



فصل پانزدهم - سلول درمانی در دیابت

سلول درمانی در بیماری دیابت

دیابت یک بیماری مزمن متابولیک است که با افزایش سطح گلوکز خون مشخص می‌شود و در طول زمان منجر به آسیب جدی به اندام‌های مختلف از جمله: قلب، چشم‌ها و کلیه‌ها می‌گردد. طبق گزارش فدراسیون بین‌المللی دیابت، در سال ۲۰۱۷، ۴۲۴/۹ میلیون بیمار دیابتی بین ۲۰ تا ۷۹ سال در سراسر جهان وجود داشته است و پیش‌بینی می‌شود این تعداد تا سال ۲۰۴۵ به ۶۲۸/۶ میلیون نفر برسد. به طور کلی براساس مطالعات، سه نوع اصلی دیابت وجود دارد: دیابت نوع ۱ (T1DM) که در آن تخریب سلول‌های β در پانکراس منجر به نقص تولید انسولین می‌شود، دیابت نوع ۲ (T2DM) که در آن مقاومت پیش‌رونده بدن به انسولین وجود دارد و در نهایت ممکن است به تخریب سلول‌های بتای پانکراس و نقص کامل تولید انسولین نیز منجر شود و دیابت حاملگی یا دیابت دوران بارداری. براساس نوع دیابت، تاکنون از شیوه‌های درمانی متفاوتی شامل: تزریق انسولین و یا استفاده از پمپ انسولین، عمل جراحی پیوند پانکراس و مصرف داروهای کاهنده گلوکز خون، در جهت بهبود علائم و کنترل بیماری استفاده شده است، اما هیچ کدام به طور قطعی تأثیرگذار نبوده‌اند و گاهی عوارض جانبی قابل توجهی نیز داشته‌اند. بنابراین، نیاز جدی به شناسایی و بهره‌مندی از روش‌های درمانی نوین با کارایی بالاتر برای بهبود بیماران دیابتی وجود دارد. در این راستا، محققان استفاده از سلول‌ها به ویژه، سلول‌های بنیادی را مورد بررسی و توجه قرار داده‌اند. مطالعاتی با پیشینه طولانی نشان داده‌اند سلول‌های بنیادی می‌توانند تقسیم‌های مکرر انجام داده و بدون تمایز بمانند. اما در صورتی که محرک خاص برای بیان ژن‌های کلیدی در محیط وجود داشته باشد، قادر به تمایز به انواع مختلف سلول‌های اختصاص یافته هستند. به این ترتیب، تاکنون تلاش‌هایی برای استفاده از سلول‌های بنیادی به منظور جایگزینی سلول‌های از کار افتاده در سطوح تحقیقات پیش‌بالینی و بالینی انجام شده است و در این زمینه از سلول‌های بنیادی مزانشیمی^۱ (MSCs) و خون‌ساز^۲ (HSCs) متفاوتی شامل سلول‌های بنیادی بالغ لوزالمعده، مغز استخوان، بند ناف، روده باریک، سلول‌های بنیادی جنینی و سلول‌های بنیادی پرتوان القایی استفاده شده است. در این میان مهمترین نوع سلول‌های بنیادی که امروزه بسیار مورد توجه هستند سلول‌های بنیادی مزانشیمی می‌باشند. به طور خلاصه، سلول‌های بنیادی مزانشیمی قادر به خودتجدیدی و تمایز به دودمان‌های مزانشیمی متعدد، مانند دودمان‌های چربی‌زا، غضروفی و استخوانی در شرایط آزمایشگاهی هستند. علاوه بر این، به دلیل بیان متوسط کمپلکس سازگاری بافتی اصلی (MHC) کلاس I، و همچنین عدم وجود MHC کلاس II و مولکول‌های تحریک‌کننده روی سطوح سلولی، ایمنی‌زایی پاییینی از خود نشان می‌دهند. از سوی دیگر، انبوهی از سیتوکین‌ها، فاکتورهای رشد و اگزوزوم‌های ترشح شده توسط سلول‌های بنیادی مزانشیمی نقش اساسی در تنظیم حساسیت به انسولین و اختلال عملکرد سلول‌های β دارند.

