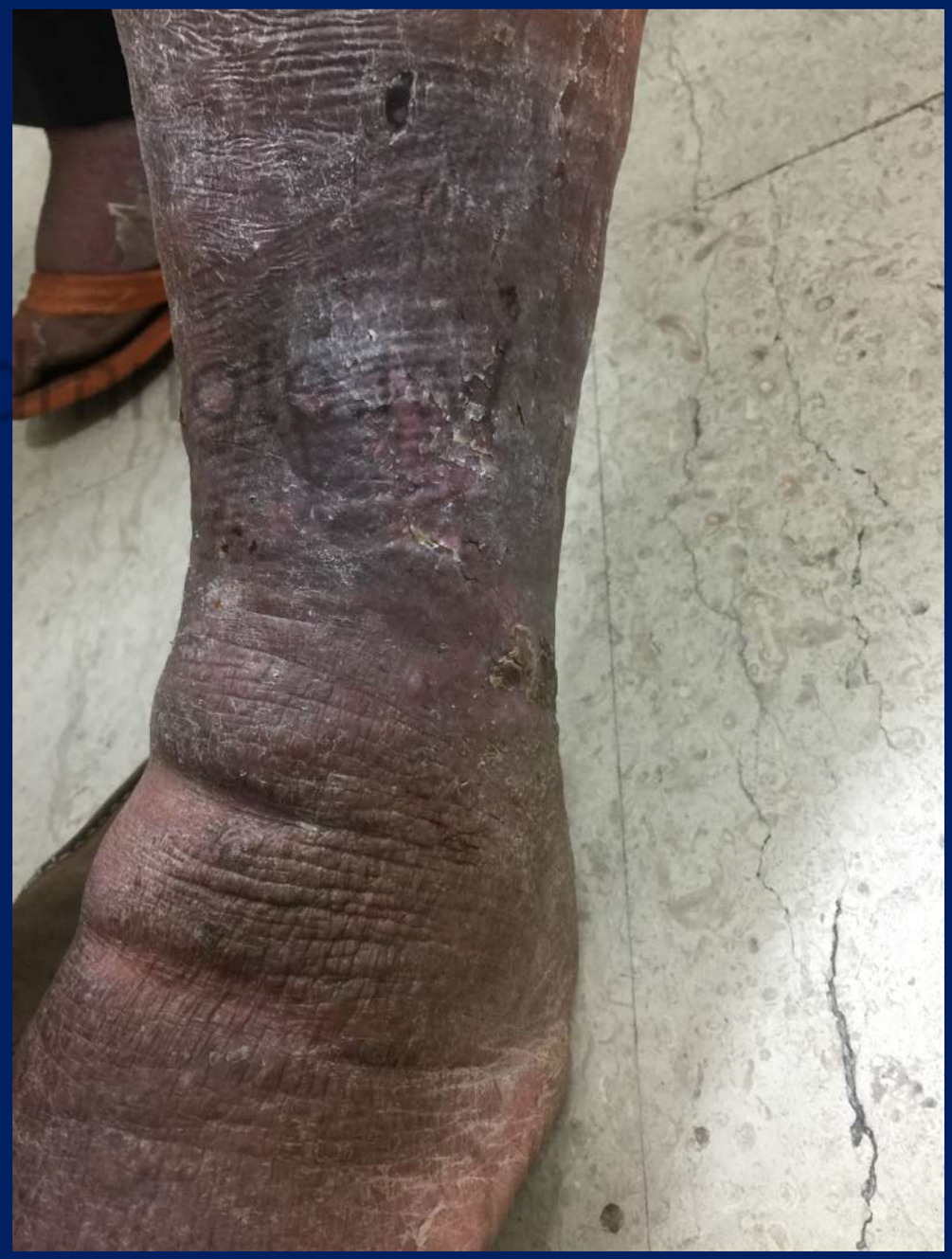
全方位傷口照護

2019-11-19

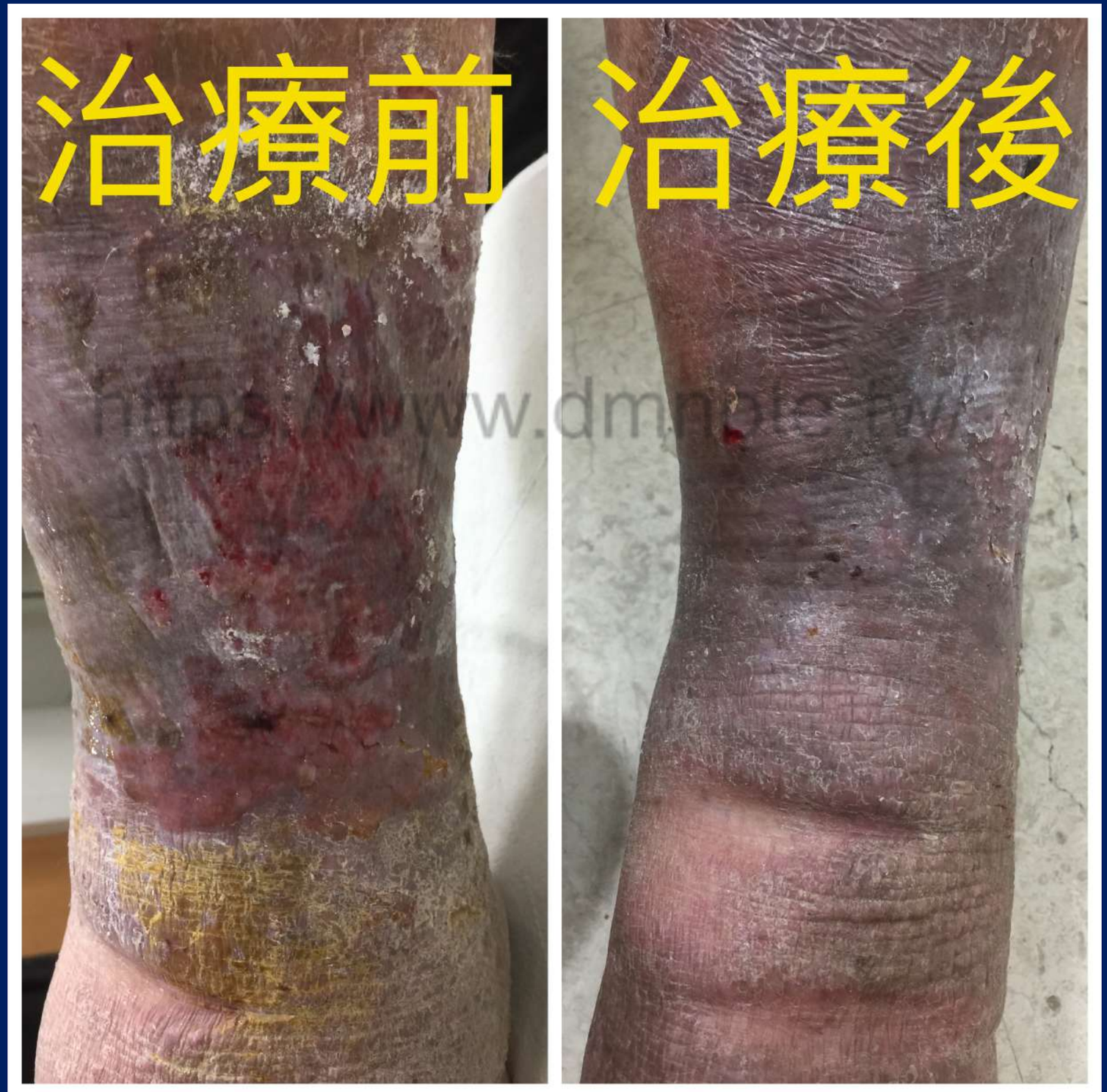
全方位傷口照護

2019-11-19

2019-12-10



全方位傷口照護



糖尿病足傷口與神經病變關係

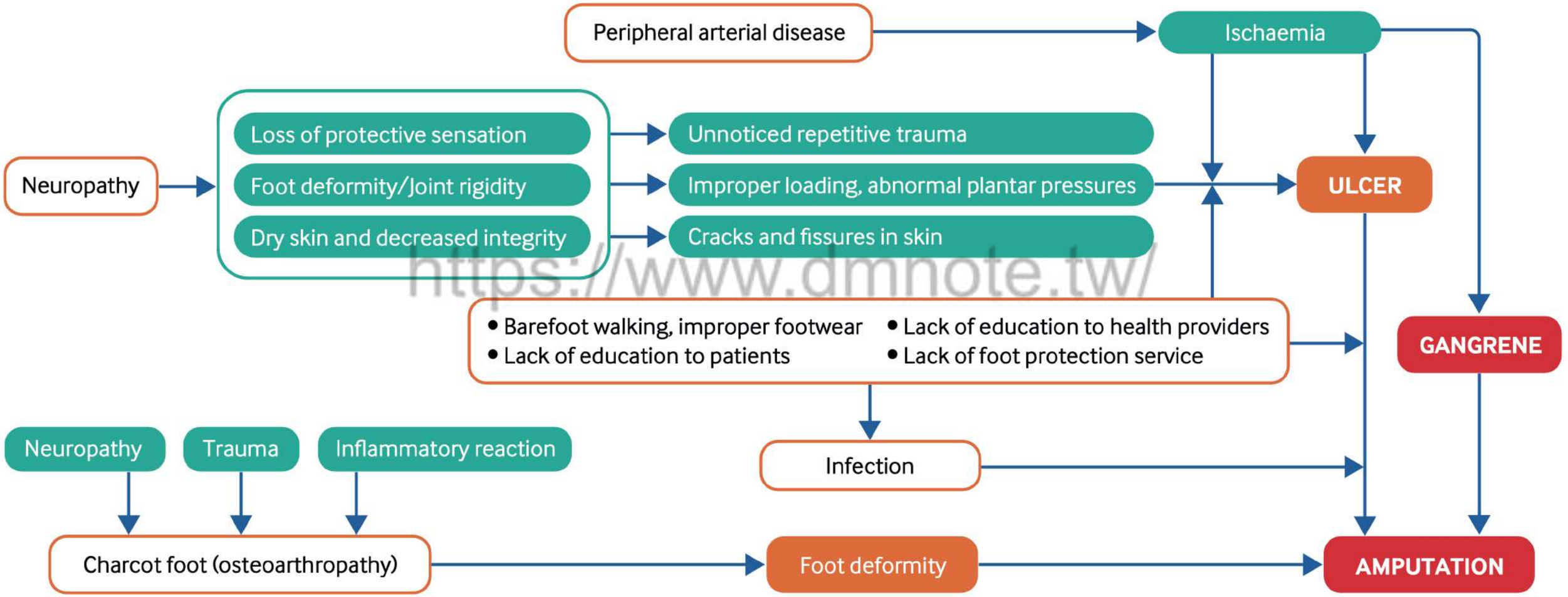


Fig 1 | Risk factors and mechanism for foot ulcer and amputation

糖尿病足傷口與神經病變照護

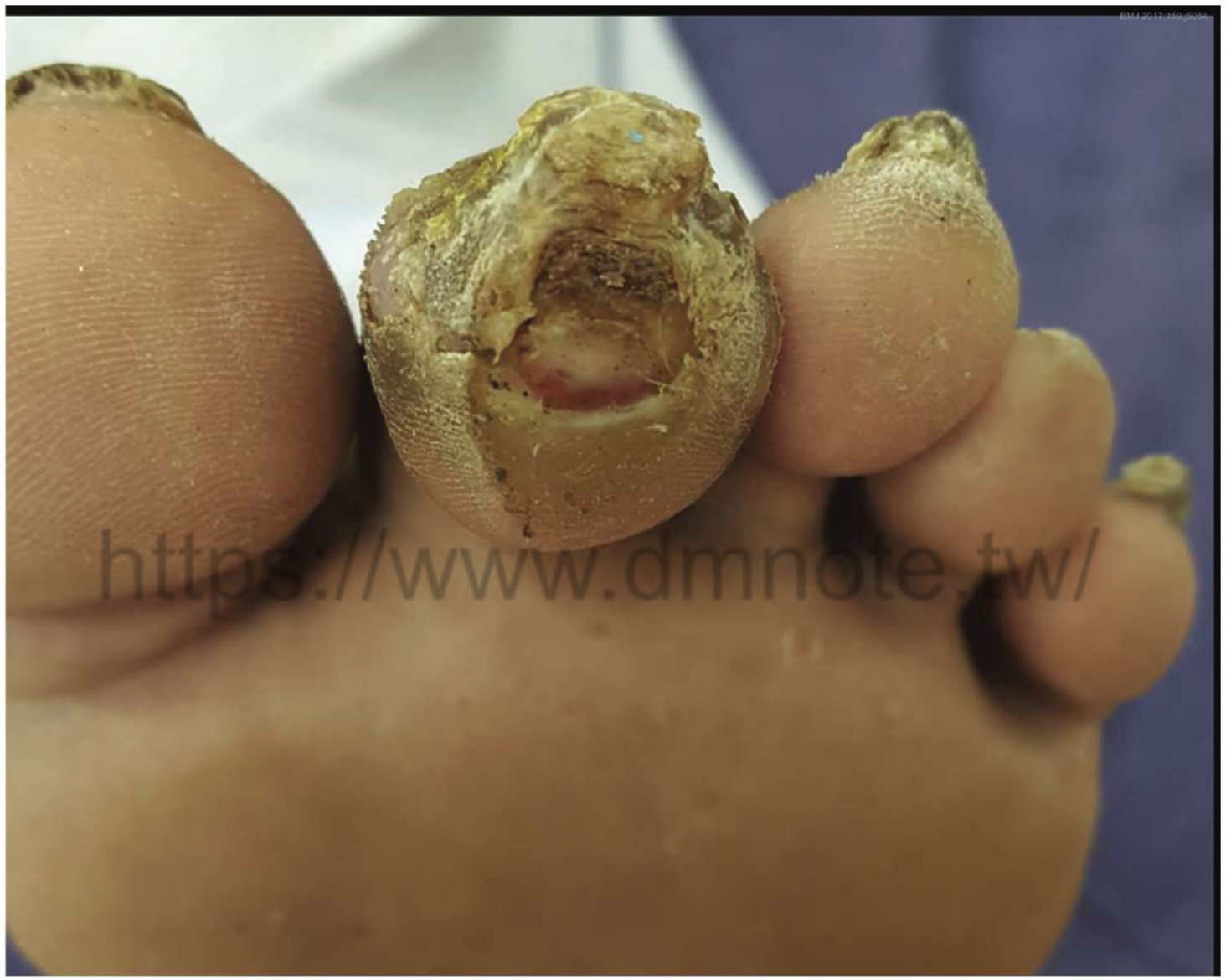


Fig 3 | Hammer toe deformity with callus and ulcer. Hammer toe is caused by weakened muscles in the foot. The joint connecting the foot with the toe bends upwards and the joint in middle of the toe bends downwards towards the floor. This results in the toe curling under the foot and being subjected to excessive ground reaction forces during walking.

