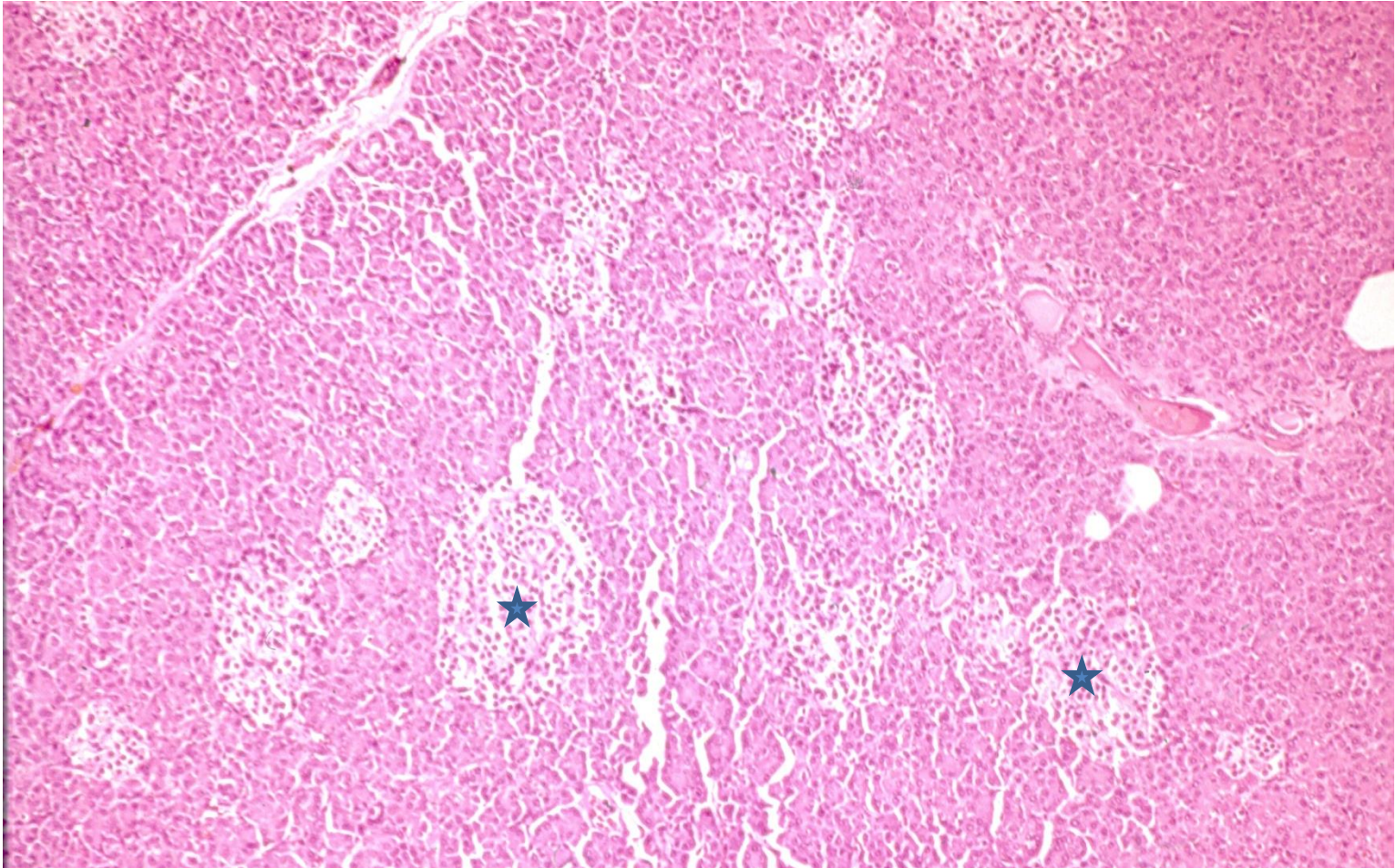
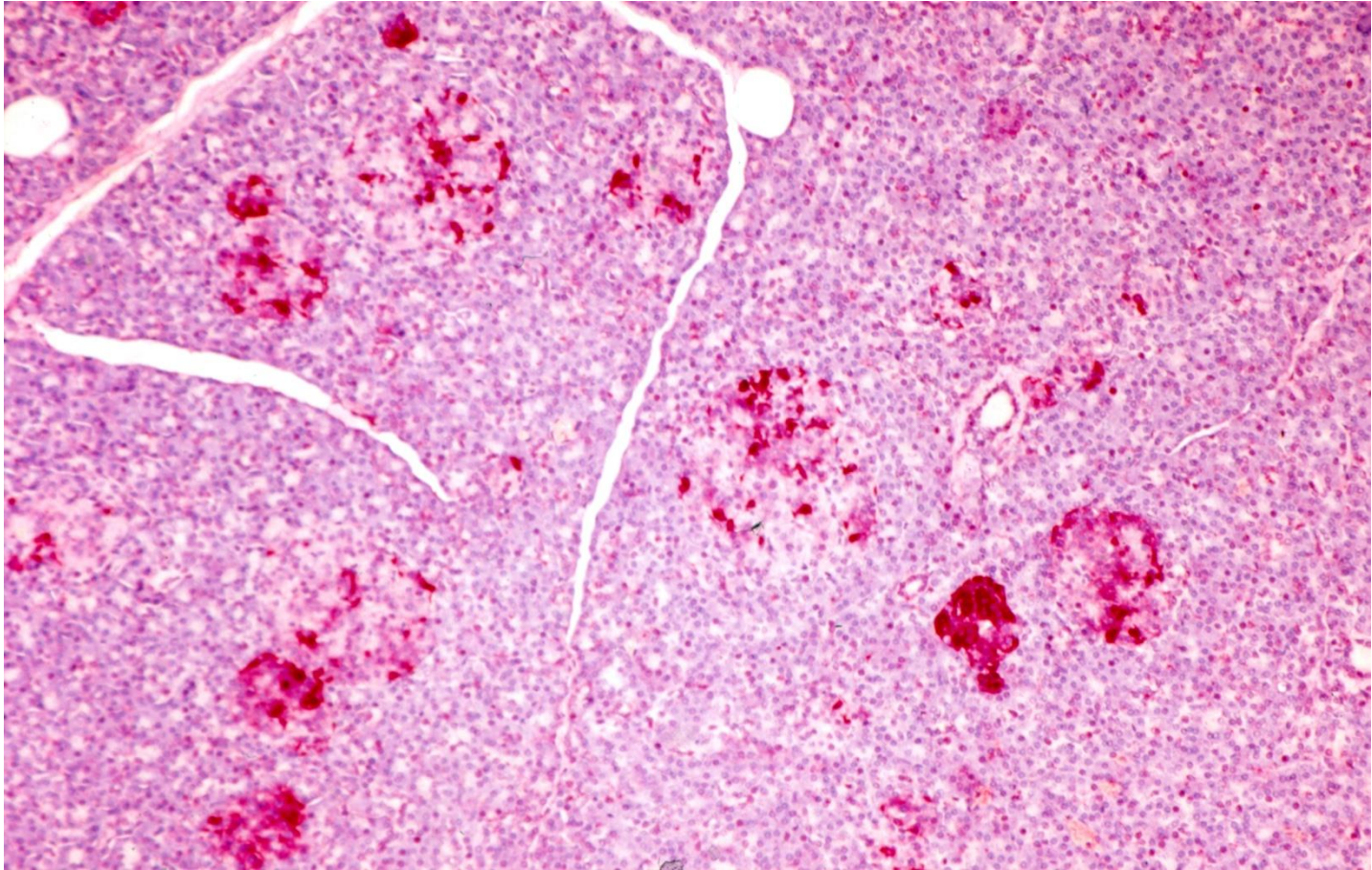


Σακχαρώδης Διαβήτης

Πάγκρεας



★ Νησίδια Langerhans



ΑΡΕΤΑΙΟΣ Ο ΚΑΠΠΑΔΟΚΗΣ (1^{ος} αι. μ.Χ.)
Περί αιτίων και σημείων οξέων και χρονίων παθών
Εισήγαγε τον όρο «Διαβήτης»



Milestones in the history of diabetes mellitus: The main contributors

Marianna Karamanou, Athanase Protogerou, Gregory Tsoucalas, George Androutsos, Effie Poulakou-Rebelakou

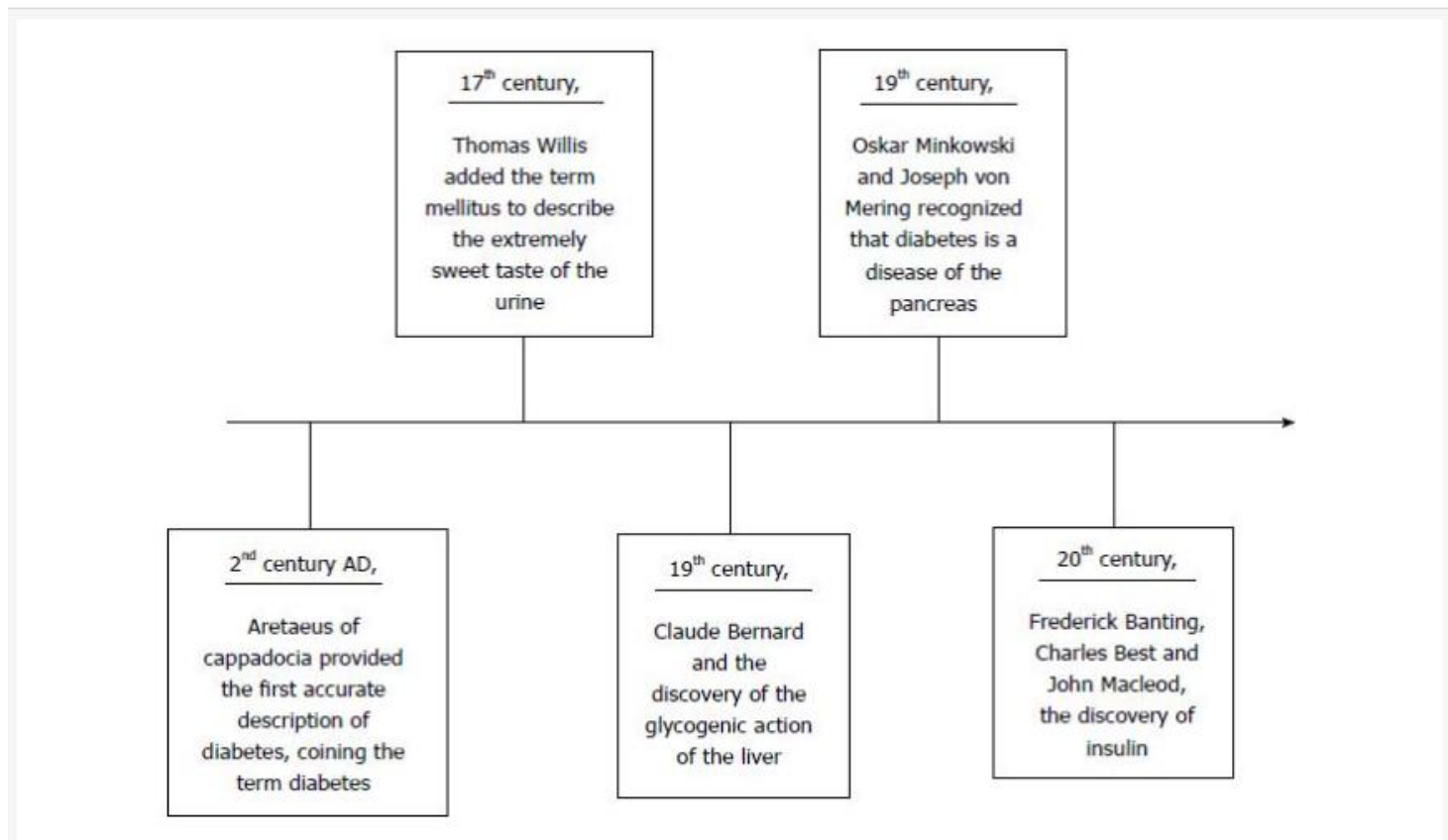


Figure 5 Timeline table presenting the main contributors in the history of diabetes mellitus.

Banting & Best: Discovery of insulin

July 27, 1921

By: UMass Diabetes Center of Excellence



Banting and Best with the first dog ever treated with insulin



415 εκατομμύρια άνθρωποι παγκοσμίως νοσούν από Σακχαρώδη Διαβήτη (2017)

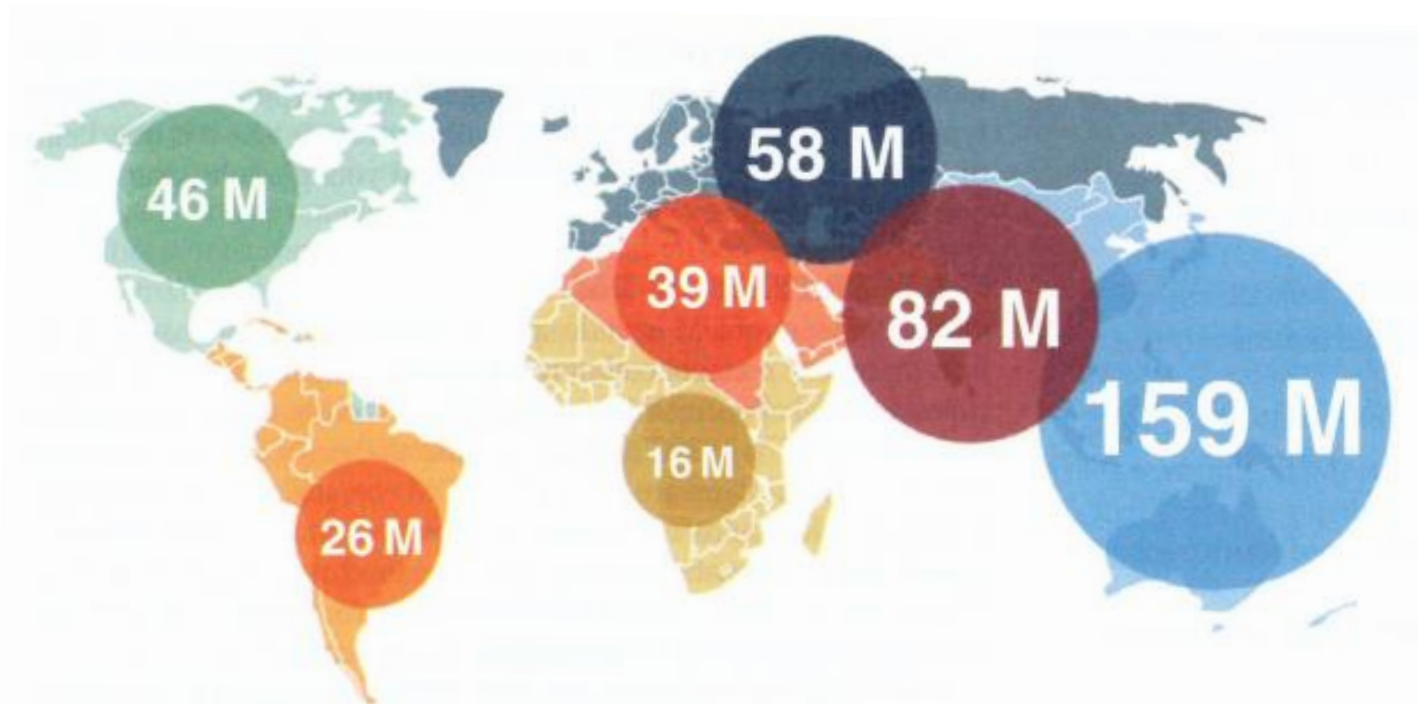


FIGURE 396-2 Worldwide prevalence of diabetes mellitus. Global estimate is 415 million individuals with diabetes in 2017. Regional estimates of the number of individuals with diabetes (20–79 years of age) are shown (2017). (Adapted from the *IDF Diabetes Atlas*, the International Diabetes Federation, 2017.)

HARRISON'S PRINCIPLES OF INTERNAL MEDICINE, 20th EDITION, 2018, McGraw Hill.