در دهه‌های گذشته، تعداد فزاینده‌ای از آزمایش‌های حیوانی و یافته‌های بالینی پیشنهاد کرده‌اند که مداخله درمانی مبتنی بر سلول‌های بنیادی به ویژه سلول‌های بنیادی مزانشیمی یک روش درمانی نوظهور و امیدوارکننده برای درمان دیابت است. برای مثال تعدادی از مطالعات نشان داده‌اند که سلول‌های بنیادی می‌توانند به سلول‌های تولیدکننده انسولین تمایز یابند و بدین ترتیب سبب ترمیم جزایر لانگرهانس تخریب شده در پانکراس مدل‌های حیوانی شوند. در سال ۲۰۱۸، مطالعه‌ای توانایی سلول‌های بنیادی مزانشیمی را در مهار فعال‌سازی مسیر آپوپتوز توسط استرس شبکه آندوپلاسمی در جزایر پانکراس پیوندی در موش را نشان داد. در همان سال Wang و همکاران نشان دادند که سلول‌های بنیادی مزانشیمی مغز استخوان^۳ (BM-MSCs) اتولوگ، FPG (گلوکز پلاسمای ناشتا) را کاهش داده و میزان موفقیت پیوند جزایر را در بیماران مبتلا به انواع خاصی از دیابت بهبود می‌بخشند.