糖尿病足傷口與神經病變照護

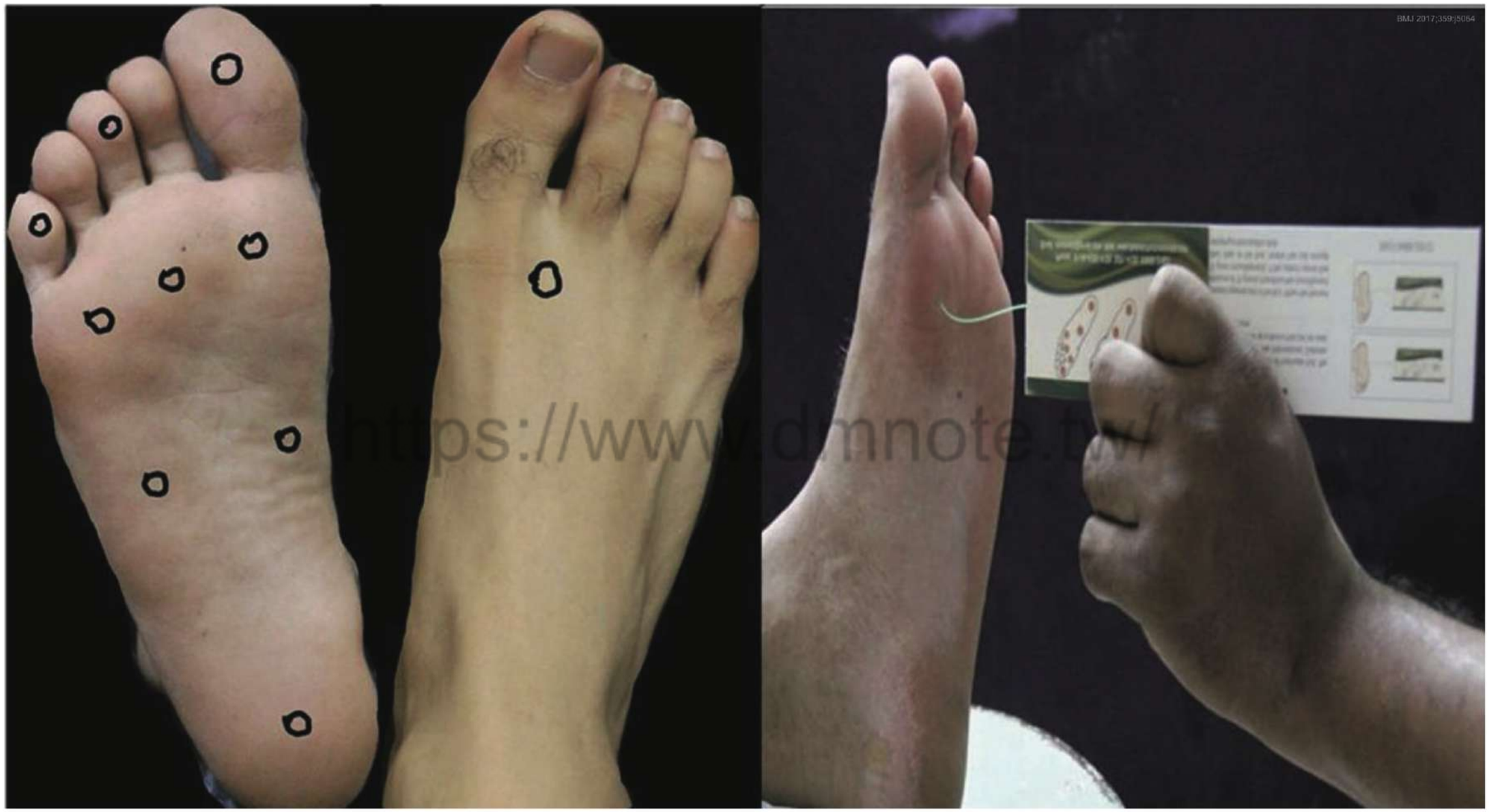


Fig 4 | Monofilament test: testing sites and application. The nine plantar sites are the distal great toe; third toe; fifth toe; first, third, and fifth metatarsal heads; medial foot, lateral foot, and heel; and one dorsal site