Ταξινόμηση Σακχαρώδη Διαβήτη με βάση την μηχανισμό παθογένεσης

I. Τύπος 1 / Type 1 (immune-mediated β -cells destruction > usually leading to absolute insulin deficiency) (παλαιότερη ονομασία σακχαρώδης διαβήτης των νέων – ινσουλινοεξαρτώμενος)

II. Τύπος 2 / Type 2 (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretory defect with insulin resistance) (παλαιότερη ονομασία σακχαρώδης διαβήτης των ενηλίκων – ινσουλινοεξαρτώμενος)

III. Σακχαρώδης διαβήτης της κύησης / Gestational Diabetes mellitus (developing during the 2nd-3rd trimester of pregnancy; insulin resistance associated with the metabolic alterations occurring during pregnancy)

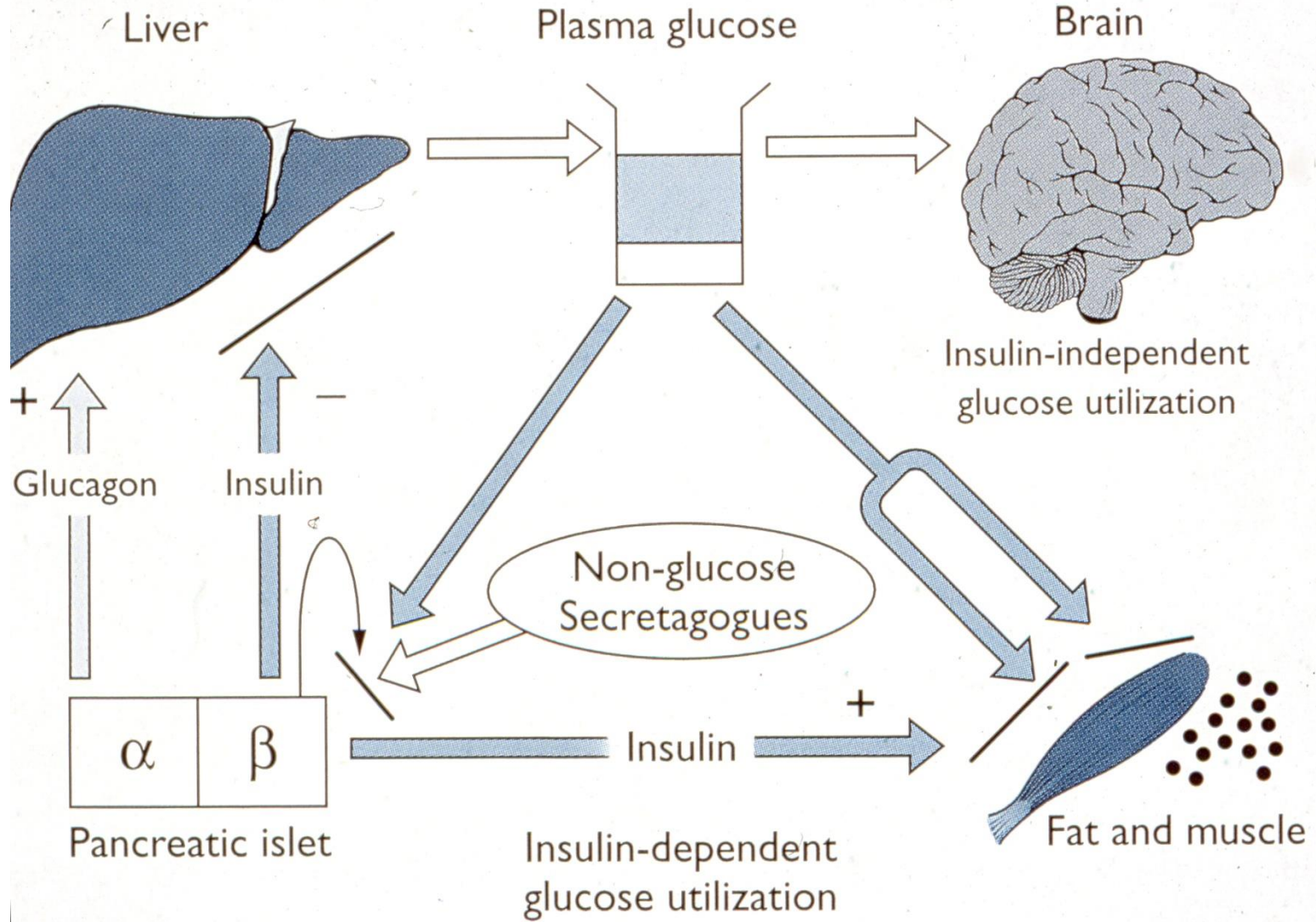
IV. Άλλοι τύποι / Other specific types

- Genetic defects of β -cell function
- Genetic defects in insulin action
- Diseases in exocrine pancreas (i.e. pancreatitis, neoplasia)
- Endocrinopathies (i.e. Cushing, pheochromocytoma)
- Drugs or Chemically induced (i.e. glucocorticoids, thiazides)
- Infections (i.e. CMV)
- Uncommon forms of immune-mediated diabetes
- Other genetic syndromes associated with DM (i.e. Down's syndrome)

ΣΥΓΚΡΙΣΗ ΤΥΠΩΝ 1 ΚΑΙ 2 ΣΑΚΧΑΡΩΔΟΥΣ ΔΙΑΒΗΤΗ

	ΤΥΠΟΣ 1	ΤΥΠΟΣ 2
Ηλικία εμφάνισης	< 20 ετών	> 30 ετών
Σωματικό βάρος	Φυσιολογικό	Παχυσαρκία
Ινσουλίνη αίματος	Χαμηλή	Φυσιολογική ή αυξημένη
Αυτοαντισώματα κατά κυττάρων νησιδίων	Ναι	Όχι
Κετοξέωση	Συχνή	Σπάνια
Ταυτόχρονη εμφάνιση σε μονοωογενή δίδυμα	50 %	60 – 80 %
Σύνδεση με HLA	Ναι	Όχι
Ανοσοπαθολ. μηχανισμοί	Ναι	Όχι
Ανεπάρκεια ινσουλίνης «Νησιδίτις»	Σοβαρή Ναι	Ελαφρά – Μέτρια Όχι
Ατροφία νησιδίων	Εκσεσημασμένη	Εστιακή
Τνώση νησιδίων	Ναι	Όχι
Αμυλοείδωση νησιδίων	Όχι	Ναι
Ελάττωση αριθμού β-κυττάρων	Μεγάλη	Μικρή – Μέτρια

Ομοιοστασία Γλυκόζης



Ομοιοστασία Γλυκόζης & Σακχαρώδης Διαβήτης

Type of Diabetes	Normal glucose tolerance	Hyperglycemia	
		Pre-diabetes*	Diabetes Mellitus
		Impaired fasting glucose or impaired glucose tolerance	Not insulin requiring Insulin required for control Insulin required for survival
Type 1			
Type 2			
Other specific types			
Gestational Diabetes			
Time (years)			
FPG	<5.6 mmol/L (100 mg/dL)	5.6–6.9 mmol/L (100–125 mg/dL)	≥7.0 mmol/L (126 mg/dL)
2-h PG	<7.8 mmol/L (140 mg/dL)	7.8–11.0 mmol/L (140–199 mg/dL)	≥11.1 mmol/L (200 mg/dL)
HbA1C	<5.6%	5.7–6.4%	≥6.5%

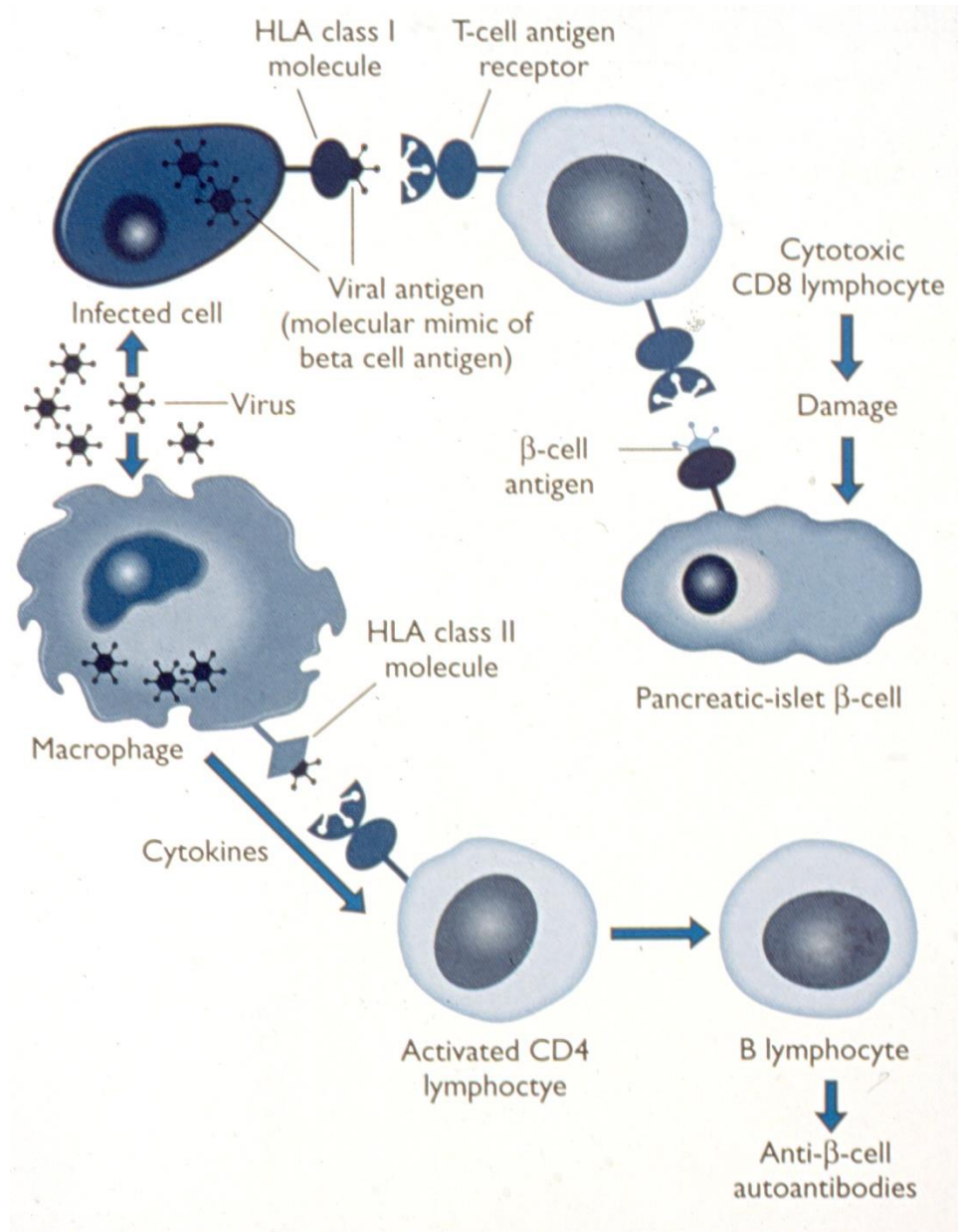
FPG: fasting plasma glucose; PG: plasma glucose; Arrows denote that changes in glucose tolerance may be bidirectional (i.e. weight loss improves glucose tolerance in individuals with DMII)

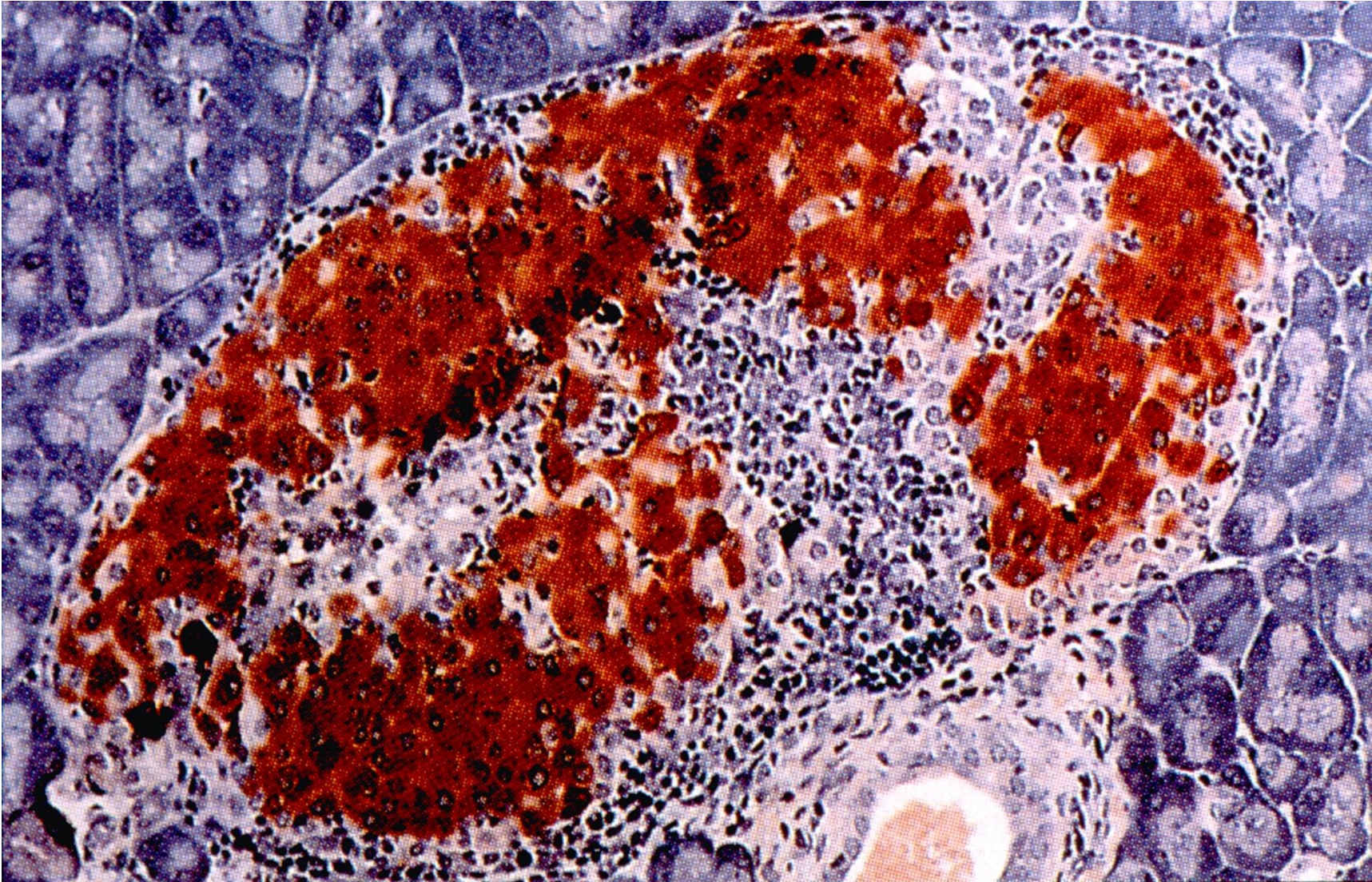
Παθογενετικός Μηχανισμός Σακχαρώδη Διαβήτη τύπου I

- Περιβαλλοντικά (όπως πρωτεΐνες από βόειο γάλα, φάρμακα, έλλειψη βιταμίνης D) ή λοιμογόνα (όπως ιοί, π.χ. Coxsachie, enteroviruses, rubella/ερυθρά) αίτια σε ασθενείς με το κατάλληλο γενετικό υπόβαθρο θεωρείται πώς αποτελεί το πρωταρχικό αίτιο > παραγωγή αυτοαντισωμάτων έναντι αντιγόνων β-κυττάρων
- Προοδευτική και επιλεκτική καταστροφή των β-κυττάρων των νησιδίων του παγκρέατος με την μεσολάβηση T λεμφοκυττάρων. Τα β-κύτταρα είναι αρκετά ευάλωτα στη τοξική δράση συγκεκριμένων κυττοκινών (TNF-α, IL-1)

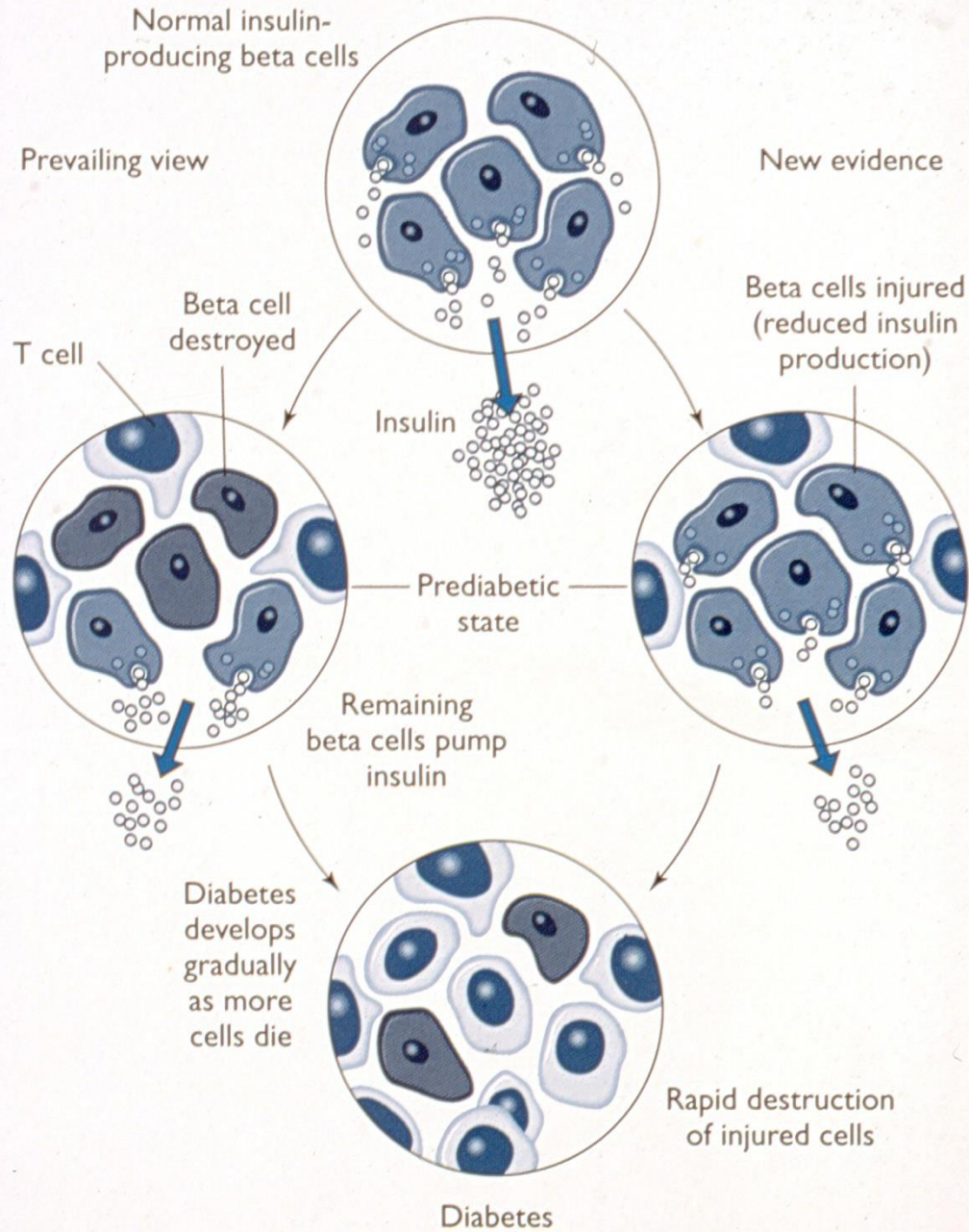
<>Νησιδίτις: διήθηση των νησιδίων Langerhans από λεμφοκύτταρα

- Διαταραχή στην έκκριση ινσουλίνης
- >70% καταστροφή των β-κυττάρων των νησιδίων του παγκρέατος => συμπτώματα από ανεπάρκεια ινσουλίνης





How Quickly does Diabetes Develop?