^۱ Mesenchymal stem cells

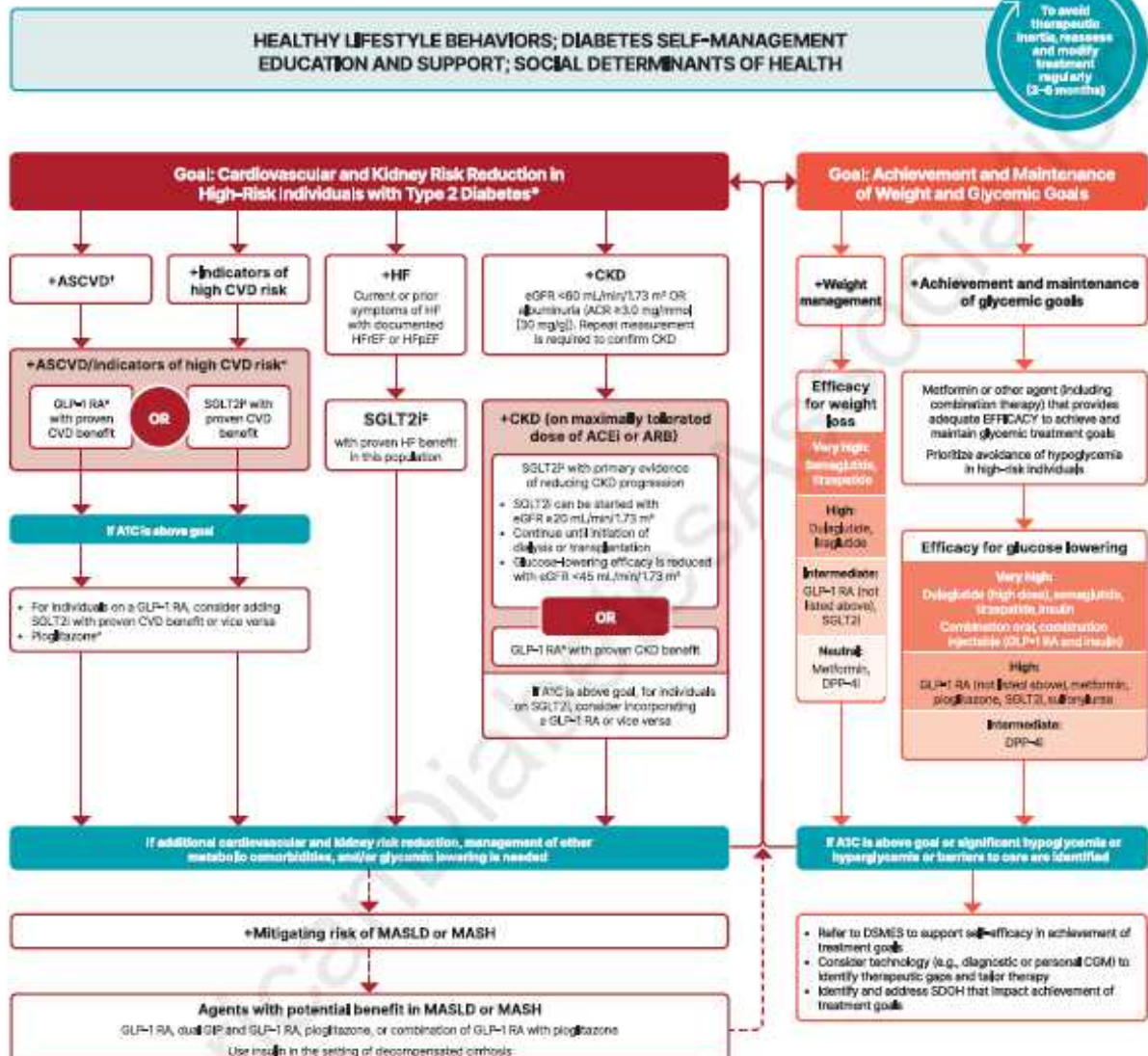
^۲ Hematopoietic Stem Cells

^۳ Bone-marrow mesenchymal cells

Voltarelli و همکاران از سلول‌های HSCs برای درمان بیماران مبتلا به دیابت نوع ۱ استفاده کرده و نتایج دلگرم‌کننده‌ای را گزارش نمودند. Bhansali و همکاران نشان دادند که سلول‌های بنیادی مشتق شده از مغز استخوان نیز یک درمان ایمن و مؤثر برای T2DM در بهبود عملکرد سلول‌های β هستند. El-Badawy و همکاران نیز دریافتند که درمان با سلول‌های بنیادی می‌تواند سطوح پپتید C را بهبود بخشد. علاوه بر این، آن‌ها تغییرات هموگلوبین گلیکوزیله (HbA1c) و پپتید C را مورد مطالعه قرار دادند، اما به‌طور سیستماتیک سطوح گلوکز پلاسمای ناشتا (FPG)، نیاز به انسولین، یا عوارض جانبی پس از درمان با سلول‌های بنیادی را تجزیه و تحلیل نکردند.

به‌طور کلی با وجود شواهد پیش‌بالینی و بالینی تأییدکننده اثرات مفید سلول‌های بنیادی در درمان بیماران دیابتی، همچنان نیاز جدی به مطالعات قوی‌تر برای مشخص نمودن بهترین نوع و دوز سلول مورد نیاز و روش استفاده از سلول مورد نظر برای اثر بخشی بیشتر وجود دارد.

Use of Glucose-Lowering Medications in the Management of Type 2 Diabetes



* In people with HF, CKD, established CVD, or multiple risk factors for CVD, the decision to use a GLP-1 RA or SGLT2i with proven benefit should be made irrespective of background use of metformin or A1C.

† ASCVD: Defined differently across CVOTs but all included individuals with established CVD (e.g., MI, stroke, and arterial revascularization procedure) and variably included conditions such as transient ischemic attack, unstable angina, angina, and symptomatic or asymptomatic coronary artery disease. Indicators of high risk: While definitions vary, most comprise ≥55 years of age with two or more additional risk factors (including obesity, hypertension, smoking, dyslipidemia, or albuminuria).