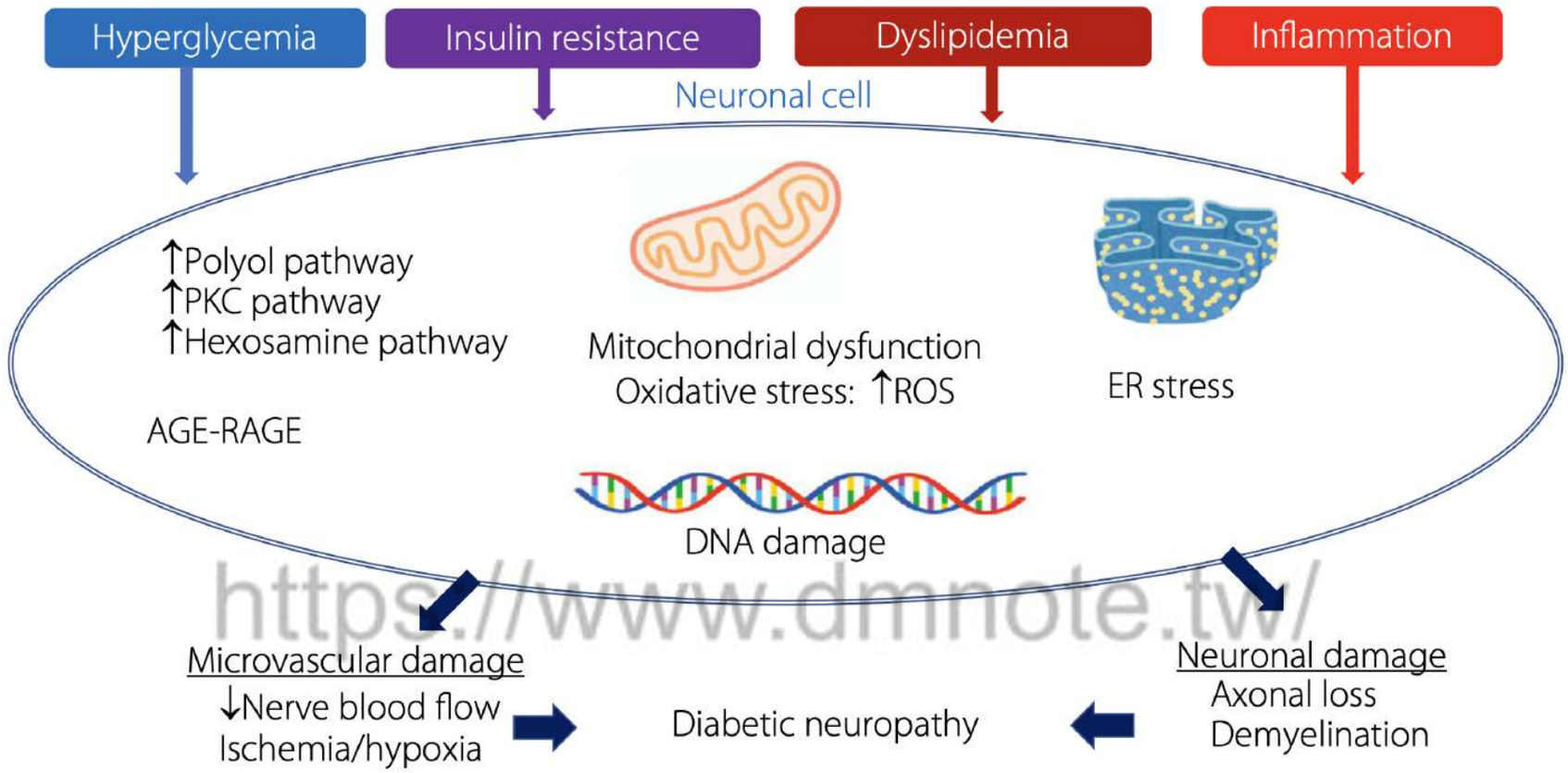
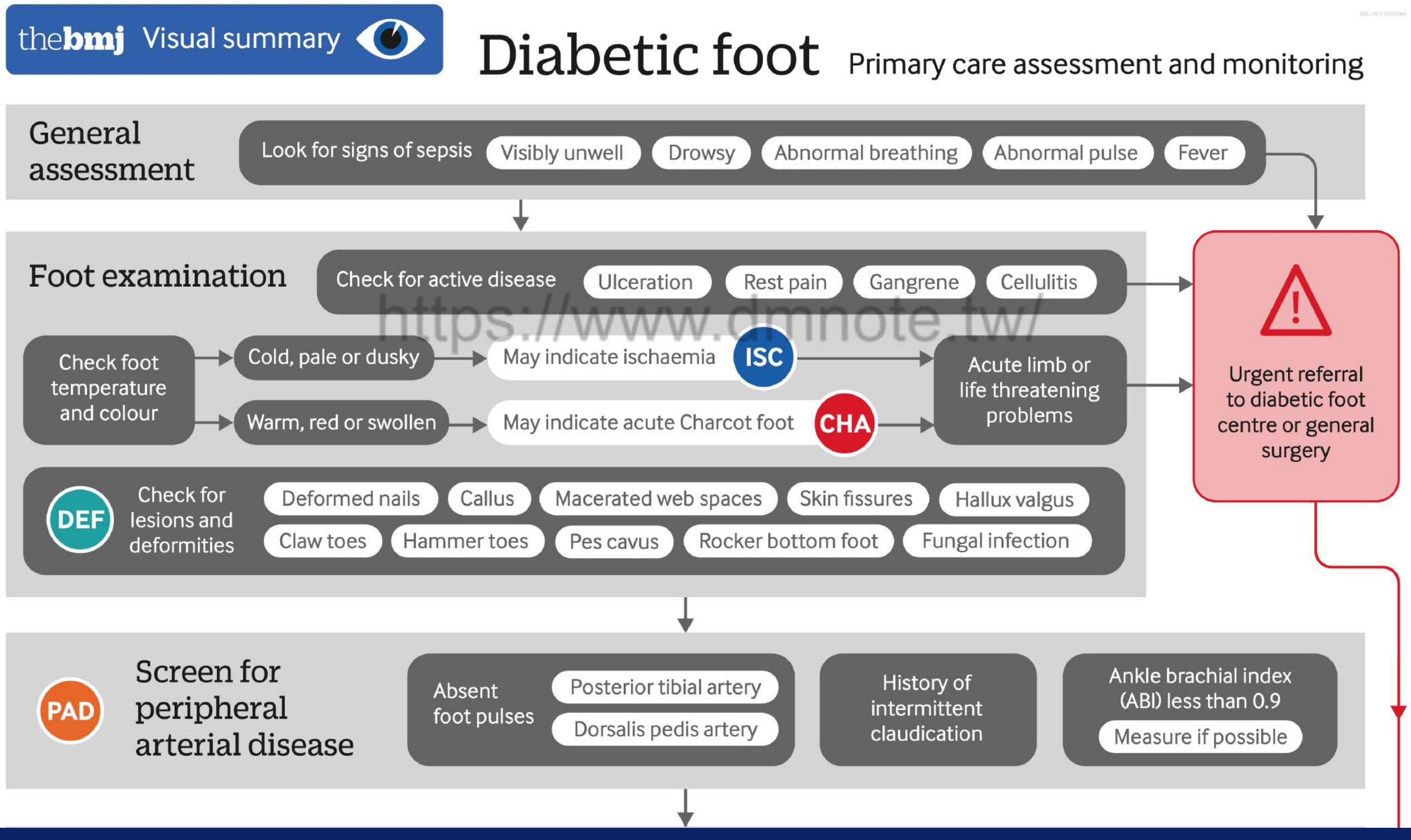
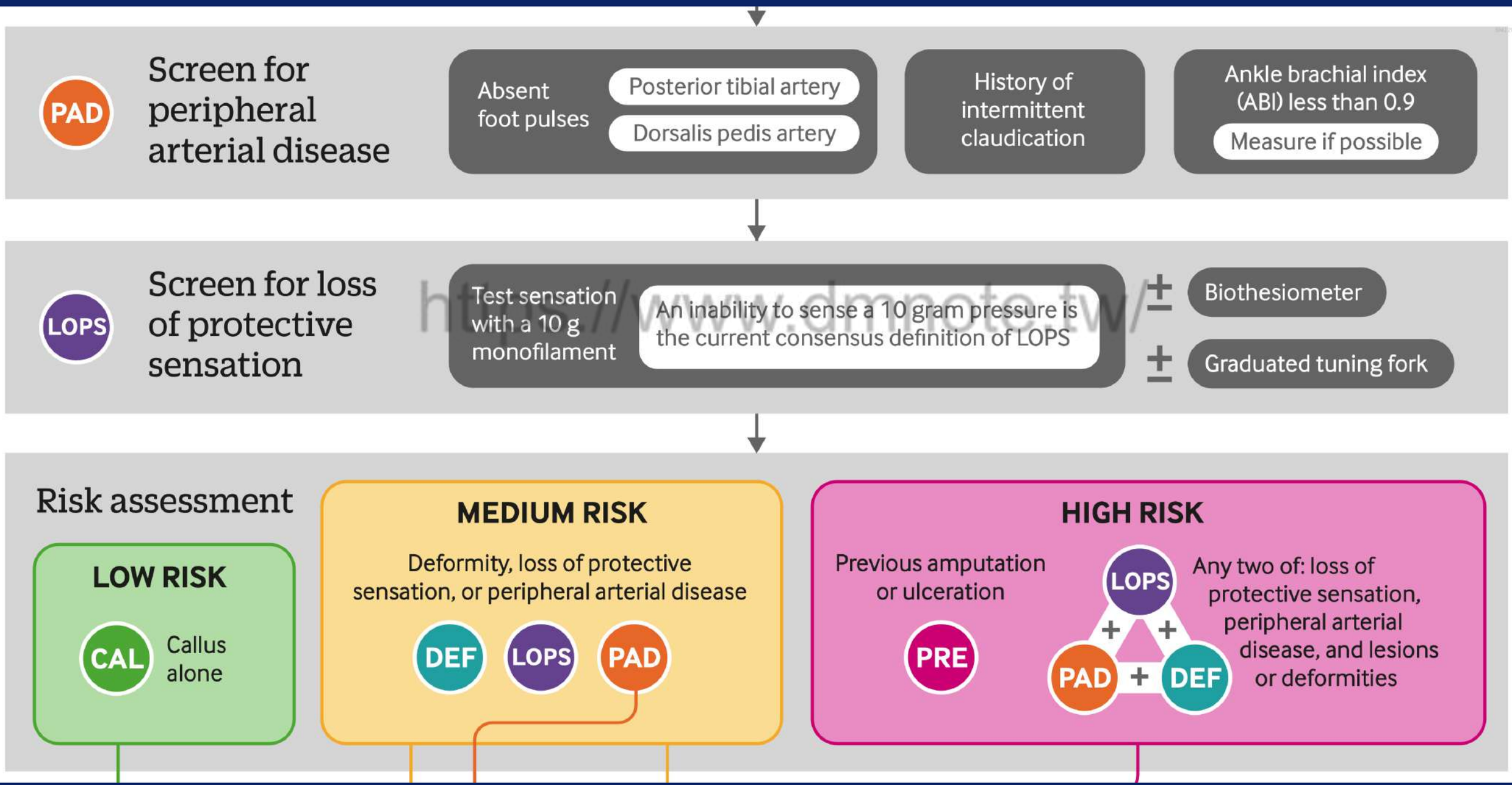


Figure 1 | Mechanisms of diabetic neuropathy. Chronic hyperglycemia induces an excessive activation of the polyol, protein kinase C and hexosamine pathways. In addition, chronic hyperglycemia leads to an increased production of advanced glycation end-products (AGEs), which induces functional and structural neuronal damage through interaction with the AGE-specific receptors. Hyperglycemia, insulin resistance and dyslipidemia contribute synergistically to mitochondrial dysfunction, overproduction of reactive oxygen species, inflammation, endoplasmic reticulum stress and deoxyribonucleic acid (DNA) damage, leading to neuronal cell damage. Both neuronal damage (demyelination and axonal loss) and endoneurial microvascular damage cause diabetic neuropathy in people with diabetes. AGE-RAGE, advanced glycation end-product–specific receptors; ER, endoplasmic reticulum; ROS, reactive oxygen species.