Παθογενετικός Μηχανισμός Σακχαρώδη Διαβήτη τύπου II

- Διαταραχή στην έκκριση ινσουλίνης + αντίσταση των ιστών στη δράση της ινσουλίνης:

A. Μειωμένη απάντηση στην παραγωγή ινσουλίνης ως απάντηση στα αντίστοιχα διεγερτικά ερεθίσματα – διαταραχή της αρχικής ταχείας αύξησης της έκκρισης ινσουλίνης

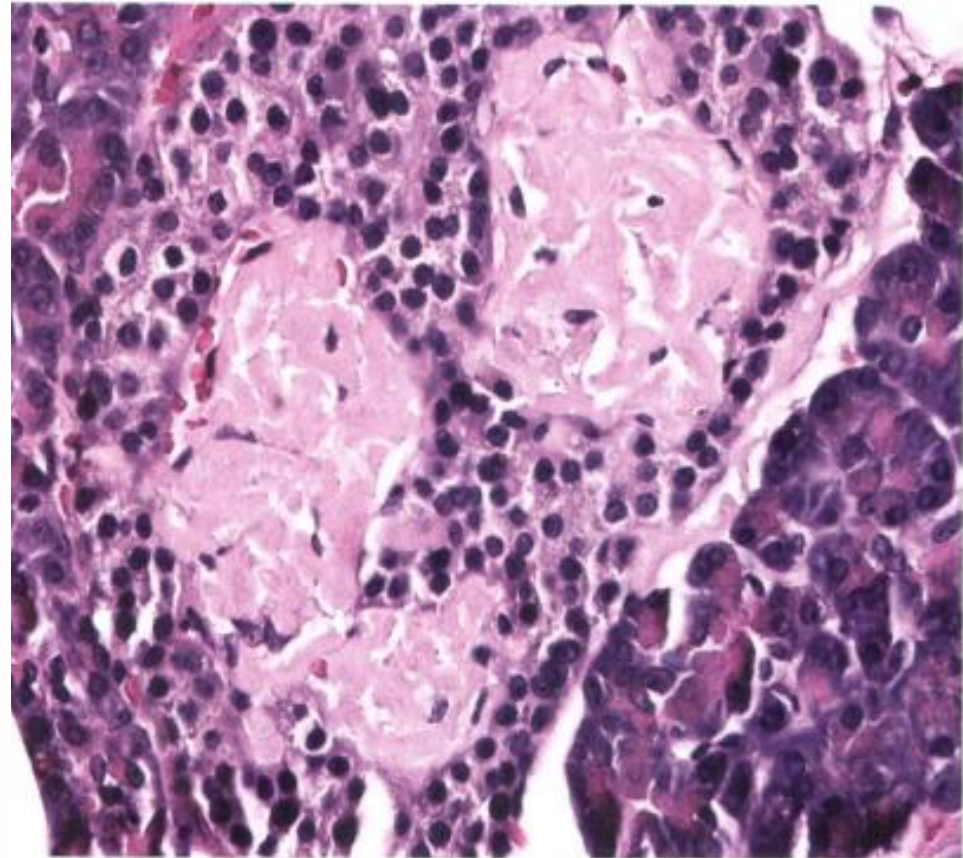
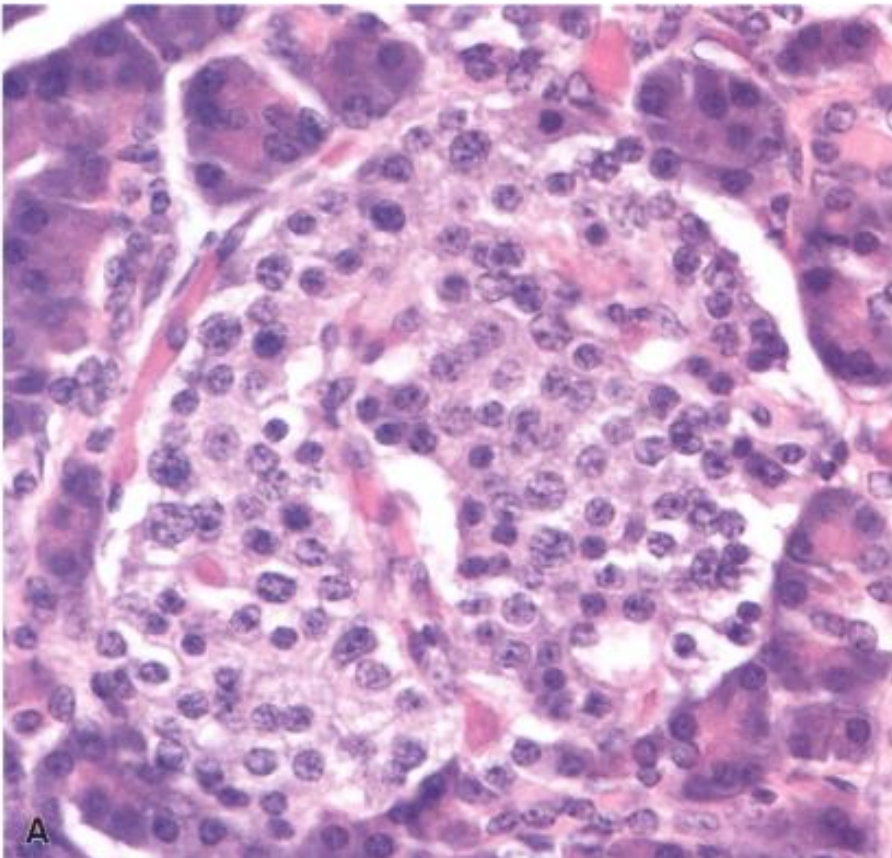
B. Αντίσταση στη δράση της ινσουλίνης (διαταραχή στην μεταβίβαση του σήματος μετά την ένωση ινσουλίνης με τον υποδοχέα της ή/και του υποδοχέα GLUT4 που εντοπίζεται στους μύες και το λιπώδη ιστό)

- Υπόθεση: αρχικά υπάρχει διαταραχή στη δράση της ινσουλίνης ή οποία οδηγεί σε αντιρροπιστική υπερέκκριση ινσουλίνης με αποτέλεσμα την αδυναμία του παγκρέατος να ανταποκριθεί και την προοδευτική «εξάντληση» των β-κυττάρων του παγκρέατος
- Η πλειονότητα των ασθενών με ΣΔII είναι Υπέρβαροι/Παχύσαρκοι:

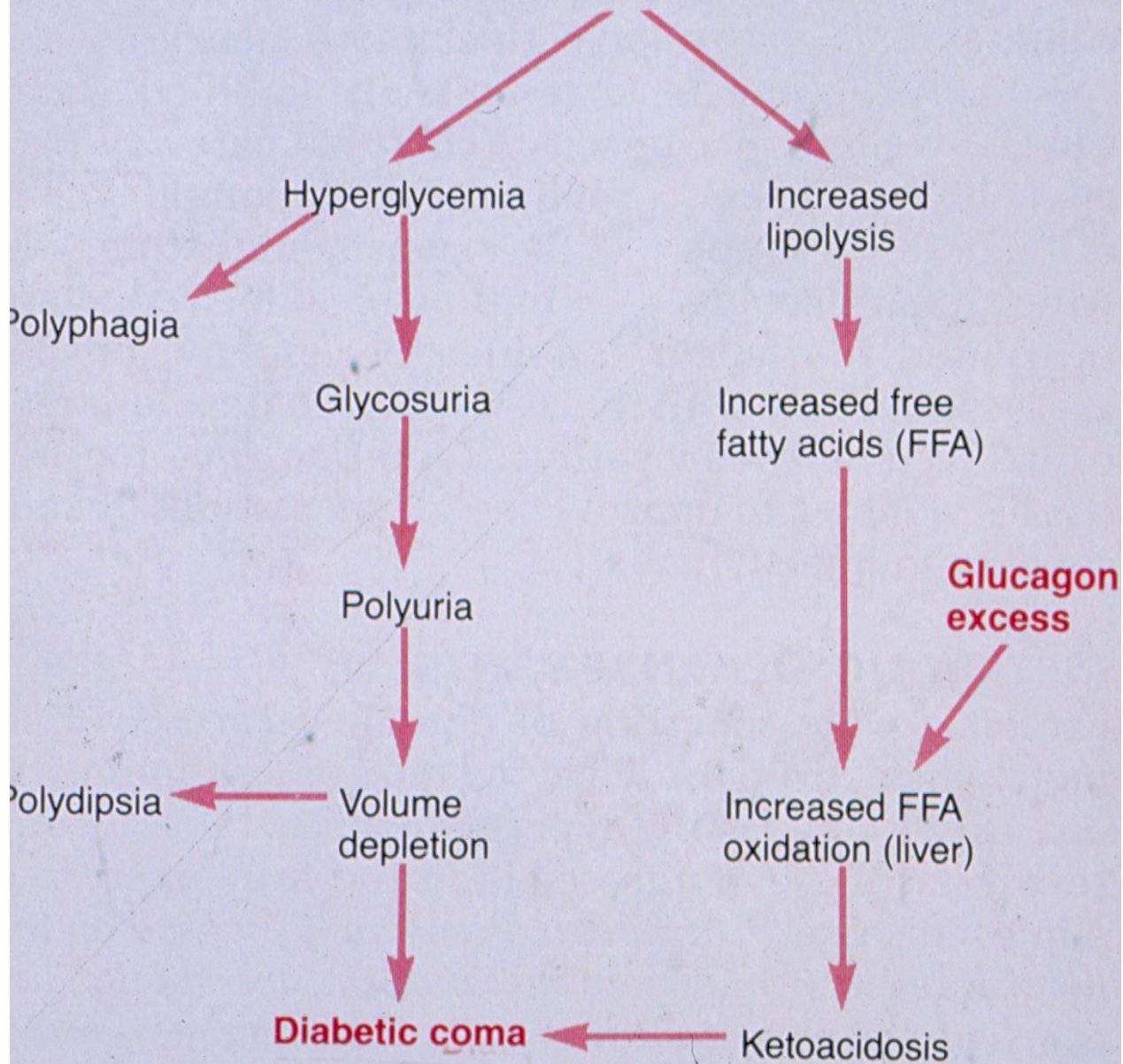
Αμυλοείδωση σε νησίδιο Langerhans παγκρέατος σε ασθενή με Σακχαρώδη Διαβήτη τύπου II

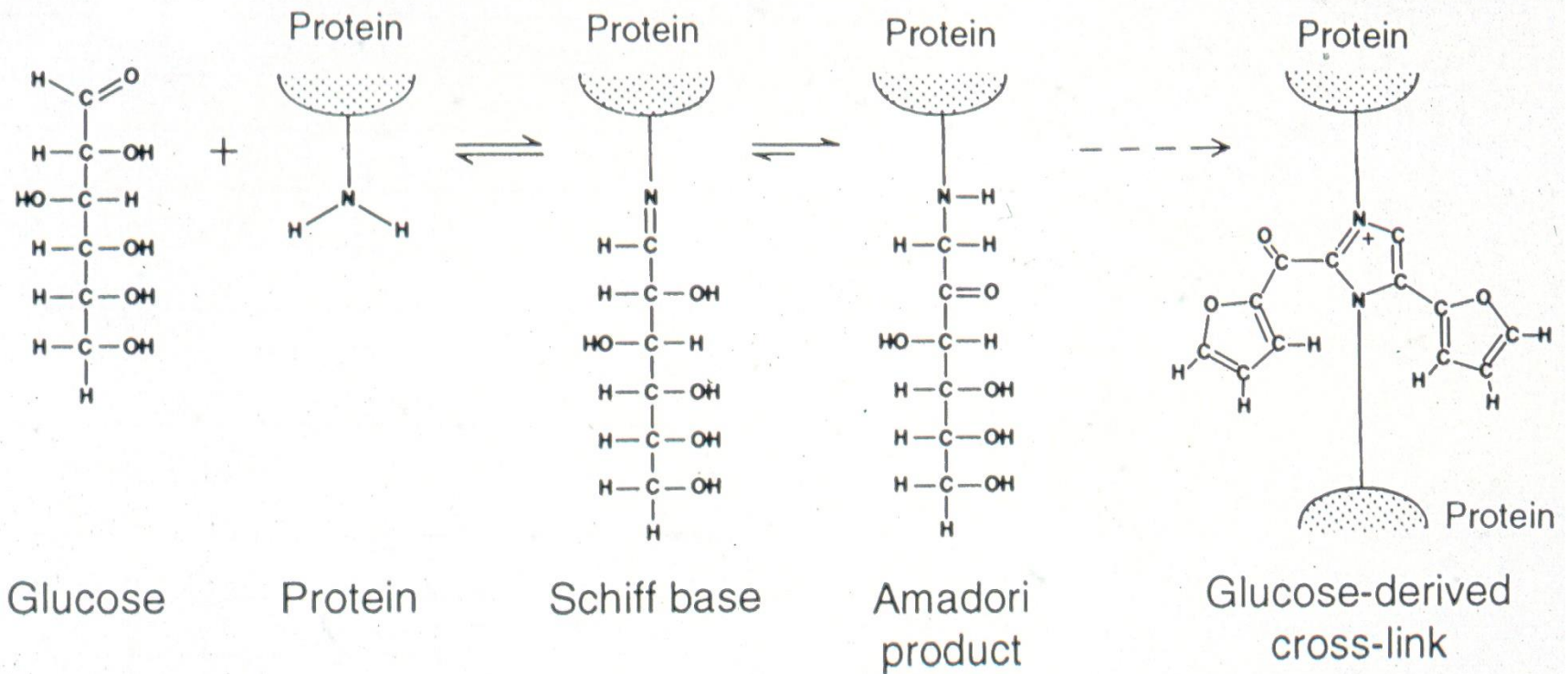
Μορφολογικά φυσιολογικό
νησίδιο Langerhans