‡ A strong recommendation is warranted for people with CVD and a weaker recommendation for those with indicators of high-risk CVD. Moreover, a higher absolute risk reduction and thus lower numbers needed to treat are seen at higher levels of baseline risk and should be factored into the shared decision-making process. See text for details.

§ For GLP-1 RA, CVOTs demonstrate their efficacy in reducing composite MACE, CV death, all-cause mortality, MI, stroke, and kidney end points in individuals with T2D with established or high risk of CVD. One kidney outcome trial demonstrated benefit in reducing persistent eGFR reduction and CV death for a GLP-1 RA in individuals with CKD and T2D.

|| For SGLT2i, CV and kidney outcomes trials demonstrate their efficacy in reducing the risks of composite MACE, CV death, all-cause mortality, MI, HF, and kidney outcomes in individuals with T2D and established or high risk of CVD.

¶ Low-dose proglitazone may be better tolerated and similarly effective as higher doses.

Figure 9.3—Use of glucose-lowering medications in the management of type 2 diabetes. The left side of the algorithm prioritizes mitigation of diabetes-related complications and end-organ effects, while the right side addresses weight and glucose management goals. ACEi, angiotensin-converting enzyme inhibitor; ACR, albumin-to-creatinine ratio; ARB, angiotensin receptor blocker; ASCVD, atherosclerotic cardiovascular disease; CGM, continuous glucose monitoring; CKD, chronic kidney disease; CV, cardiovascular; CVD, cardiovascular disease; CVOT, cardiovascular outcomes trial; DPP-4i, dipeptidyl peptidase 4 inhibitor; DSMES, diabetes self-management education and support; eGFR, estimated glomerular filtration rate; GLP-1 RA, glucagon-like peptide 1 receptor agonist; HF, heart failure; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; HMF, hospitalization for heart failure; MACE, major adverse cardiovascular events; MASH, metabolic dysfunction–associated steatohepatitis; MASLD, metabolic dysfunction–associated steatotic liver disease; MI, myocardial infarction; SDOH, social determinants of health; SGLT2i, sodium–glucose cotransporter 2 inhibitor; T2D, type 2 diabetes. Adapted from Davies et al. [89].



فصل شانزدهم - خلاصه راهنمای بالینی دیابت

خلاصه راهنمای بالینی دیابت

مفاهیم کلیدی: اهداف باید به صورت فردی تعیین شود. گروه‌های خاص (کودکان، زنان باردار و افراد مسن) نیاز به ملاحظات ویژه دارند. در بیماران مبتلا به هیپوگلیسمی شدید یا مکرر می‌توان اهداف قند خون بالاتری در نظر گرفت. کنترل شدیدتر قند خون با کاهش بیشتر عوارض میکروواسکولار همراه است اگرچه خطر هیپوگلیسمی را افزایش می‌دهد. در صورتی که علیرغم رسیدن قند خون ناشتا به حد هدف HbA_{1c} به حد مطلوب نرسیده باشد، گام بعدی کنترل قند پس از غذاست.

غربالگری برای تشخیص دیابت

برای تشخیص دیابت و یا ارزیابی ریسک ابتلا به دیابت در آینده، اندازه‌گیری HbA_{1c}، گلوکز پلاسمای ناشتا (FPG) و آزمون تحمل گلوکز پس از مصرف ۷۵ گرم گلوکز مناسب است. سطوح HbA_{1c} بین ۵/۷٪-۶/۴٪ بیانگر افزایش خطر ابتلا به دیابت است. HbA_{1c} ≤ ۶/۵٪، غلظت گلوکز پلاسما در حالت ناشتا ≤ ۱۲۶ mg/dl و غلظت گلوکز پلاسما ۲ ساعت پس از مصرف ۷۵ گرم گلوکز خوراکی ≤ ۲۰۰ mg/dl معیارهای تشخیص دیابت به شمار می‌آیند.