糖尿病足傷口與神經病變照護



糖尿病足傷口與神經病變照護



糖尿病足傷口與神經病變照護

Risk assessment

LOW RISK

CAL — Callus alone

MEDIUM RISK

Deformity, loss of protective sensation, or peripheral arterial disease

DEF　LOPS　PAD

HIGH RISK

Previous amputation or ulceration

PRE

LOPS + PAD + DEF

Any two of: loss of protective sensation, peripheral arterial disease, and lesions or deformities

Patient education

- Glycaemic control
- When to seek help
- Modifiable risk factors
- Foot care

Manage PAD*

- Statins + 1 antiplatelet
- Exercise to improve circulation
- Consider referral for further investigations and revascularization

Routine referral

⚠ Urgent referral

DIABETIC FOOT CENTRE

Foot protection services

- Callus debridement
- Nail care

Surgical management

- Wound debridement
- Amputations

Liaison or referral

Vascular intervention and orthoses services

* Adapted from NICE guidance on diabetic foot and peripheral arterial disease

Primary care follow-up

LOW RISK	MEDIUM RISK	HIGH RISK
Every year	Every 3–6 months	Every 1–2 months

視網膜玻璃體變

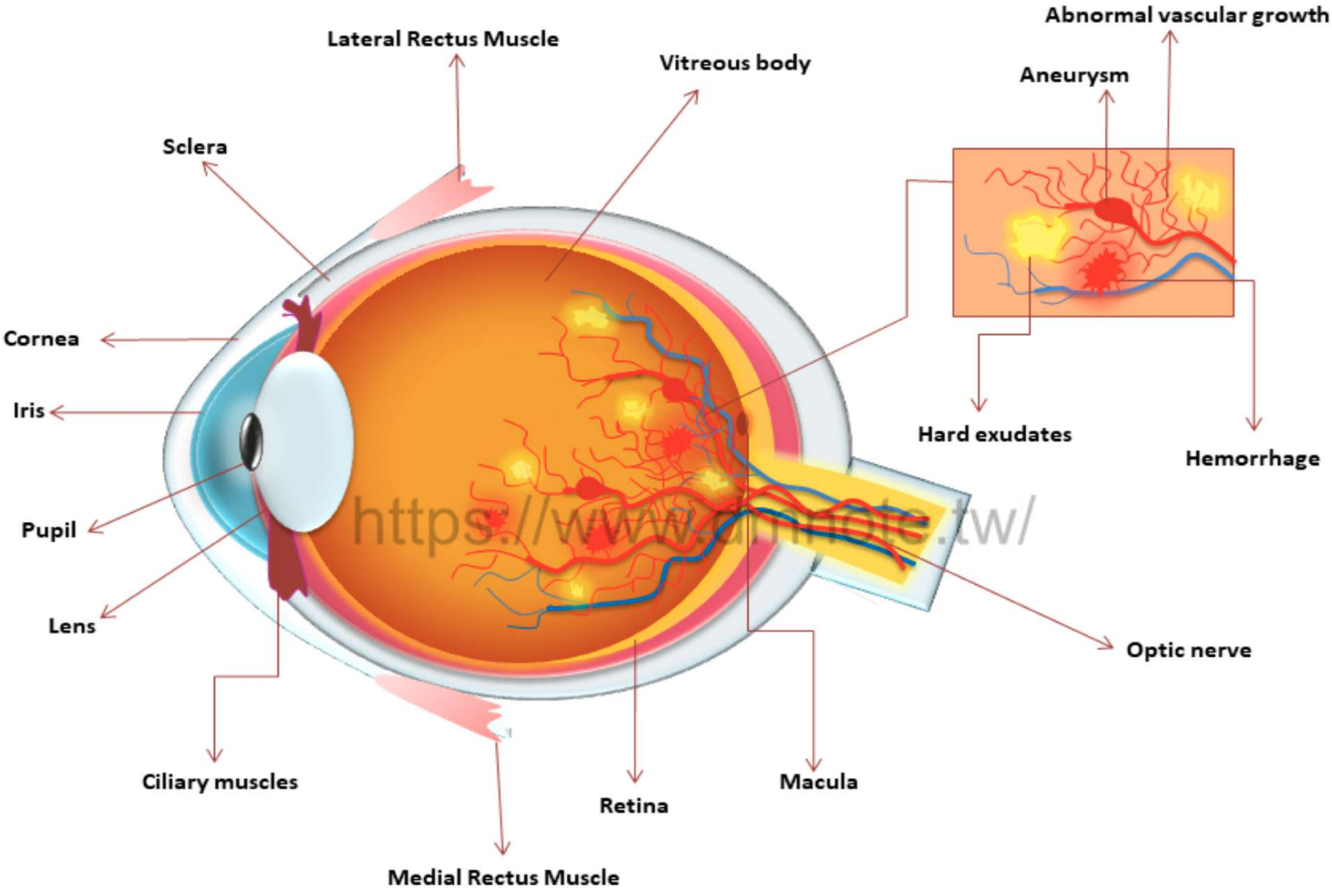


Figure 1. Different pathological complications in diabetic retinopathy: Anatomy of complications faced such as retinal vessel hemorrhage and microaneurysms, abnormal vascular development on the retinal surface, and the accumulation of yellowish thick fluids towards the middle of the retina results in edema formation.

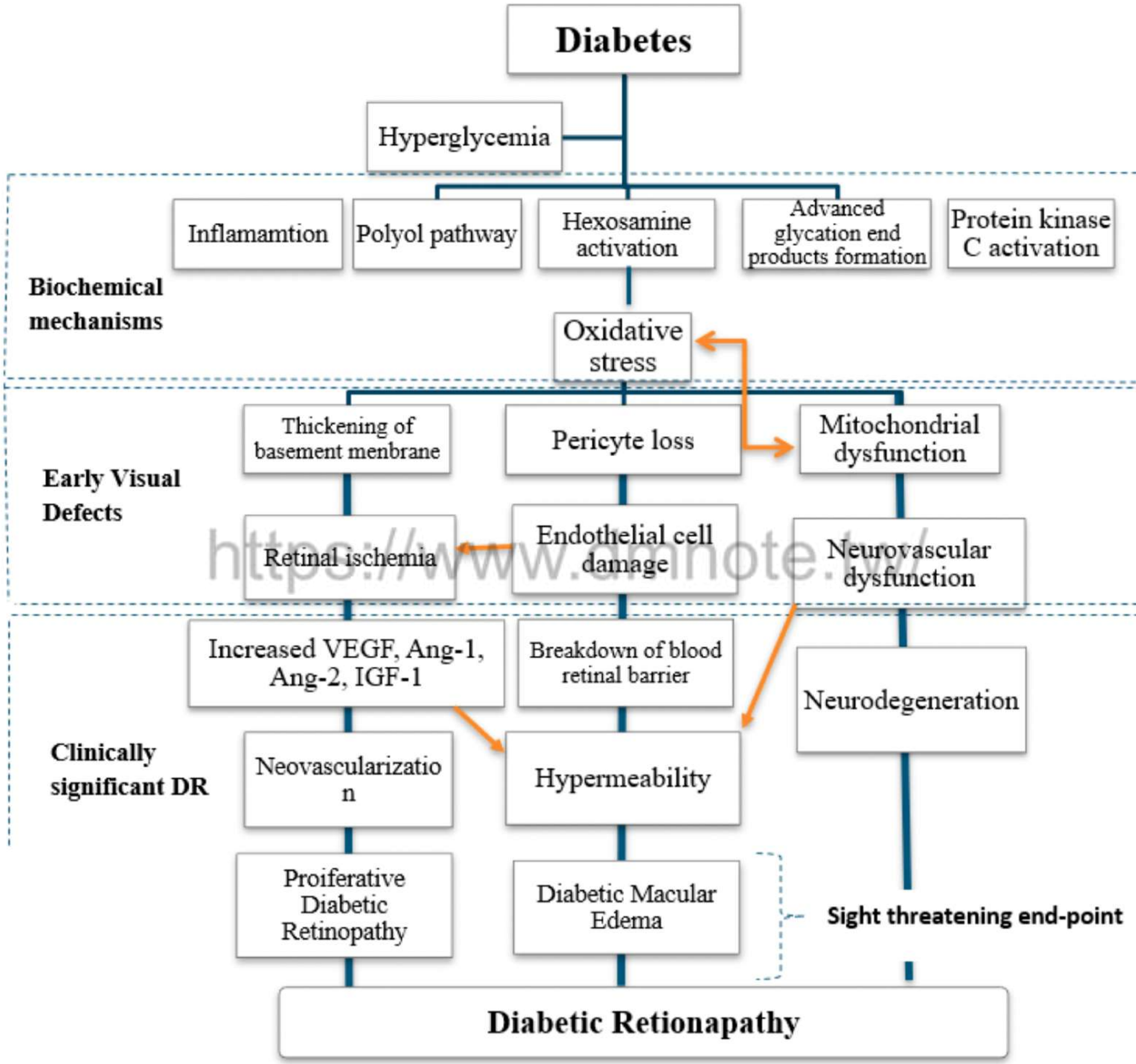


Figure 2. Diagrammatic synopsis of the pathogenesis and pathophysiology of DR.