Αμυλοείδωση σε νησίδιο
Langerhans

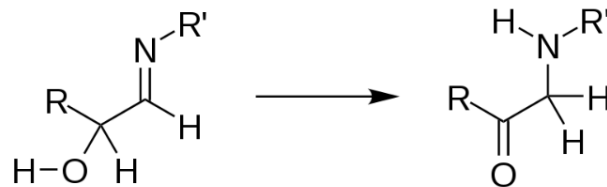


INSULIN DEFICIENCY

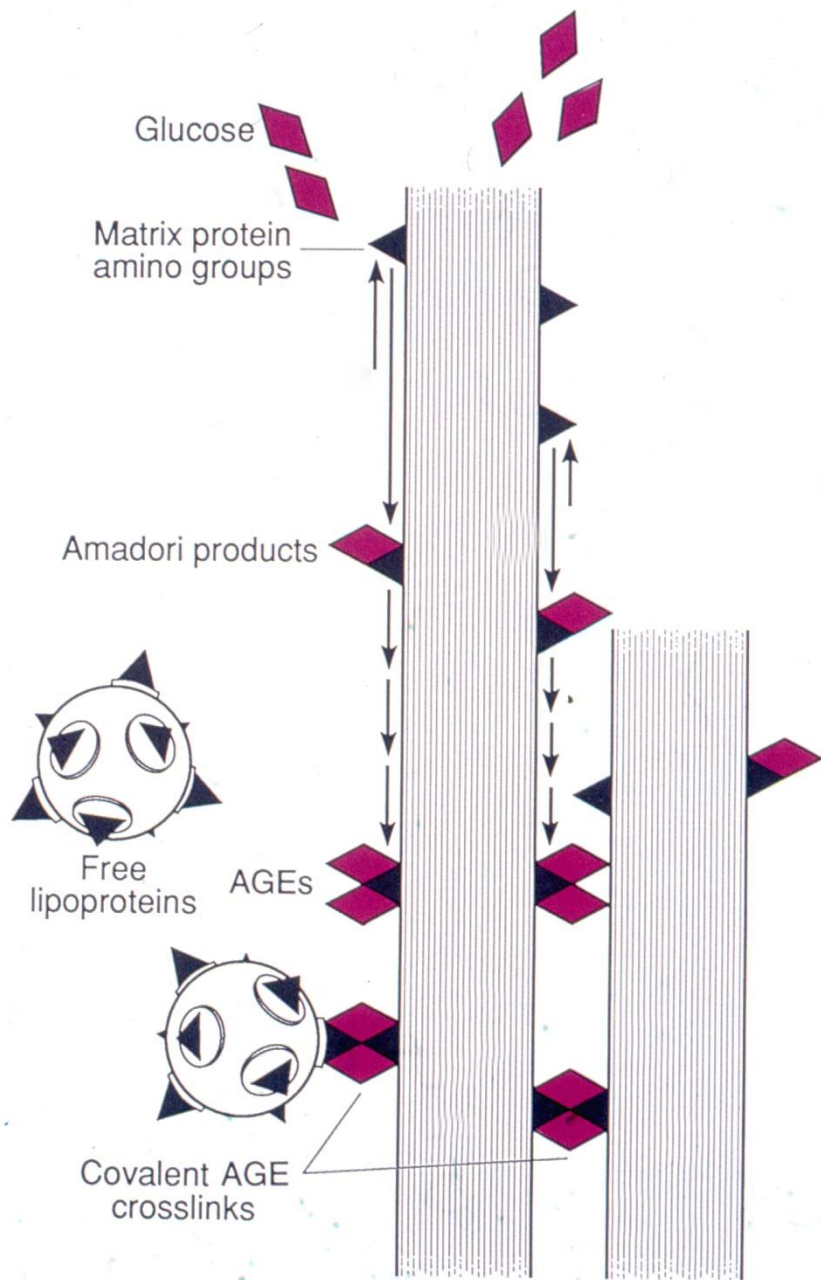


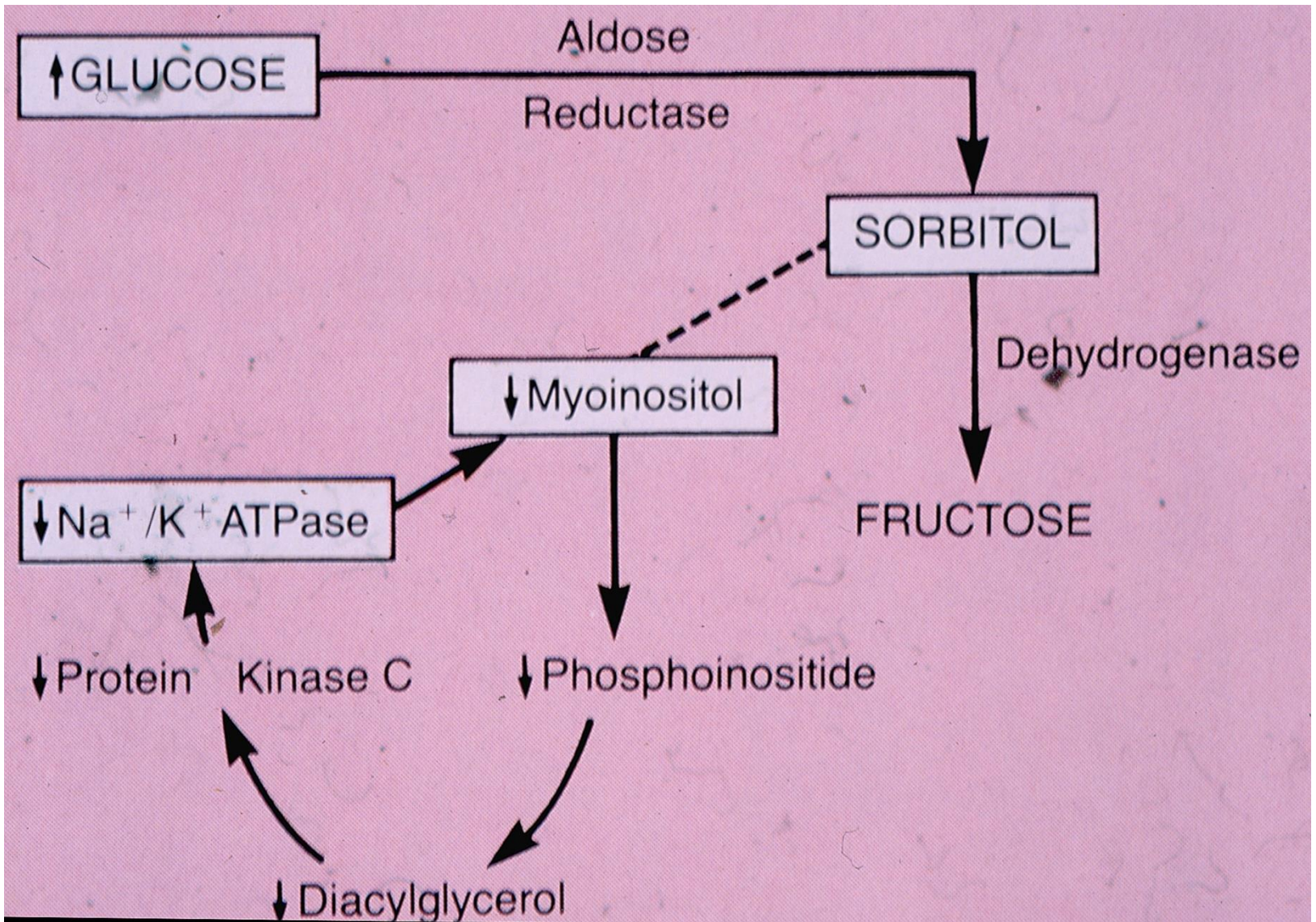


Amadori rearrangement

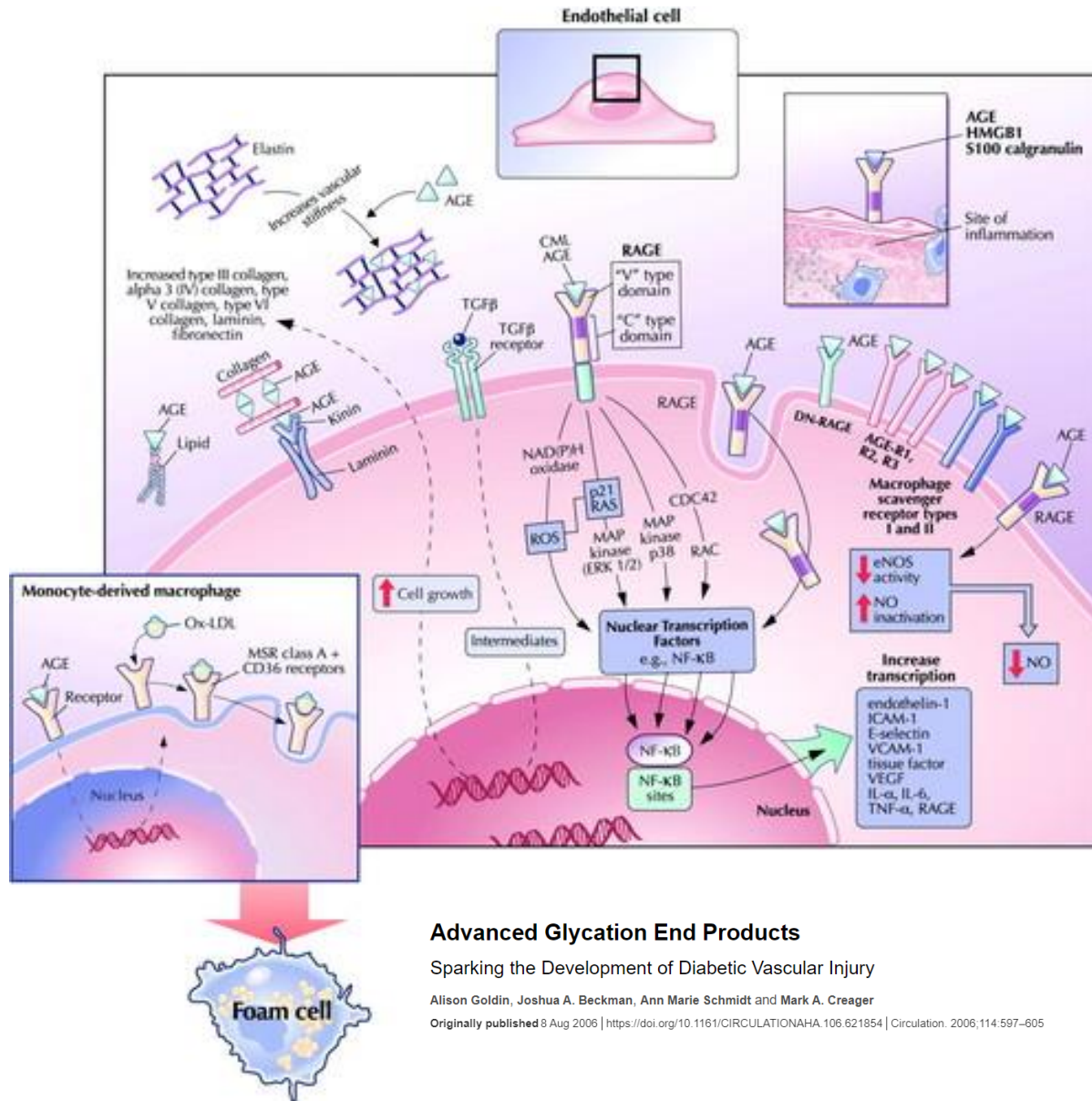


AGEs: advanced glycation end products:
μη ενζυματική τροποποίηση λιπιδίων ή πρωτεϊνών





AGEs – δέσμευση σε υποδοχείς στην επιφάνεια των ενδοθηλιακών κυττάρων

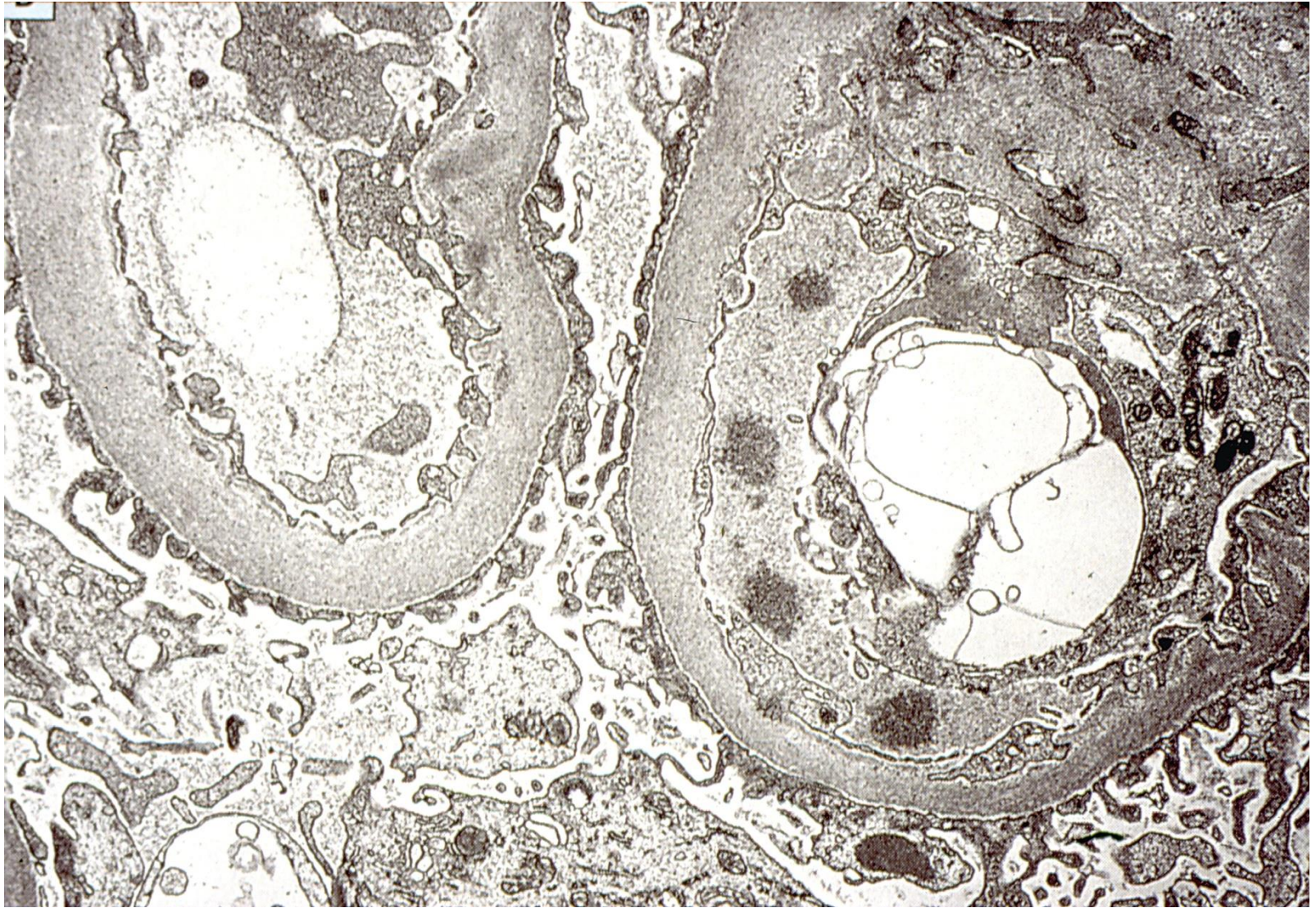


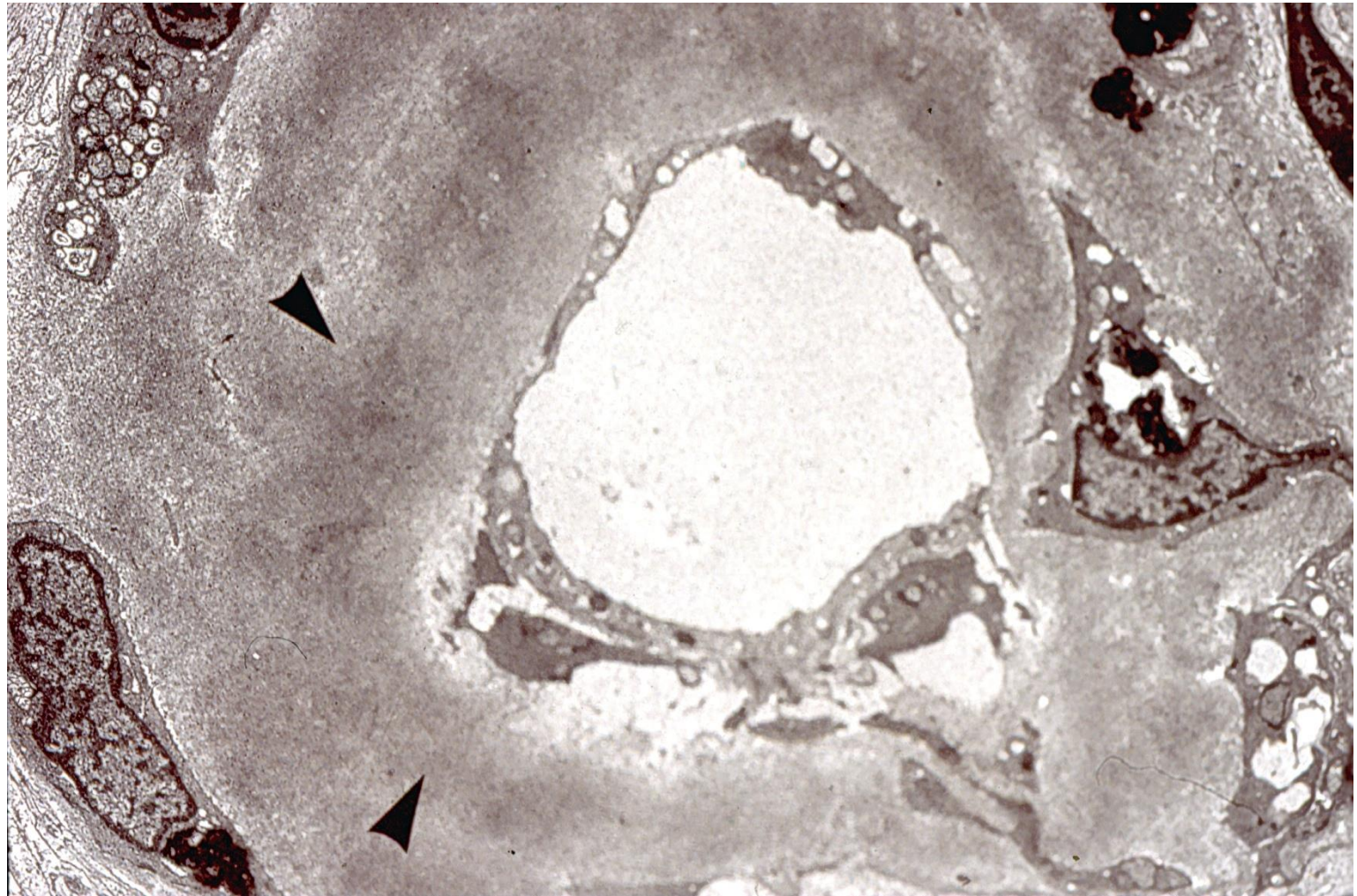
Advanced Glycation End Products

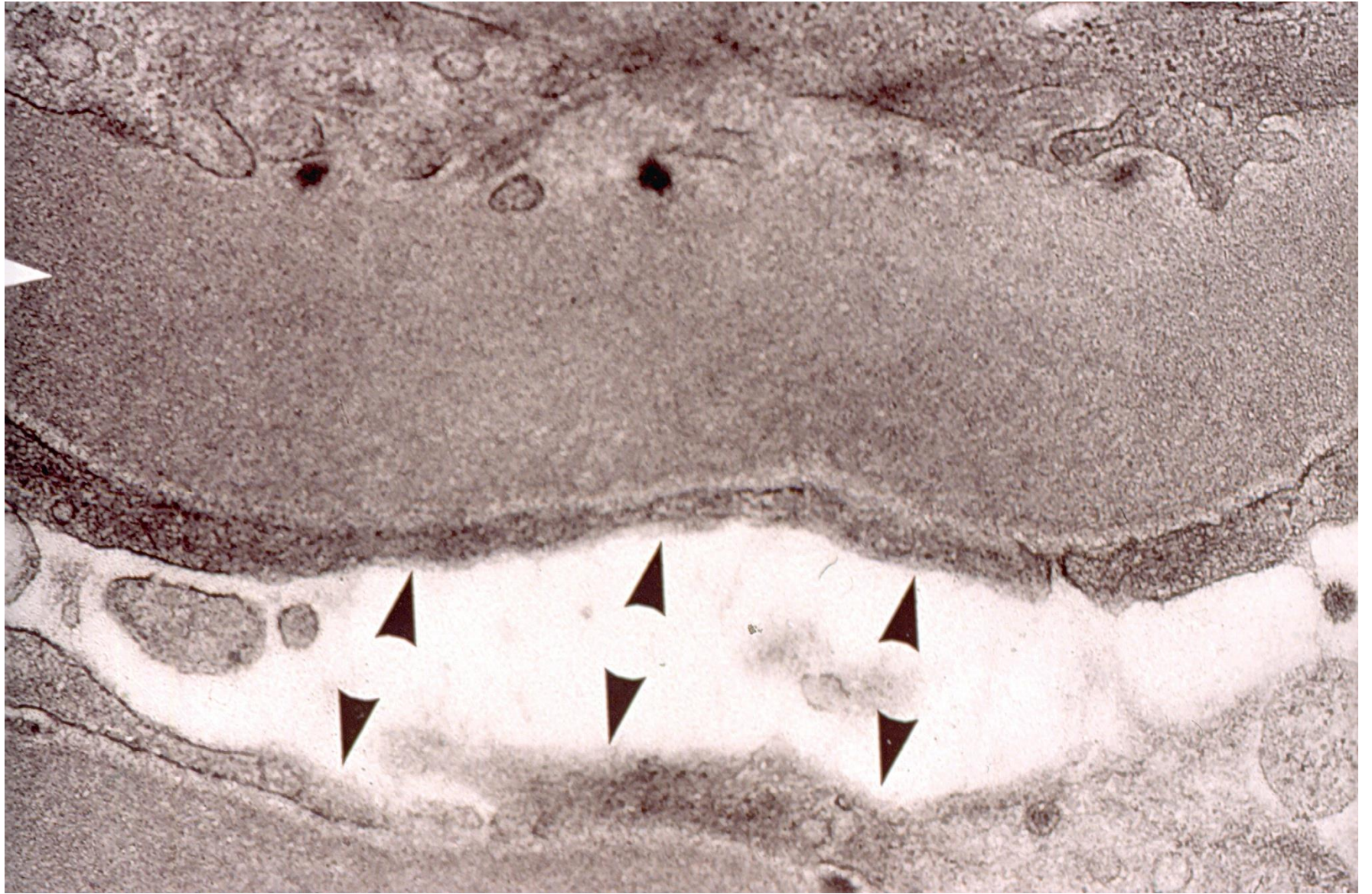
Sparking the Development of Diabetic Vascular Injury

Alison Goldin, Joshua A. Beckman, Ann Marie Schmidt and Mark A. Creager

Originally published 8 Aug 2006 | <https://doi.org/10.1161/CIRCULATIONAHA.106.621854> | Circulation. 2006;114:597-605