مقادیر ارائه شده HbA_{1c} (جهت تشخیص و یا درمان) براساس روش‌های مورد تأیید NGSP می‌باشد. ولی با توجه به این که آزمایش HbA_{1c} در برخی مواقع با استفاده از کیت‌ها یا سیستم‌های فاقد این تأییدیه گزارش می‌شود، لازم است پزشک در همکاری با آزمایشگاه از دارا بودن این تأییدیه اطلاع حاصل نماید. در صورت عدم وجود شرایط مطلوب، پیشنهاد می‌گردد پزشک با در نظر گرفتن وضعیت بیمار و نیز میزان قند خون (ناشتا و بعد از غذا) جهت تشخیص و درمان بیمار تصمیم‌گیری نماید.

مراقبت بیماران مبتلا به دیابت نوع ۲	مراقبت بیماران مبتلا به دیابت نوع ۱	معاینه فیزیکی/ارزیابی آزمایشگاهی
برای طبقه‌بندی بیماران، عوارض را شناسایی کنید. برنامه‌ای برای مدیریت بیماری و ادامه مراقبت‌ها تدوین کنید.		معاینه فیزیکی کامل
هر سه ماه یک بار در موارد شدید بیماری، عدم پاسخ به درمان و عدم کنترل قند خون می‌توان تعداد ویزیت‌ها را افزایش داد.		ویزیت در مطب
هر ویزیت با هدف کاهش وزن به حد مطلوب		نمایه توده بدنی (اندازه‌گیری وزن در هر ویزیت، اندازه‌گیری قد سالی یک‌بار)
هر سه ماه یک بار (D)، در صورت رسیدن به هدف درمانی دو بار در سال (D). کنترل شدیدتر قند خون (HbA _{1c} < ۶٪) با کاهش بیشتر عوارض همراه است اگرچه خطر هیپوگلیسمی را افزایش می‌دهد و باید به طور فردی تصمیم‌گیری شود (B). در بزرگسالان مسن‌تر برای جلوگیری از حمله‌های هیپو/هیپرگلیسمی که منجر به عوارض حاد و مزمن می‌شوند می‌توان هدف HbA _{1c} را در حد ۸٪ تعیین نمود. در صورت وجود سابقه هیپوگلیسمی شدید، عوارض پیشرفته و امید به زندگی پایین نیز هدف درمانی HbA _{1c} < ۸٪ در نظر گرفته شود.		HbA _{1c} HbA _{1c} < ۷٪
برای فشار خون بالاتر از ۱۲۰/۸۰ تغییر شیوه زندگی با افزایش فعالیت بدنی، کاهش وزن و کاهش مصرف نمک توصیه می‌شود (A). برای افرادی که علیرغم تغییر شیوه زندگی فشار خون تأیید شده بالاتر یا مساوی ۱۳۰/۸۰ داشته باشند، توصیه به شروع درمان دارویی می‌شود (A). خط اول درمان دارویی ACEi یا ARB می‌باشد (A). اگر فشار خون با مصرف سه دسته دارویی (مشمول بر دیورتیک) به هدف درمانی نرسد، تجویز آنتاگونیست مینرالوکورتیکوئید باید مد نظر قرار گیرد (A).		فشارخون SBP < 130 mmHg DBP < 80 mmHg