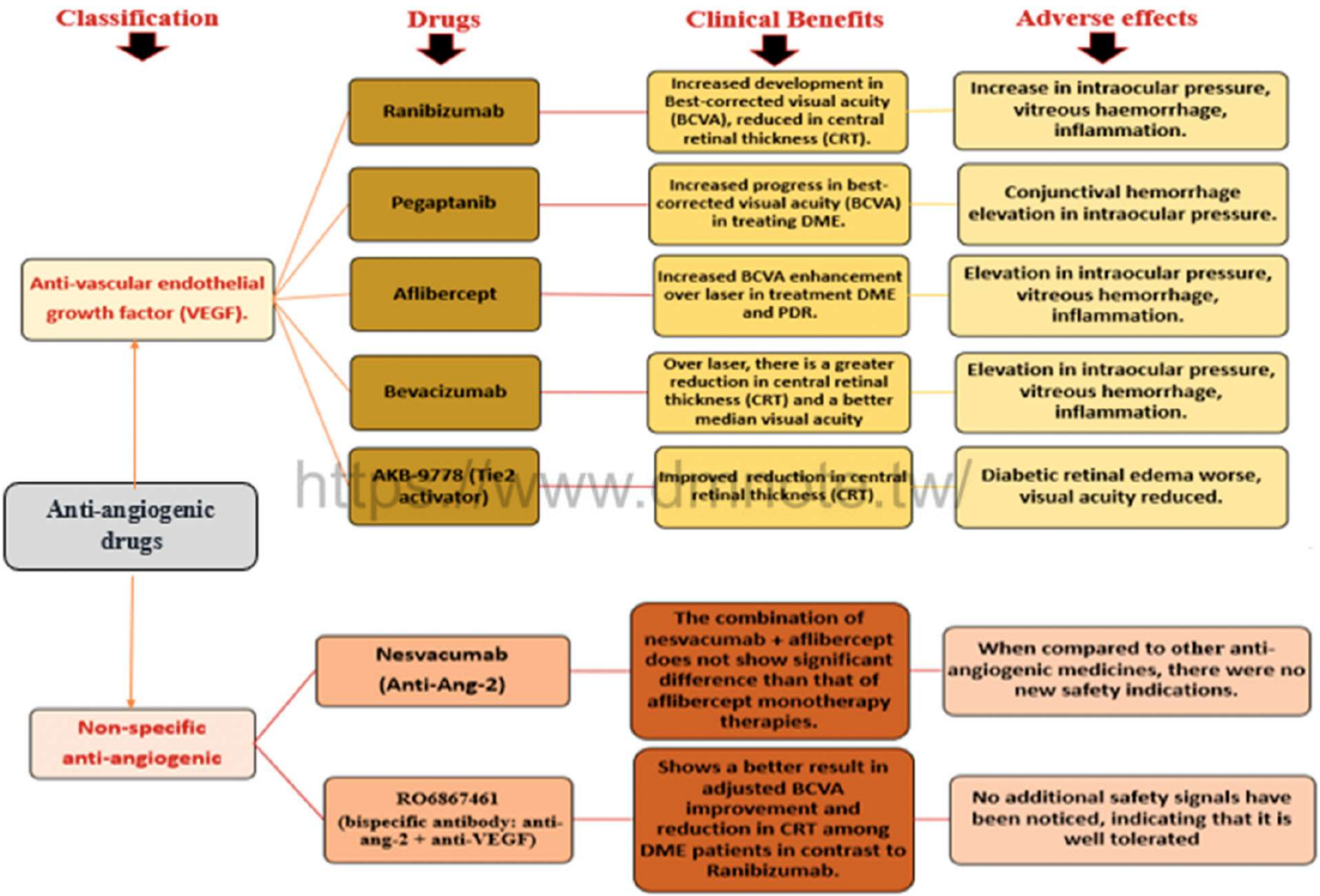


Figure 5. Anti-angiogenic drugs for the treatment of diabetic retinopathy (DR) [10].

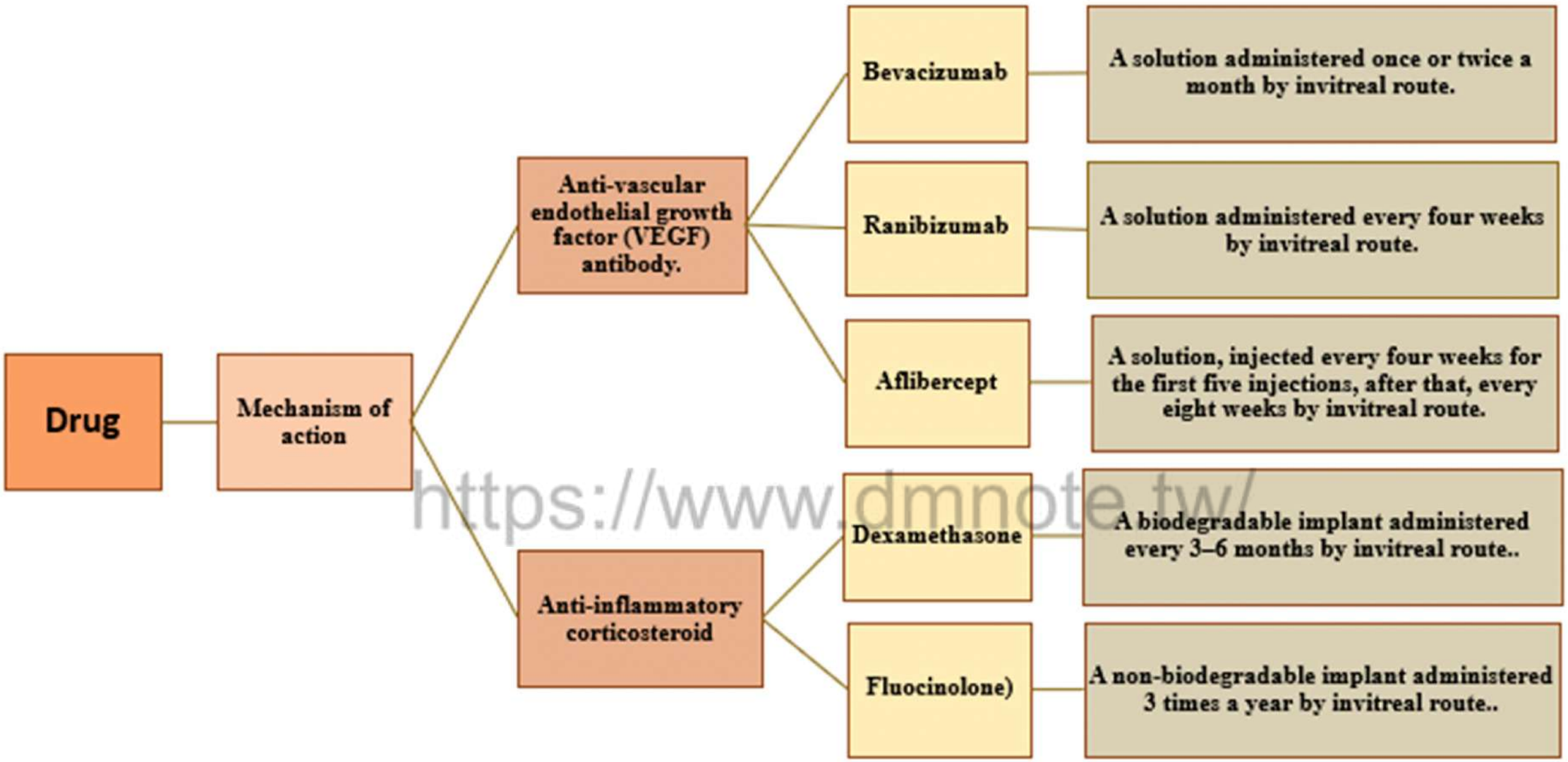
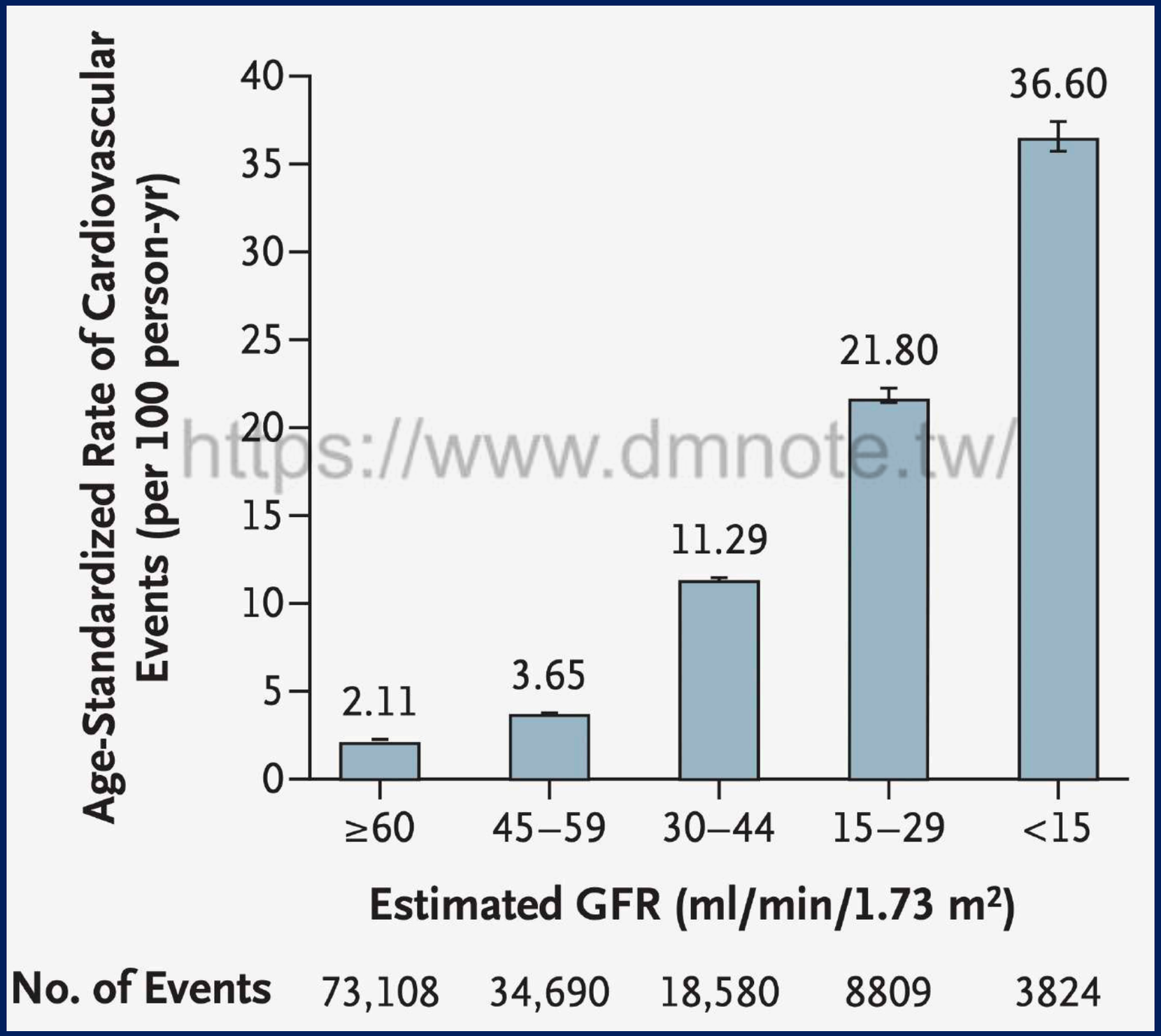