The burden and risks of emerging complications of diabetes mellitus

Dunya Tomic^{1,2}, Jonathan E. Shaw^{1,2,3} and Dianna J. Magliano^{1,2,3}✉

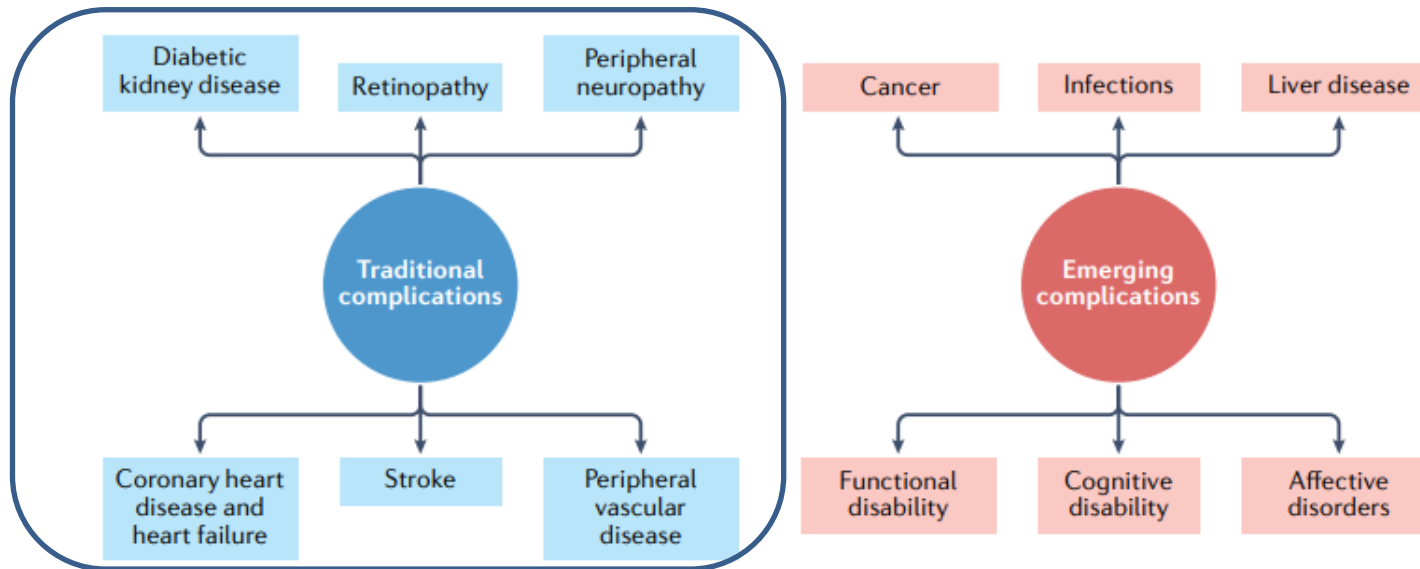
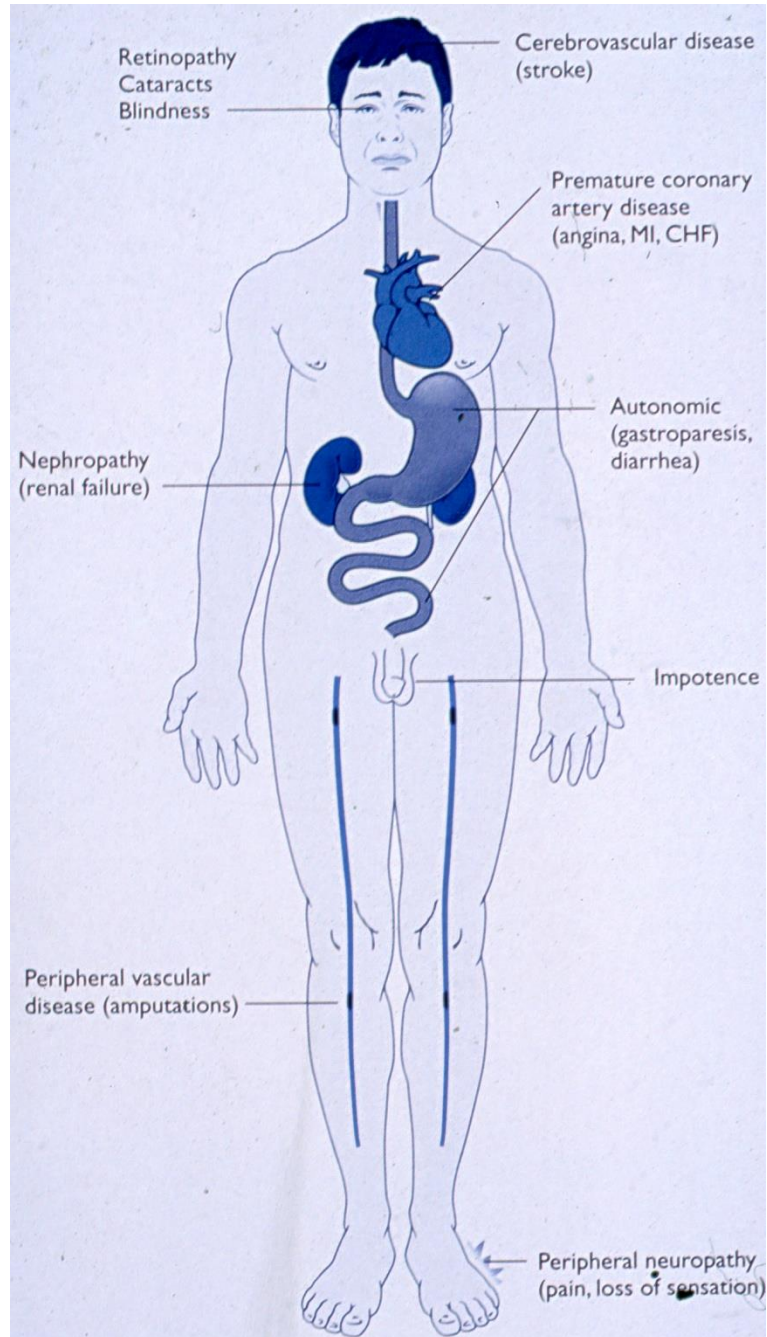
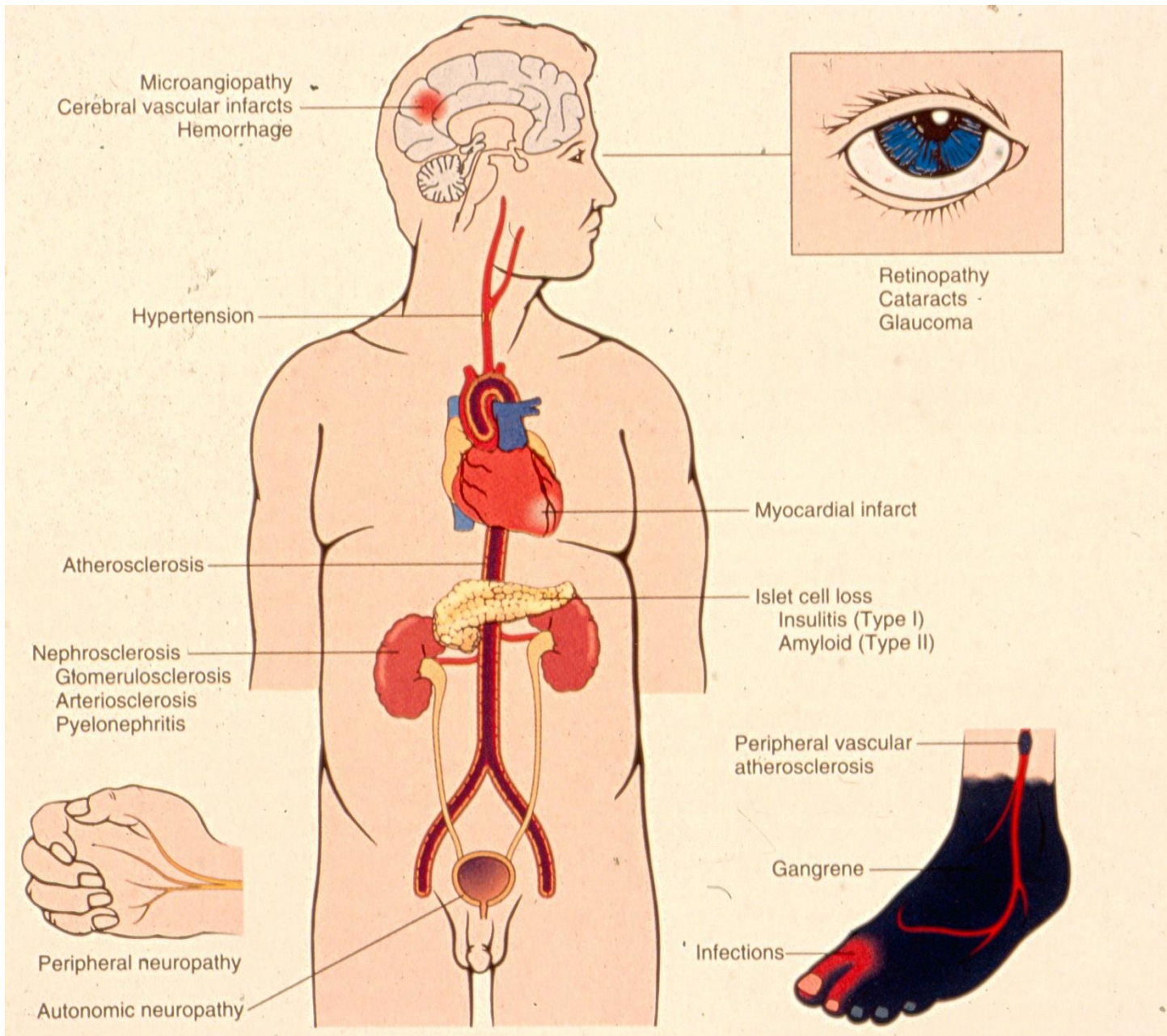


Fig. 1 | Major traditional complications and emerging complications of diabetes mellitus. The traditional complications of diabetes mellitus include stroke, coronary heart disease and heart failure, peripheral neuropathy, retinopathy, diabetic kidney disease and peripheral vascular disease, as represented on the left-hand side of the diagram. With advances in the management of diabetes mellitus, associations between diabetes mellitus and cancer, infections, functional and cognitive disability, liver disease and affective disorders are instead emerging, as depicted in the right-hand side of the diagram. This is not an exhaustive list of complications associated with diabetes mellitus.

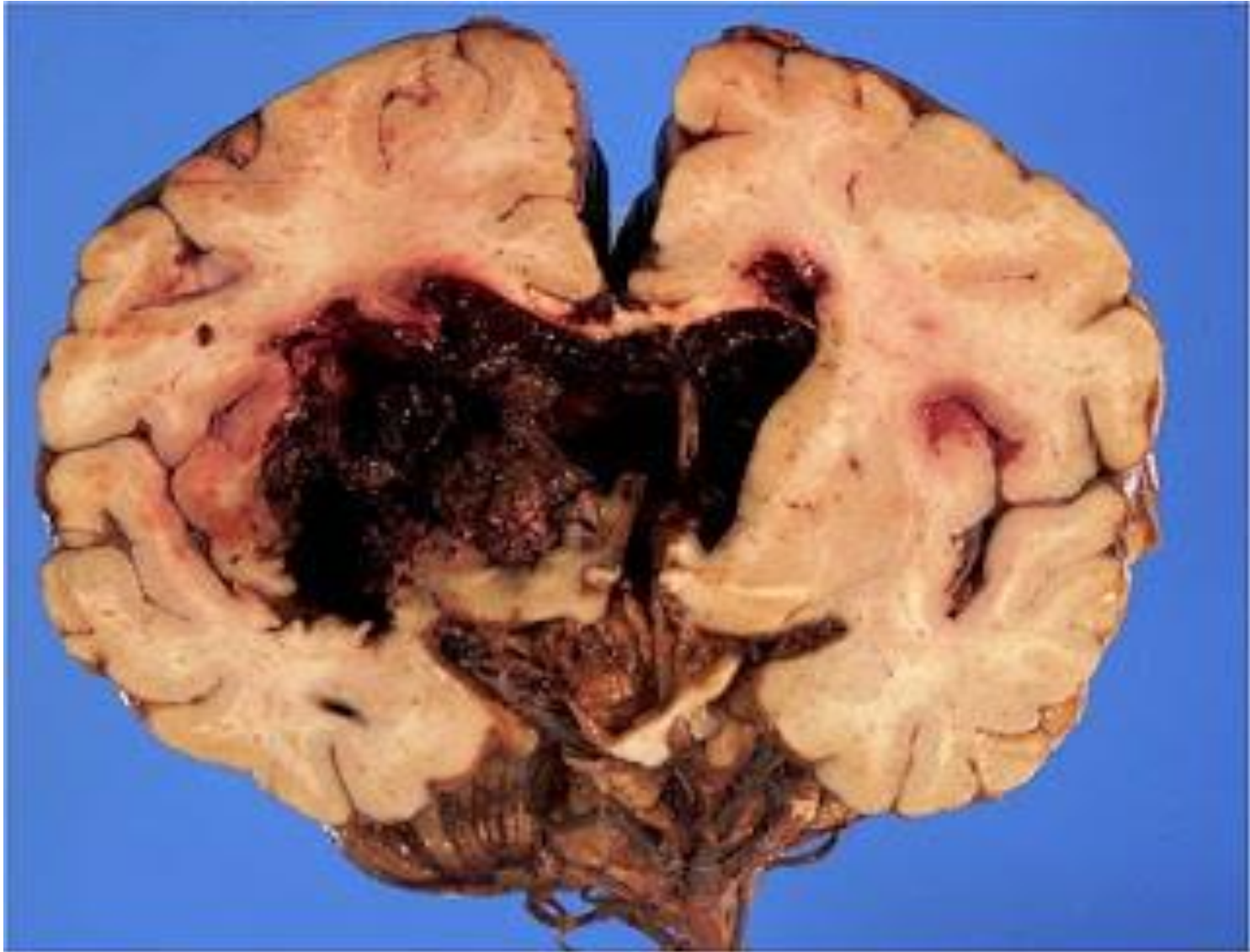




ΒΥΘΟΣ ΣΕ ΣΑΚΧΑΡΩΔΗ ΔΙΑΒΗΤΗ
(Διαβητική αμφιβληστροειδοπάθεια)



ΕΓΚΕΦΑΛΙΚΗ ΑΙΜΟΡΡΑΓΙΑ



ΔΟΘΙΗΝΑΣ

(μόλυνση θυλάκου τρίχας)



ΨΕΥΔΑΝΘΡΑΚΑΣ

(Δοθιήνες που συγκεντρώνονται μαζί)

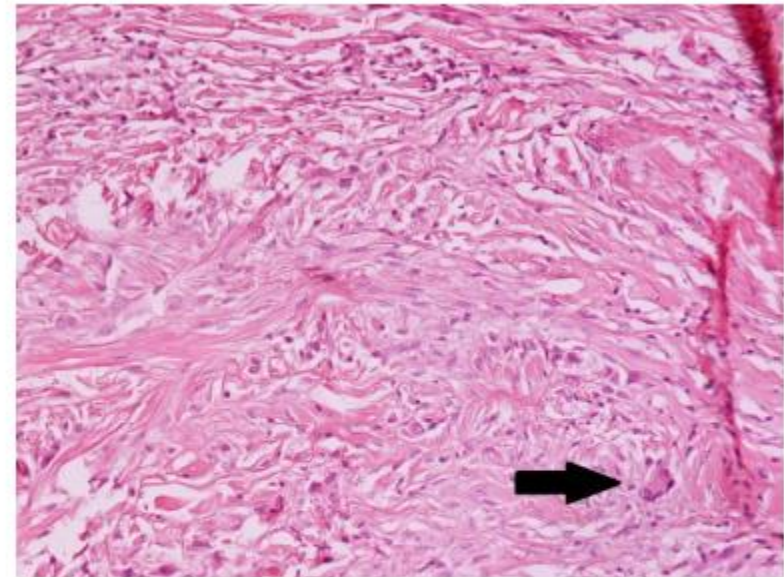


ΛΙΠΟΕΙΔΙΚΗ ΝΕΚΡΟΒΙΩΣΗ

σπάνια επιπλοκή (<0,3% των ασθενών με ΣΔ) εκφυλιστική διαταραχή του κολλαγόνου με παρουσία κοκκιωμάτων και εναπόθεση λίπους



Εικόνα 1. Δερματικές βλάβες ράχης

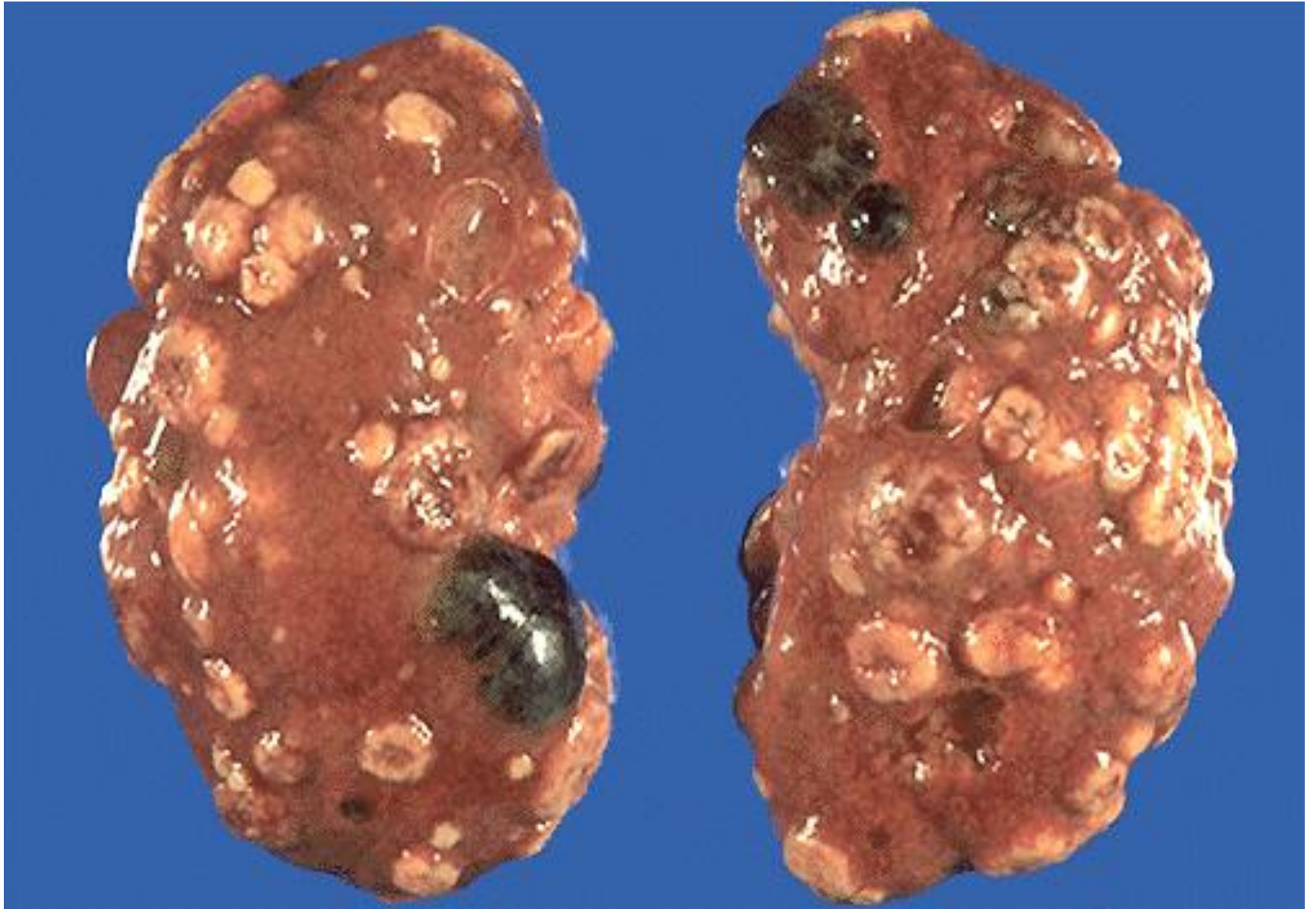


Εικόνα 5. Λιποειδική νεκροβίωση. Κοκκιωματώδης φλεγμονή - εκφυλισμένο κολλαγόνο, γιγαντοκύτταρο τύπου Langhans (βέλος), Η-Ε, x20