اهداف درمانی در پروفایل چربی: LDL < 100 mg/dl LDL < 70 mg/dl در بیماران پرخطر (یا بیماری شناخته شده قلبی CVD) HDL > 40 mg/dl در مردان HDL > 50 mg/dl در زنان TG < 150 mg/dl اندازه‌گیری سالانه، در صورت عدم کنترل ارزیابی با فواصل کوتاه‌تر تا رسیدن به اهداف درمانی. در بیماران دیابتی بدون توجه به پروفایل لیپید در صورت وجود بیماری شناخته شده قلبی یا در افراد بالای ۴۰ سال و وجود یک یا چند فاکتور خطر بیماری‌های قلبی-عروقی درمان با استاتین توصیه می‌شود (A).	
در زمان تشخیص و سپس سالانه، در صورتی که در یک بازه زمانی ۶ ماهه دو نمونه از سه نمونه ادرار از نظر میکروآلبومینوری (A) مثبت باشد درمان با ACEI یا ARB توصیه می‌شود.	ارزیابی دفع آلبومین در ادرار با: نسبت میکروآلبومین ادرار به کراتینین (نمونه ادرار تصادفی روش ارجح است) یا در نمونه ادرار ۲۴ ساعته باید ۵ سال پس از تشخیص بیماری آغاز شود (D)، سپس سالانه تکرار گردد. در صورتی که در یک بازه زمانی ۶ ماهه دو نمونه از سه نمونه ادرار از نظر میکروآلبومینوری (A) مثبت باشد درمان با ACEI یا ARB توصیه می‌شود.
بررسی عملکرد کلیوی و کراتینین بیماران مبتلا به دیابت سالانه اندازه‌گیری شود. در صورت وجود بیماری مزمن کلیوی (CKD) مرحله‌بندی بیماری انجام شود (D).	
درمان با آسپیرین ۷۵-۱۶۲ میلی گرم در روز برای پیشگیری اولیه در بیماران مبتلا به دیابت نوع ۱ و ۲ که در معرض خطر بالای بیماری‌های قلبی-عروقی قرار دارند (به جز در موارد منع مصرف) ممکن است مد نظر قرار گیرد و برای پیشگیری ثانویه در همه بیماران دیابتی با سابقه بیماری‌های قلبی-عروقی توصیه می‌شود (A).	
در زمان تشخیص دیابت، در صورت طبیعی بودن معاینه چشم سالیانه.	معاینه چشم توسط افتالمولوژیست و یا اپتومتریست مجرب در تشخیص و مدیریت رتینوپاتی دیابتی در بیماران با سن ۱۰ سال و بیشتر، ۲-۳ سال پس از شروع بیماری سپس سالانه (B).
مشاهده پا در هر ویزیت. معاینه کامل سالانه شامل معاینه عروقی (بررسی نبض‌ها، حرارت، رنگ، جریان خون مویرگی، لنگش متناوب) معاینه نورولوژیک (حداقل دو مورد از موارد: تست مونوفیلان، آزمون تعیین آستانه درک ارتعاش، حس لامسه، رفلکس‌های تاندونی)، معاینه درماتولوژیک (مشاهده کلی پوست، ضایعات موضعی، کالوس‌های بین انگشتان، زخم و ناخن‌ها)، بررسی بیومکانیکال پا (بررسی دامنه حرکت مفاصل، شکل پا، انگشتان، برجستگی‌های استخوانی) و کفش بیمار (B).	
اندازه‌گیری گلوکز پلاسما در بیمارانی که تحت درمان با داروهای خوراکی بوده و یا نوبت‌های کمتری انسولین دریافت می‌کنند می‌تواند در هدایت درمان و خودمدیریتی بیماری کمک کننده باشد.	اهداف قند پلاسما در پایش فردی قند خون قند خون قبل از غذا ۸۰-۱۳۰ mg/dl قند خون بعد از غذا > ۱۸۰ mg/dl در بیمارانی که روزانه چند نوبت انسولین دریافت می‌کنند و یا تحت درمان با پمپ انسولین هستند اندازه‌گیری گلوکز پلاسما ۳ بار یا بیشتر در روز (A) شامل قبل از غذا، بعد از غذا، زمان خواب و قبل از شروع ورزش توصیه می‌شود.