Figure 4. Summary of the treatments for diabetic macular edema that are currently available in the market [80].

腎臟病變

Chronic Kidney Disease and the Risks of Cardiovascular Events



Ref:N Engl J Med 2004;351:1296-305.

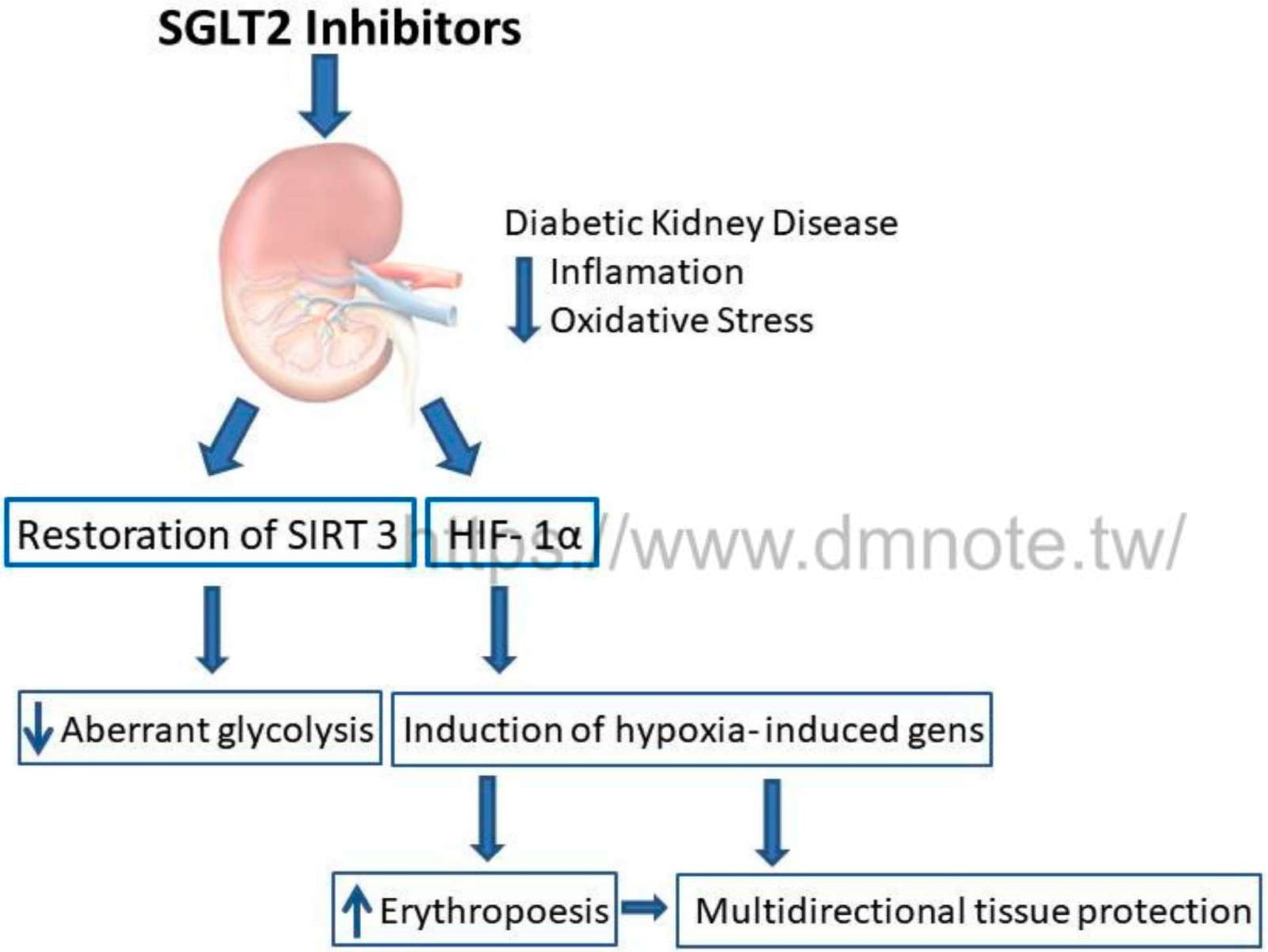


Figure 1. Interaction between SGLT2 inhibition, HIF1α and tissue protection in diabetic kidney disease.