ΔΙΑΒΗΤΙΚΟ ΕΛΚΟΣ ΚΑΤΩ ΑΚΡΟΥ



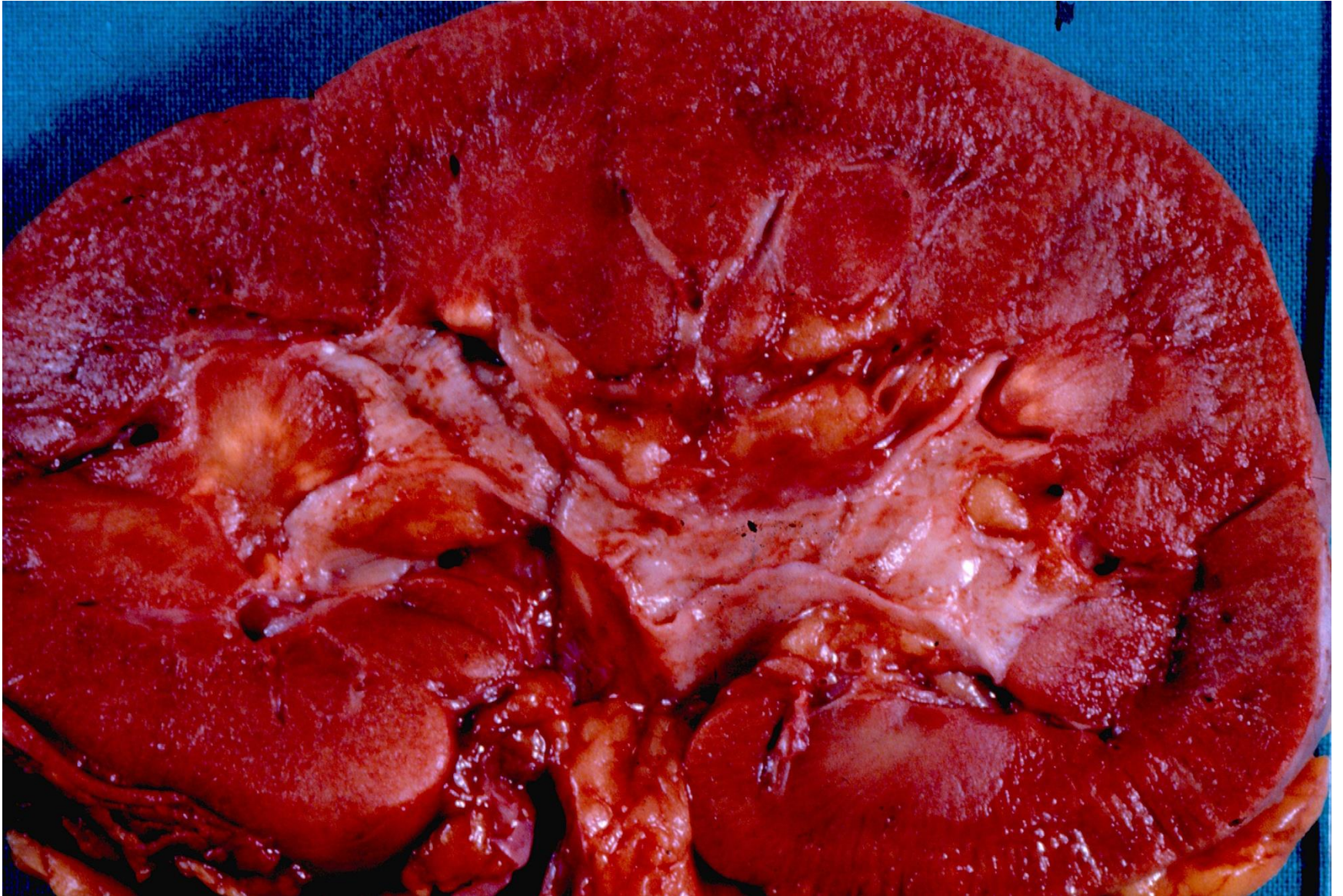
ΠΥΕΛΟΝΕΦΡΙΤΙΣ



ΕΜΦΡΑΚΤΟ ΝΕΦΡΟΥ



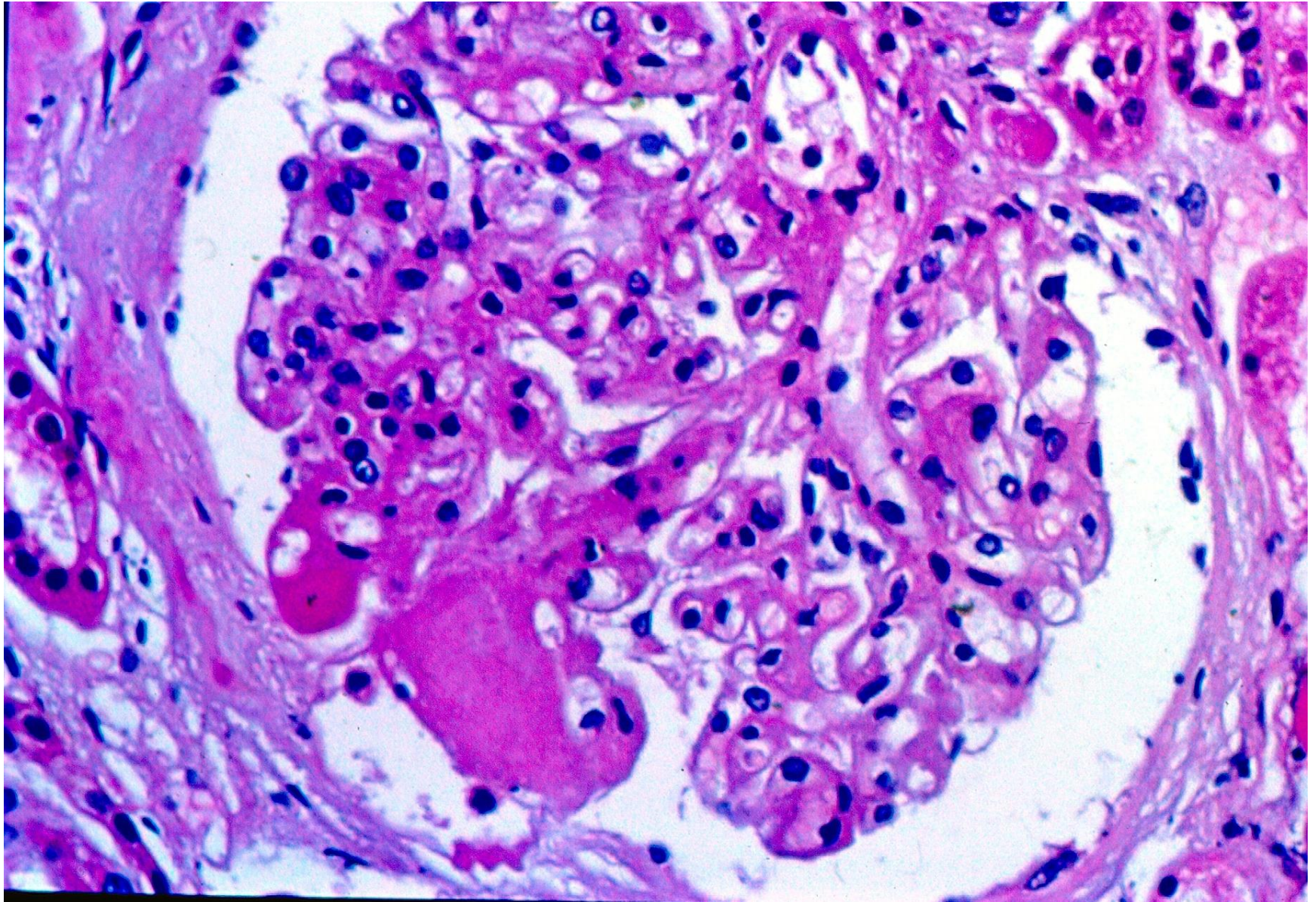
ΝΕΚΡΩΣΗ ΘΗΛΩΝ ΝΕΦΡΟΥ



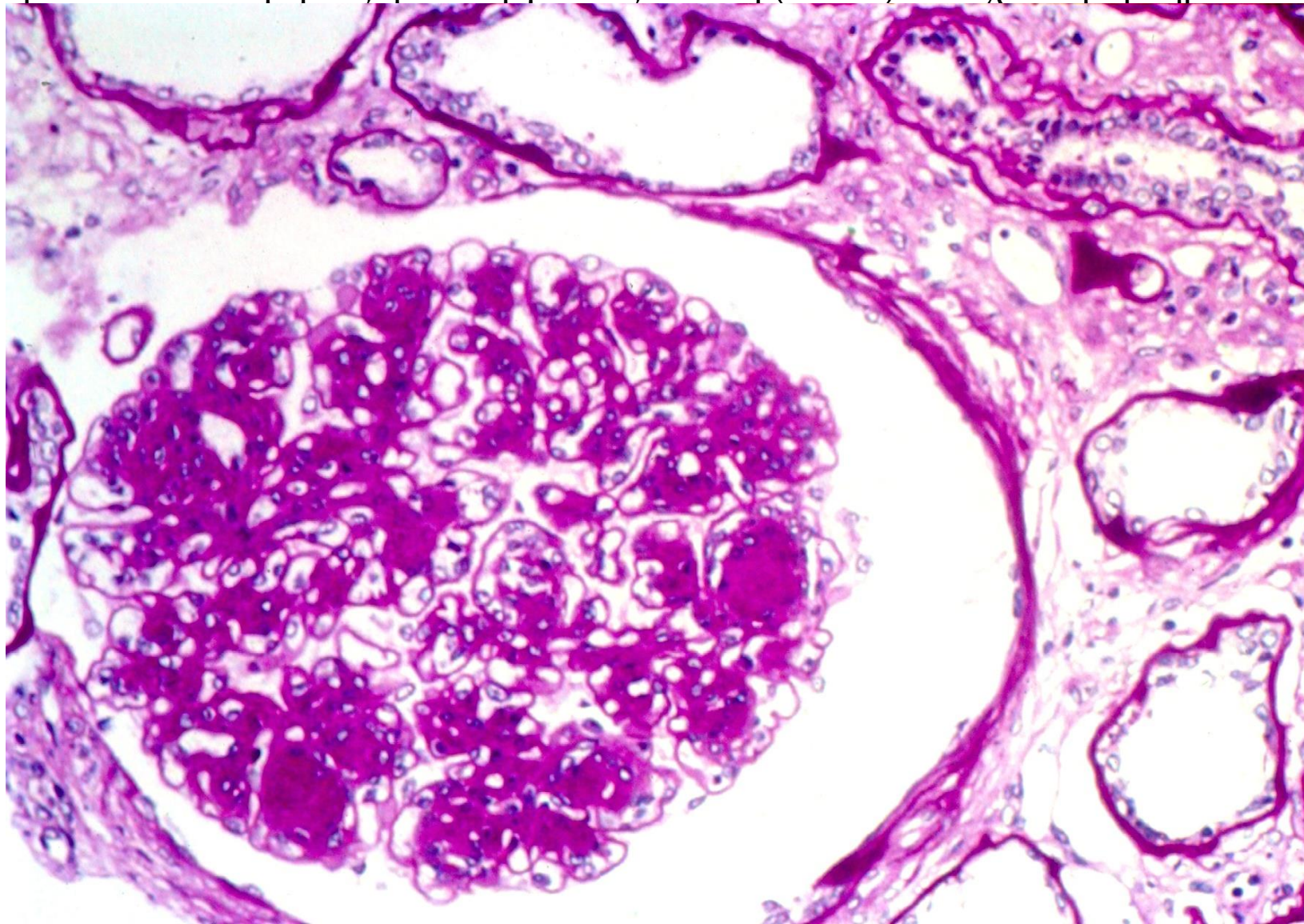
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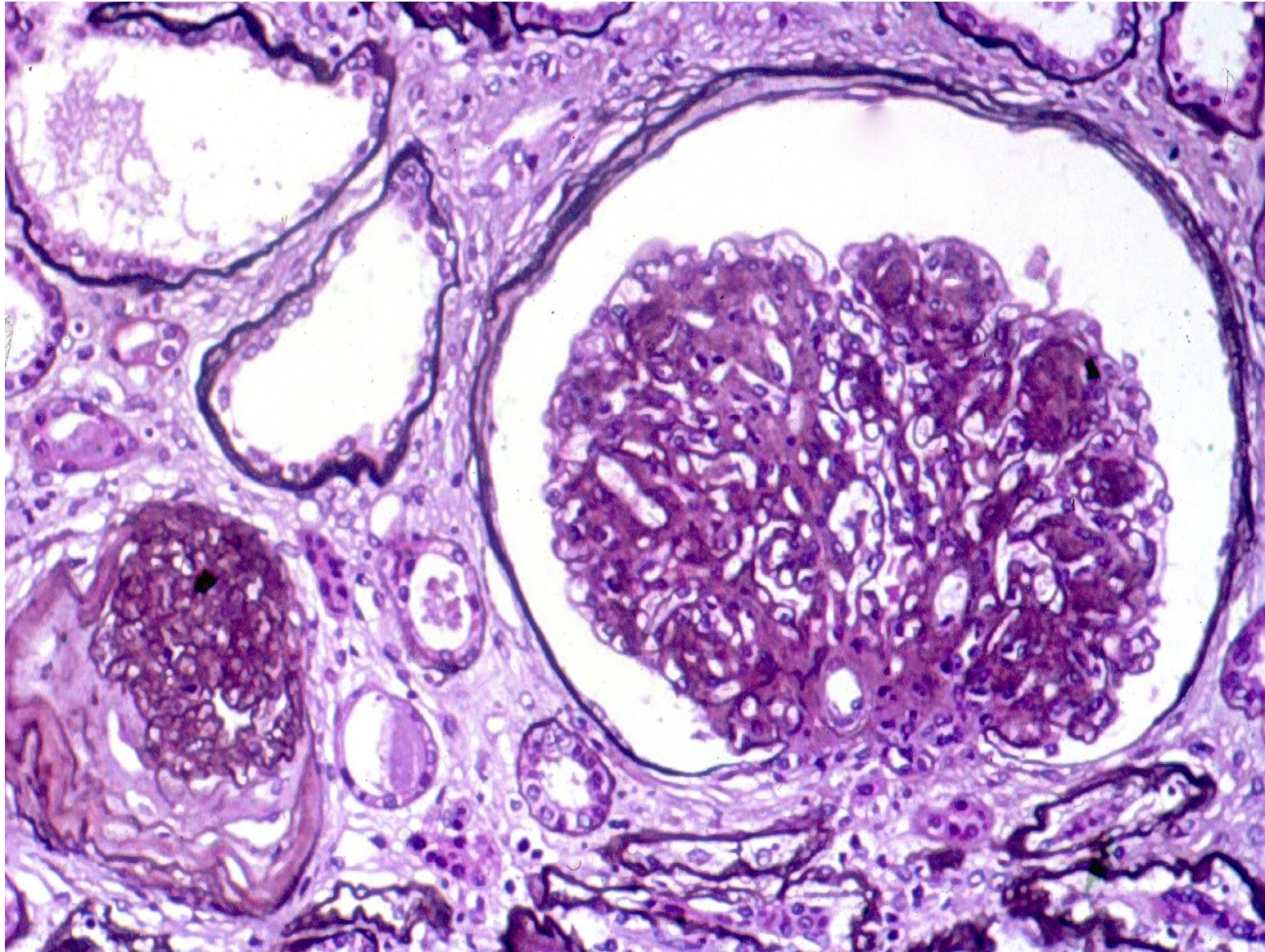
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(ΚΙΜΜΕΛΣΤΙΕΛ-ΥΙΛΣΟΝ)