در هر ویزیت بر کنترل قند و فشار خون بالا تأکید کنید، کاهش وزن با استفاده از رژیم غذایی مدیترانه‌ای، رژیم‌های کم چربی / با کالری محدود و یا کم کربوهیدرات برای همه افراد دارای اضافه وزن، افراد چاق در معرض خطر و یا بیماران مبتلا به دیابت توصیه می‌شود (A). الگوهای خوردن را با تأکید بر کربوهیدرات، سدیم، و چربی‌های اشباع شده بررسی کنید، شمارش کربوهیدرات یک استراتژی کلیدی در کنترل گلیسمیک است (A). حداقل ۱۵۰ دقیقه در هفته فعالیت بدنی هوازی با شدت متوسط (A)، در صورتی که منعی وجود نداشته باشد، افراد مبتلا به دیابت نوع ۲ باید به انجام تمرین‌های مقاومتی ۳ بار در هفته تشویق شوند (A).	مروری بر اهداف آموزش و خودمراقبتی
در فرد هوشیار درمان ارجح گلوکز (۱۵-۲۰ گرم) است، گرچه هر نوع کربوهیدرات حاوی گلوکز نیز مناسب است. پس از ۱۵ دقیقه قند خون بیمار را پایش کنید. در صورت ادامه هیپوگلیسمی درمان را تکرار کنید. پس از بازگشت SMBG به مقادیر طبیعی، برای پیشگیری از تکرار هیپوگلیسمی فرد باید یک وعده غذایی و یا یک میان وعده دریافت کند. برای کلیه افراد در معرض خطر هیپوگلیسمی شدید گلوکاگون ۱ mg SC/IM نسخه شود.	هیپوگلیسمی
بررسی بیماری‌های روان‌پزشکی (افسردگی)، توجه به علایم افسردگی و مشاوره با روان‌پزشک در صورت نیاز	غربالگری افسردگی
سالانه در همه بیماران توصیه می‌شود.	واکسیناسیون آنفلونزا
همه‌ی بیماران دیابتی با سن ۱۹ تا ۶۴ سال باید دو مرحله واکسن پنوموکوک مطابق با گایدلاین کشوری واکسیناسیون دریافت کنند. همه‌ی افراد دیابتی با سن بالای ۵۶ سال باید واکسن پنوموکوک دریافت کنند.	واکسیناسیون پنومونی
طبق برنامه واکسیناسیون هپاتیت B کشوری انجام شود.	واکسیناسیون هپاتیت B
توصیه به قطع مصرف دخانیات و ارجاع برای ترک آن به مراکز مربوطه	قطع سیگار
معاینه دهان و دندان هر ۶ ماه (D)	بهداشت دهان
مشاوره جهت تنظیم خانواده در خانم‌های در سن باروری، انجام تست‌های بررسی تشخیص دیابت ۱۲-۴ هفته بعد از ختم حاملگی در خانم‌هایی که مبتلا به دیابت بارداری در حاملگی اخیر بوده‌اند. غربالگری دیابت در آن‌ها باید هر ۳ سال یک بار انجام شود.	تنظیم خانواده
در دیابت نوع ۱ آنتی‌بادی‌ها (آنتی تیروئید پروکسیداز، آنتی تیروگلوبولین) و TSH همزمان با تشخیص و پس از آن به صورت دوره‌ای هر ۱ تا ۲ سال چک شود.	بررسی تیروئید
آزمایش‌های کبدی سالانه باید انجام شود.	انجام آزمایش‌های کبدی
بعد از تشخیص دیابت نوع ۱ در کودکان باید تست ترانس گلوتامیناز بافتی و یا آنتی‌اندومیزیال (EMA یا TTG) جهت غربالگری بیماری سلیاک انجام شود (این تست‌ها در نقص ایمنی IgA قابل اعتماد نیستند). توصیه به غربالگری در افراد بزرگسال مبتلا به دیابت نوع یک با توجه به علائم بالینی و شک به این بیماری	بیماری سلیاک



منابع

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4. Canadian Diabetes Association(CDA), <http://www.diabetes.ca>
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