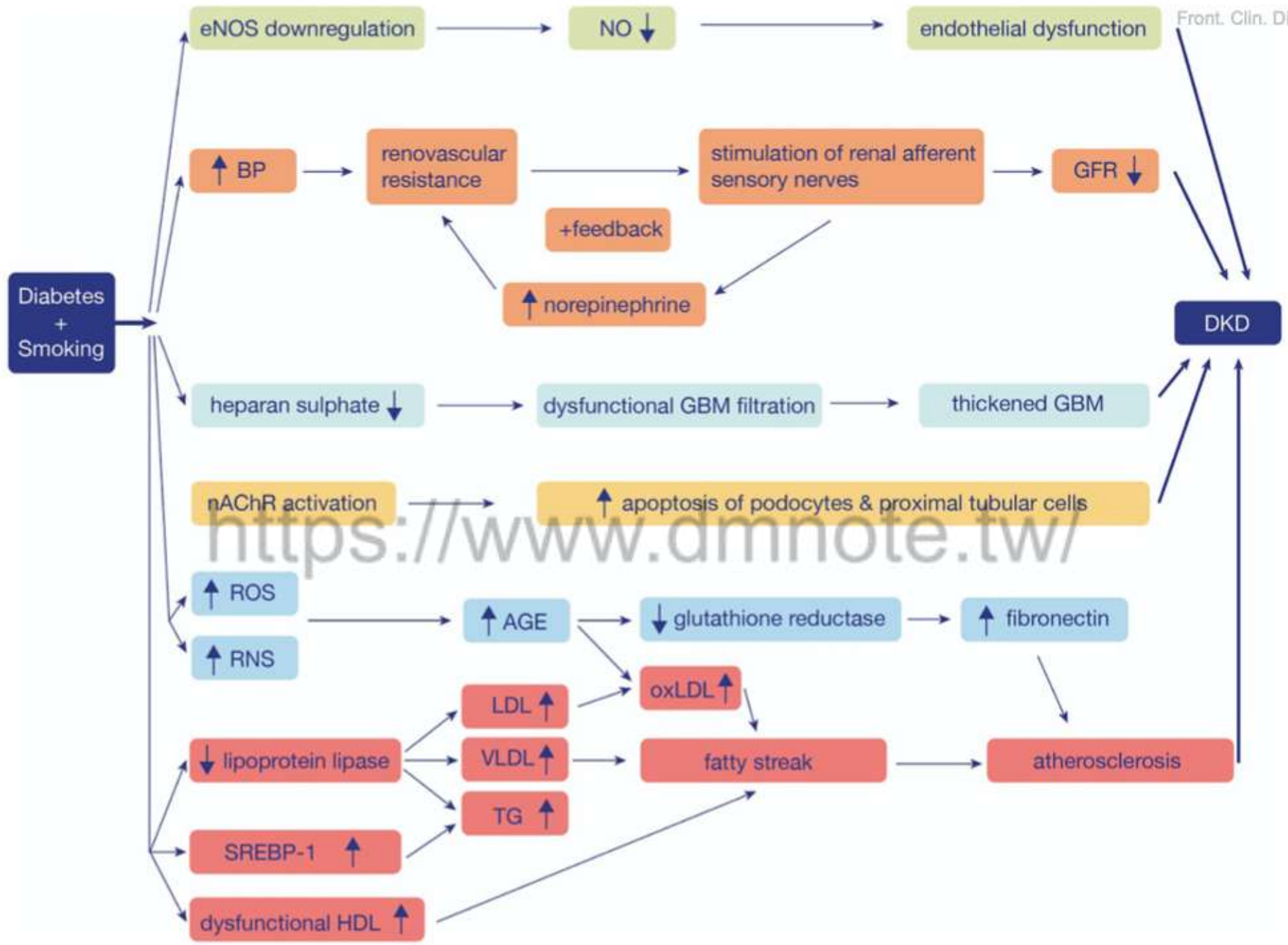


FIGURE 1 | The mechanistic pathway of the role of smoking and diabetes in the progression of diabetic kidney disease. eNOS, Endothelial nitric oxide synthase; NO, Nitric oxide; BP, Blood pressure; GFR, Glomerular Filtration Rate; DKD, Diabetic Kidney Disease; GBM, Glomerular basement membrane; nAChR, Nicotinic acetylcholine receptor; ROS, Reactive oxygen species; RNS, Reactive nitrogen species; AGE, Advanced glycation end products; LDL, Low-density lipoprotein; VLDL, Very Low Density Lipoprotein; TG, Triglyceride; oxLDL, oxidized Low-density lipoprotein; SREBP-1, sterol regulatory element binding protein-1; HDL, High-density lipoprotein.

心血管疾病

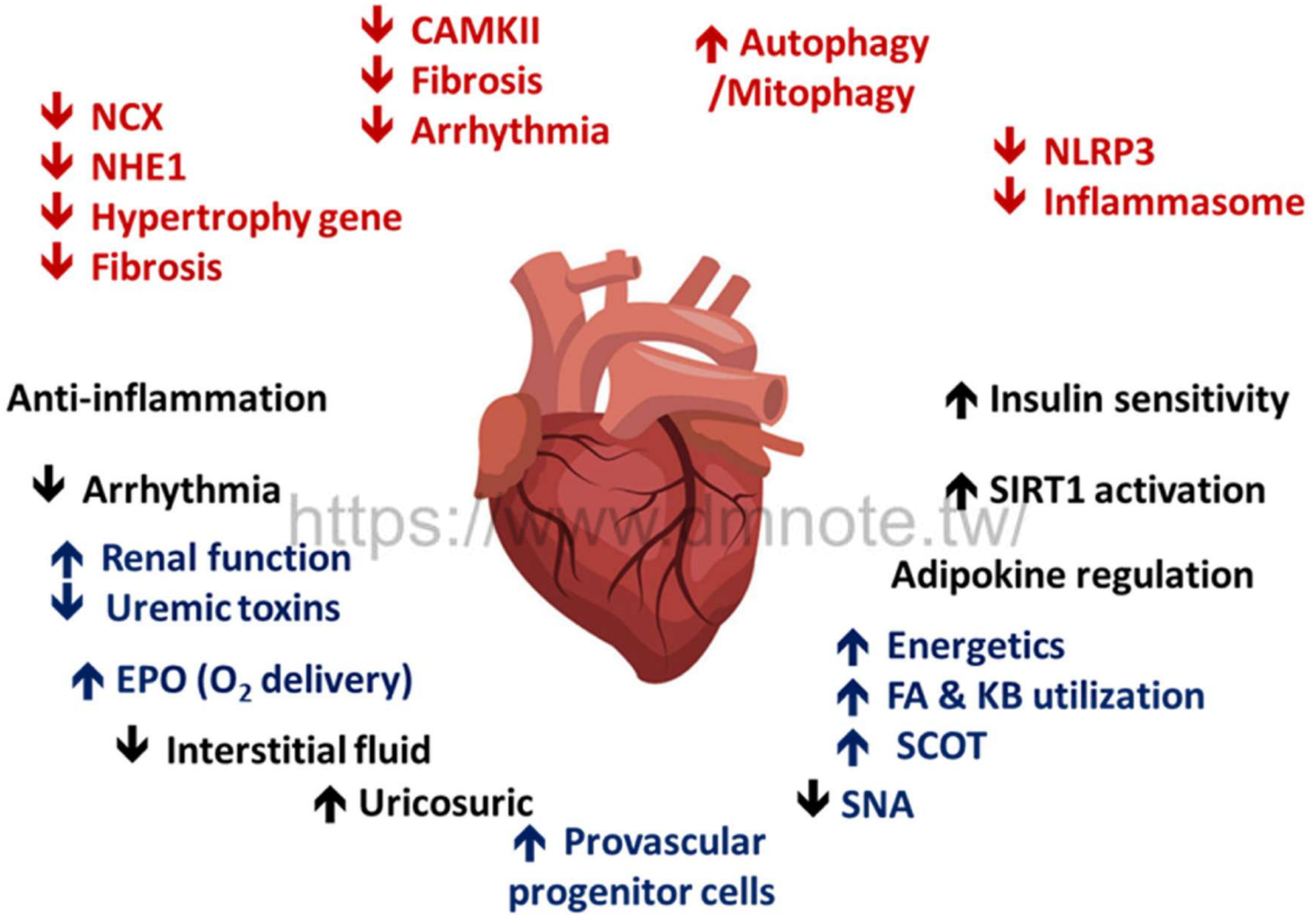


Figure 4. Direct (red) myocardial and indirect/systemic (blue) effects of SGLT2i: the cardioprotective effect of SGLT2i. SGLT2i could alleviate cardiac fibrosis by modulating autophagy and inflammation. SGLT2i also indirectly alleviates cardiac injury by modulating metabolism and the sympathetic tone.

總結

- 糖尿病的診斷與分類
- 血糖控制目標
- 糖尿病的治療
- 糖尿病的併發症

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內分泌新陳代謝科主任

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