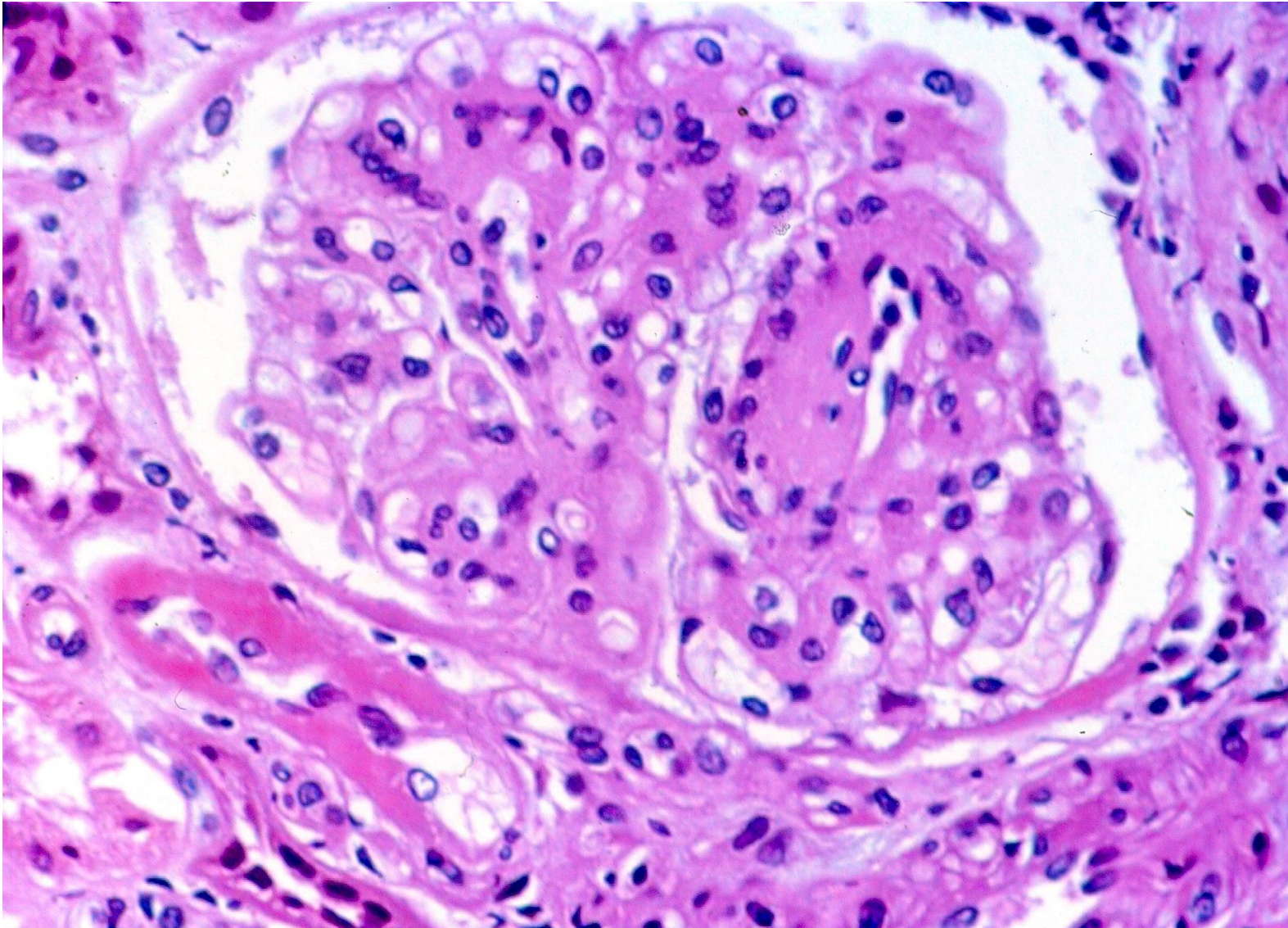
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(KIMMELSTIEL-WILSON) - εναπόθεση εξωκυττάριας ουσίας η οποία
προκύπτει από μη ενζυματική γλυκοζυλίωση (AGEs) - πάχυνση αρτηριολίων



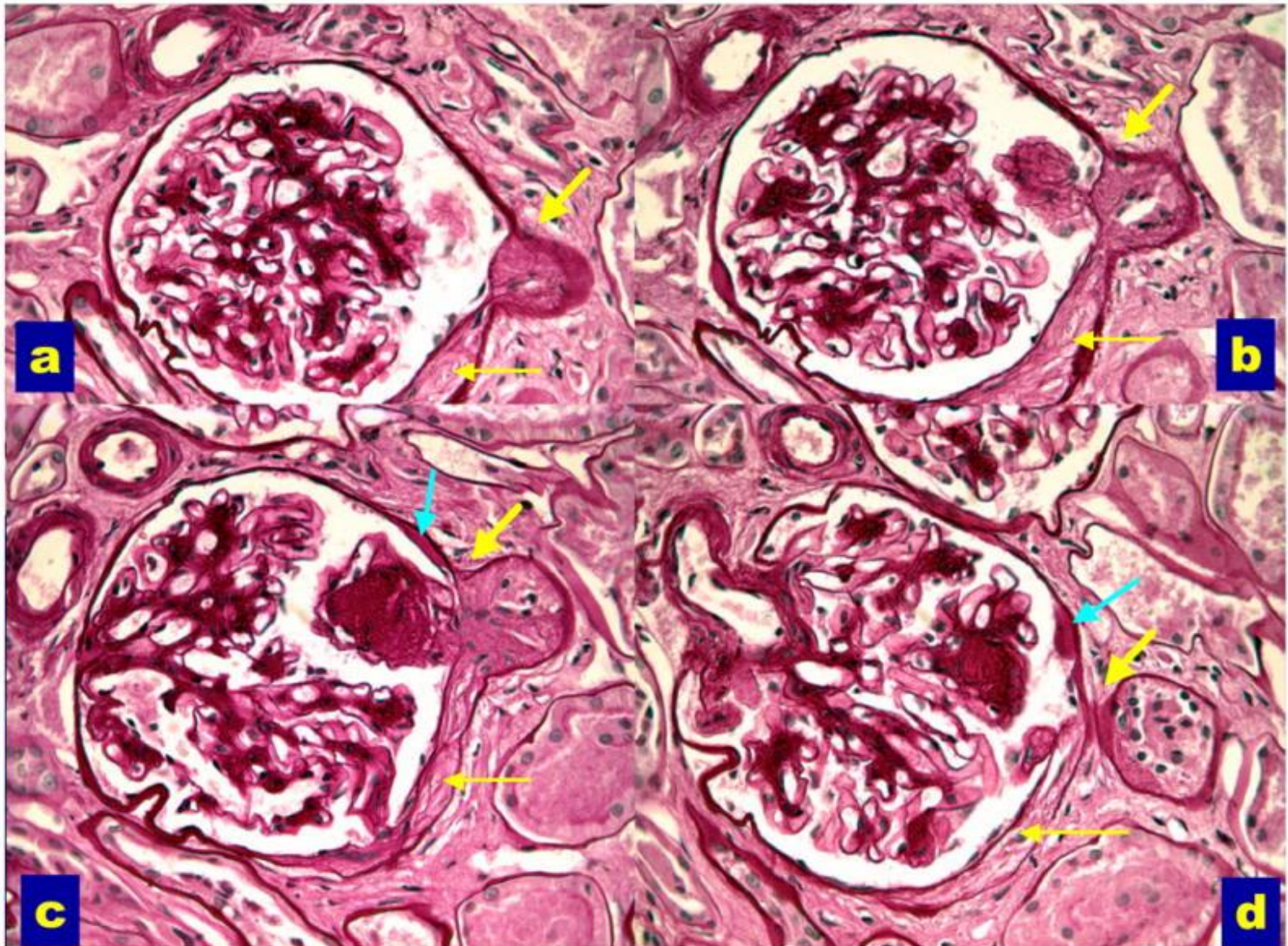
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(KIMMELSTIEL-WILSON) ΚΑΙ ΙΝΩΣΗ ΣΠΕΙΡΑΜΑΤΟΣ



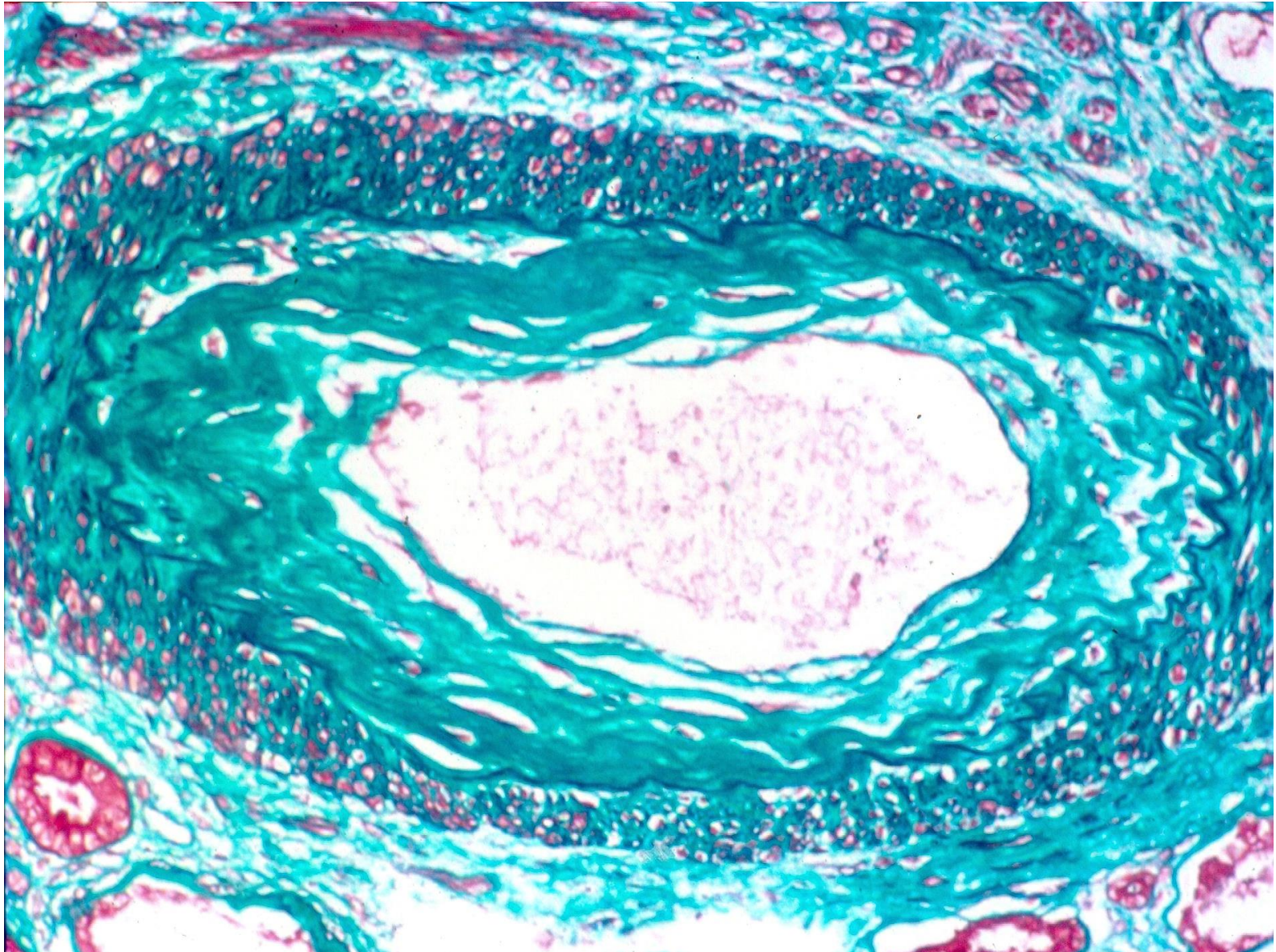
ΔΙΑΧΥΤΗ ΔΙΑΒΗΤΙΚΗ ΣΠΕΙΡΑΜΑΤΟΣΚΛΗΡΥΝΣΗ
ΚΑΙ ΑΡΤΗΡΙΔΙΟΣΚΛΗΡΥΝΣΗ



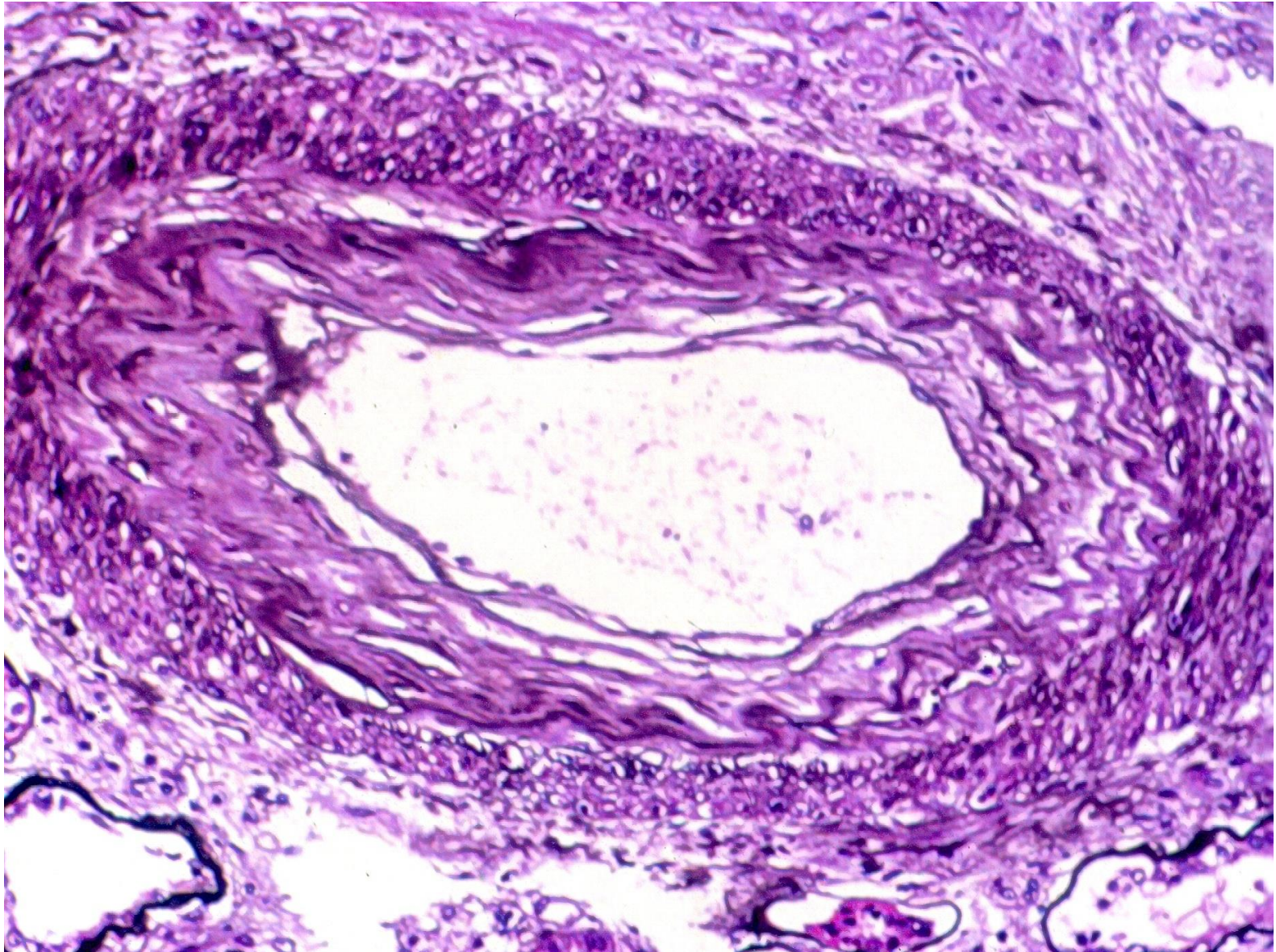
ΥΑΛΙΝΗ ΣΤΗΝ ΚΑΨΑ BOWMAN



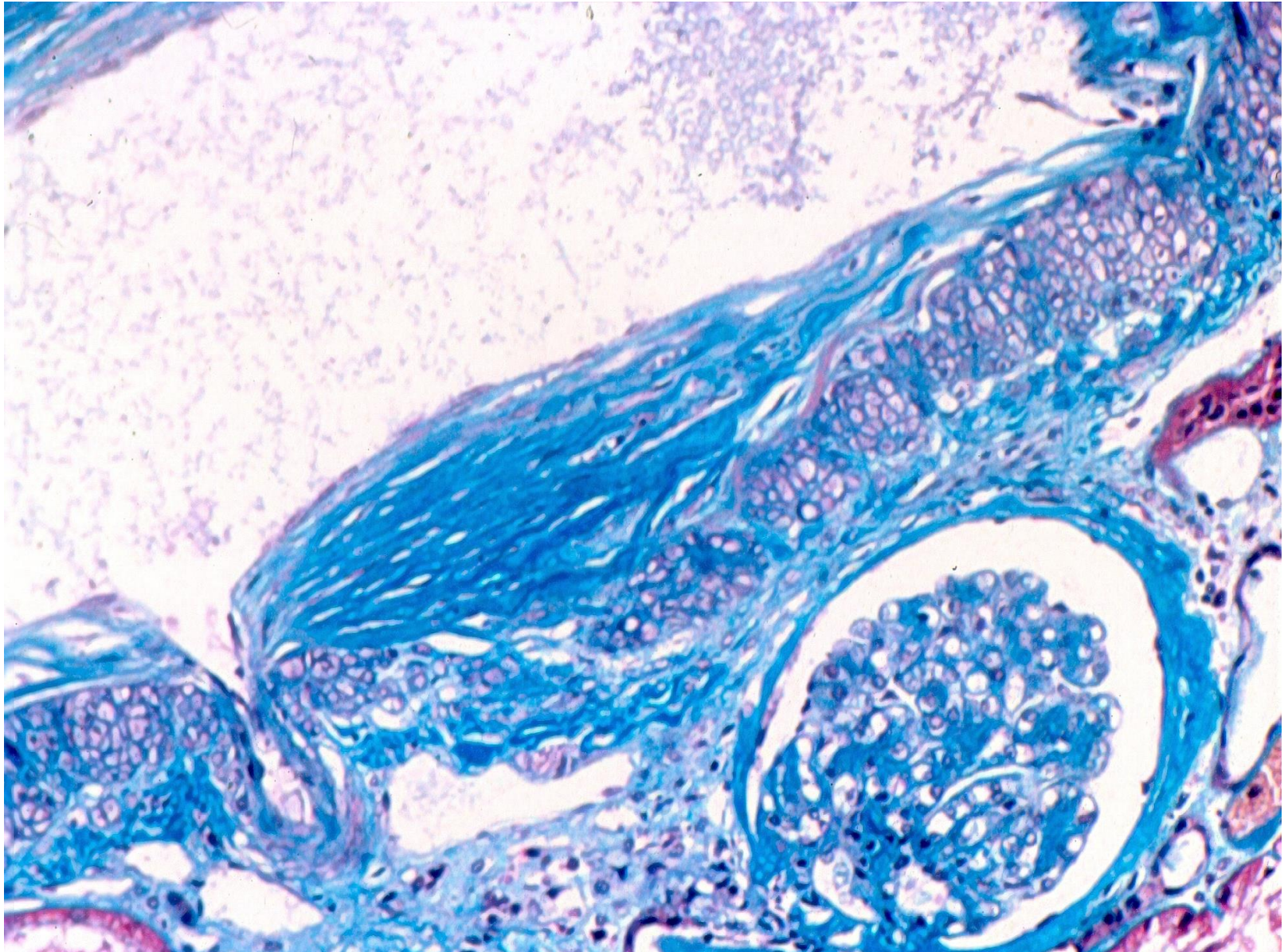
ΑΡΤΗΡΙΟΣΚΛΗΡΥΝΣΗ

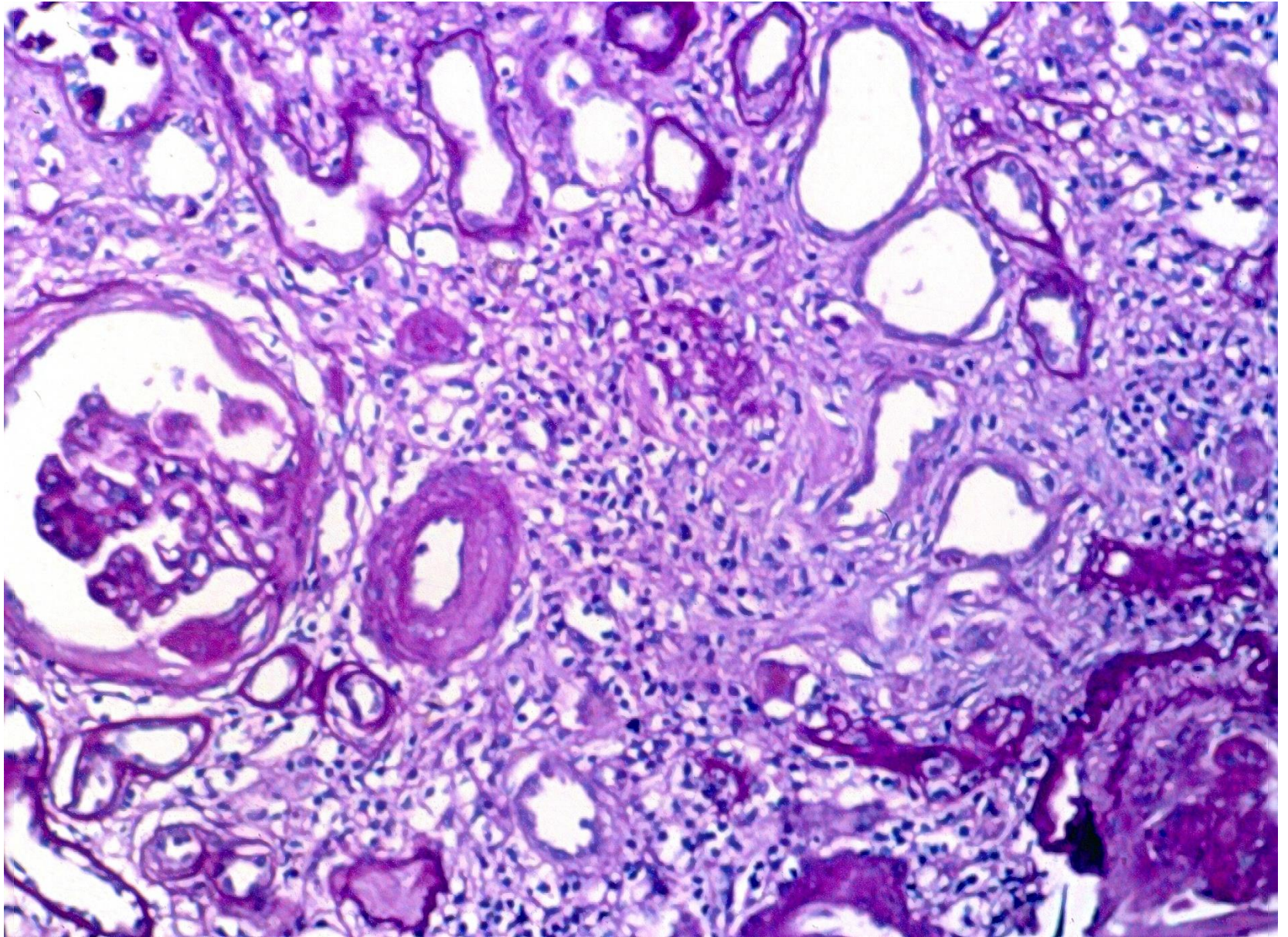


ΑΡΤΗΡΙΟΣΚΛΗΡΥΝΣΗ



ΑΡΤΗΡΙΟΣΚΛΗΡΥΝΣΗ





The burden and risks of emerging complications of diabetes mellitus

Dunya Tomic^{1,2}, Jonathan E. Shaw^{1,2,3} and Dianna J. Magliano^{1,2,3✉}

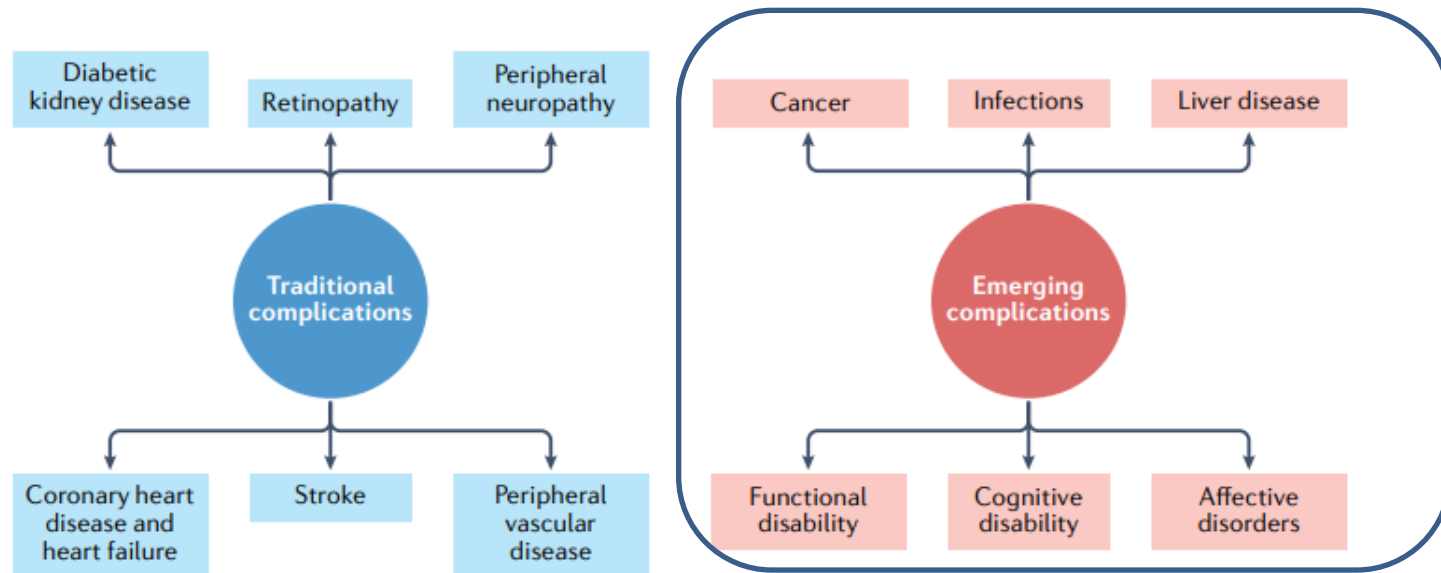


Fig. 1 | Major traditional complications and emerging complications of diabetes mellitus. The traditional complications of diabetes mellitus include stroke, coronary heart disease and heart failure, peripheral neuropathy, retinopathy, diabetic kidney disease and peripheral vascular disease, as represented on the left-hand side of the diagram. With advances in the management of diabetes mellitus, associations between diabetes mellitus and cancer, infections, functional and cognitive disability, liver disease and affective disorders are instead emerging, as depicted in the right-hand side of the diagram. This is not an exhaustive list of complications associated with diabetes mellitus.

Table 1 | Summary of major systematic reviews and original studies reporting a cancer risk associated with diabetes mellitus

Study	Diabetes mellitus type	Study type included (n)	Outcome	Risk associated with diabetes mellitus (95% confidence interval)
Wang et al. ^a (2012) ²⁴	All	Cohort (3,626,368 ^b)	Hepatocellular carcinoma	RR 2.01 (1.61–2.51)
El-Serag et al. ^a (2006) ²⁵	All	Cohort, cross-sectional (2,938,889 ^b)	Hepatocellular carcinoma	RR 2.5 (cohort studies) (1.9–3.2) and OR 2.5 (case–control) (1.8–3.5)
Huxley et al. ^a (2005) ²⁷	T2DM	Cohort, cross-sectional (9,220)	Pancreatic cancer	OR 1.82 (1.66–1.89)
	1–4 years duration		OR 2.05 (1.87–2.25)	
	5–9 years duration		OR 1.54 (1.31–1.81)	
	≥10 years duration		OR 1.51 (1.16–1.96)	
Carstensen et al. ^c (2016) ³⁰	T1DM	Cohort (9,149)	Pancreatic cancer	HR 1.53 (males) (1.30–1.79) and HR 1.25 (females) (1.02–1.53)
Jiang et al. ^a (2011) ³¹	All	Cohort (8,244,732 ^b)	Colorectal cancer	RR 1.27 (1.21–1.34)
Deng et al. ^a (2012) ³³	All	Cohort, cross-sectional (3,659,341)	Colorectal cancer	RR 1.26 (1.20–1.31)
De Bruijn et al. ^a (2013) ³²	All	Cohort, randomized controlled trials (1,930,309)	Colorectal cancer	HR 1.26 (1.14–1.40)
			Breast cancer	HR 1.23 (1.12–1.34)
Liao et al. ^a (2014) ³⁴	All	Cohort (5,302,259)	Endometrial cancer	RR 1.89 (1.46–2.45)
			Endometrial cancer disease-specific mortality	RR 1.32 (1.10–1.60)
Saed et al. ^a (2019) ³⁵	All	Cohort, cross-sectional (459,167 ^b)	Endometrial cancer	RR 1.72 (1.48–2.01)
Friberg et al. ^a (2007) ³⁶	All	Cohort, cross-sectional (96,003)	Endometrial cancer	RR 2.10 (1.75–2.53)
	T1DM		RR 3.15 (1.07–9.29)	
Larsson et al. ^a (2007) ³⁸	T2DM	Cohort, cross-sectional (1,430,122 ^b)	Breast cancer	RR 1.20 (1.12–1.28)
Anothaisintawee et al. ^a (2013) ³⁷	All	Cohort, cross-sectional (1,090,503 ^b)	Breast cancer	OR 1.14 (1.09–1.19)
Boyle et al. ^a (2012) ³⁹	All	Cohort, cross-sectional (21,029 ^b)	Breast cancer (postmenopausal)	RR 1.15 (1.07–1.24)
Zhang et al. ^a (2017) ⁴³	All	Cohort (2,392,245 ^b)	Ovarian cancer	RR 1.32 (1.14–1.52)
Weng et al. ^a (2017) ⁴⁴	All	Cohort (3,708,313)	Ovarian cancer	RR 1.19 (1.06–1.34)
Wang et al. ^a (2020) ⁴⁵	All	Cohort, cross-sectional (6,036,434 ^b)	Ovarian cancer	RR 1.20 (1.10–1.31)
Lee et al. ^a (2013) ⁴⁶	All	Cohort, cross-sectional (1,707,359 ^b)	Ovarian cancer	RR 1.17 (1.02–1.33)
Bonovas et al. ^a (2004) ⁴⁷	All	Cohort, cross-sectional (890,678 ^b)	Prostate cancer	RR 0.91 (0.86–0.96)
Long et al. ^a (2012) ⁴⁹	All (Asia only)	Cohort, cross-sectional (1,751,274)	Prostate cancer	RR 1.31 (1.12–1.54)

HR, hazard ratio; OR, odds ratio; RR, relative risk; T1DM, type 1 diabetes mellitus; T2DM, type 2 diabetes mellitus. ^aSystematic review. ^bTotal number of participants obtained through sum of individual study cohort sizes listed in tables or otherwise. ^cOriginal study.

Table 2 | Summary of major systematic reviews and original studies reporting an infection risk associated with diabetes mellitus

Study	Diabetes mellitus type	Study type included (n)	Outcome	Risk associated with diabetes mellitus (95% confidence interval)
Fang et al. ^a (2021) ⁷⁰	All	Cohort (12,379)	Infection-related hospitalization	HR 1.67 (1.52–1.83)
			Hospitalization for foot infections	HR 5.99 (4.38–8.19)
Luk et al. ^a (2021) ⁷²	All	Cohort (6,164,082)	Hospitalization for kidney infection (male individuals)	RR 2.50 (1.70–3.50)
			Hospitalization for kidney infection (female individuals)	RR 2.10 (1.70–2.70)
			Hospitalization for tuberculosis (male individuals)	RR 2.20 (2.00–2.40)
			Hospitalization for tuberculosis (female individuals)	RR 2.10 (1.80–2.40)
			Hospitalization for sepsis (male individuals)	RR 2.30 (2.10–2.50)
			Hospitalization for sepsis (female individuals)	RR 2.30 (2.10–2.50)
Magliano et al. ^b (2015) ⁷³	T1DM	Cohort (85,144)	Infection-related mortality	SMR 4.42 (3.68–5.34)
			Pneumonia-related mortality	SMR 6.23 (4.30–9.00)
			Septicaemia-related mortality	SMR 10.00 (6.70–14.90)
			Osteomyelitis-related mortality	SMR 16.30 (5.20–50.40)
Magliano et al. ^a (2015) ⁷³	T2DM	Cohort (1,023,838)	Infection-related mortality	SMR 1.47 (1.42–1.53)
			Pneumonia-related mortality	SMR 1.20 (1.20–1.30)
			Septicaemia-related mortality	SMR 1.80 (1.70–2.00)
			Osteomyelitis-related mortality	SMR 3.50 (2.90–4.30)
Martin et al. ^b (2016) ⁷⁴	All	RCTs, cohort, cross-sectional (32,067); 90 studies	Surgical site infection	OR 1.77 (adjusted measures; 1.13–2.78); heterogeneity (I ²) = 71%
McGumaghan et al. ^a (2021) ⁷⁰	All	Cohort (5,463,300)	Fatal or critical care unit-treated COVID-19	OR 1.40 (1.30–1.49)
			Rawshani et al. ^a (2021) ⁵⁰	T1DM
T2DM	Cohort (411,976)	HR 2.22 (2.13–2.32)		
You et al. ^a (2020) ⁵¹	T2DM	Cohort (5,473)	Intensive care unit-treated COVID-19	OR 1.59 (1.02–2.49)
Moon et al. ^a (2020) ⁵²	All	Cohort (5,307)	Oxygen treatment in COVID-19	OR 1.35 (1.10–1.66)
			Ventilator requirement in COVID-19	OR 1.93 (1.28–2.92)

COVID-19, coronavirus disease 2019; HR, hazard ratio; OR, odds ratio; RCT, randomized controlled trial; RR, relative risk; SMR, standardized mortality ratio. ^aOriginal study. ^bSystematic review.

Table 3 | Summary of original studies reporting risk of liver disease associated with diabetes mellitus

Study	Diabetes mellitus type	Study type included (n)	Outcome	Risk associated with diabetes mellitus (95% confidence interval)
Pang et al. (2018) ¹⁰⁷	All	Cohort (512,891)	NAFLD	HR 1.76 (1.47–2.16)
Li et al. (2017) ¹⁰⁸	T2DM	Cohort (18,111)	NAFLD	OR 1.40 (1.22–1.62)
Loomba et al. (2012) ¹¹³	All	Cross-sectional (1,069)	NASH	OR 1.93 (1.37–2.73)
			Liver fibrosis	OR 3.31 (2.26–4.85)

HR, hazard ratio; NAFLD, nonalcoholic fatty liver disease; NASH, nonalcoholic steatohepatitis; OR, odds ratio; T2DM, type 2 diabetes mellitus.

Table 4 | Summary of major systematic reviews reporting risk of affective disorders, cognitive disability and functional disability associated with diabetes mellitus

Author	Diabetes mellitus type	Study type included (n)	Outcome	Risk associated with diabetes mellitus (95% confidence interval)
Rotella et al. (2013) ¹²⁹	All	Cohort, cross-sectional (497,223)	Depression	HR 1.25 (1.10–1.44)
Nouwen et al. (2019) ¹⁹⁴	T2DM	Cohort, cross-sectional (48,808)	Depression	RR 1.24 (1.09–1.40)
Smith et al. (2013) ¹³²	All	Cohort, cross-sectional (12,626)	Anxiety disorders	OR 1.20 (1.10–1.31)
			Anxiety symptoms	OR 1.48 (1.02–1.93)
Lu et al. (2009) ¹⁵⁴	All	Cohort (23,257)	Vascular dementia	RR 2.38 (1.79–3.18)
			Alzheimer disease	RR 1.39 (1.16–1.66)
Cheng et al. (2012) ¹⁵⁵	All	Cohort (44,714)	Vascular dementia	RR 2.48 (2.08–2.96)
			Alzheimer disease	RR 1.46 (1.20–1.77)
			All-cause dementia	RR 1.51 (1.31–1.74)
			MCI	RR 1.21 (1.02–1.45)
Li et al. (2019) ¹⁵⁶	All	Cohort (1,257,144 ^a)	All-cause dementia	RR 1.69 (1.38–2.07)
Xue et al. (2019) ¹⁵⁷	All	Cohort, cross-sectional (4,349,111)	All-cause dementia	RR 1.43 (1.33–1.53)
Pal et al. (2018) ¹⁵⁸	T2DM	Cohort (6,865)	Progression to dementia in MCI	OR 1.53 (1.20–1.97)
Wong et al. (2013) ¹⁷³	All	Cohort, cross-sectional (162,534 ^a)	Mobility disability	OR 1.51 (1.38–1.64)
			ADL disability	OR 1.82 (1.40–2.36)
			IADL disability	OR 1.65 (1.55–1.74)
Yang et al. (2016) ¹⁷²	All age ≥60 years	Cohort (14,685)	Falls	RR 1.64 (1.27–2.11)

ADL, activities of daily living; HR, hazard ratio; IADL, independent activities of daily living; MCI, mild cognitive impairment; OR, odds ratio; RR, relative risk. ^aTotal number of participants obtained through sum of individual study cohort sizes listed in tables or otherwise.

Box 1 | **Diabetes mellitus and skeletal muscle atrophy**

- Individuals with diabetes mellitus exhibit skeletal muscle atrophy that is typically mild in middle age and becomes more substantial with increasing age.
- This muscle loss leads to reduced strength and functional capacity and, ultimately, increased mortality.
- Skeletal muscle atrophy results from a negative balance between the rate of synthesis and degradation of contractile proteins, which occurs in response to disuse, ageing and chronic diseases such as diabetes mellitus.
- Degradation of muscle proteins is more rapid in diabetes mellitus, and muscle protein synthesis has also been reported to be decreased.
- Proposed mechanisms underlying skeletal muscle atrophy include systemic inflammation (affecting both protein synthesis and degradation), dysregulation of muscle protein anabolism and lipotoxicity.
- Mouse models have also revealed a key role for the WWP1/KLF15 pathway, mediated by hyperglycaemia, in the pathogenesis of muscle atrophy.

See REFS^{195–198}.

Σας ευχαριστώ