2 高齢者糖尿病における生活指導のあり方

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加齢による糖尿病の増加と高齢化の進行に伴って，高齢の糖尿病患者が増加している．糖尿病は内服薬による薬物療法のみでなく，食事療法・運動療法などの非薬物療法や血糖自己測定，インスリンの自己注射などのセルフケアが重要となる病態であり，総合的な生活指導によるセルフケアの援助が重要である．また，高齢になると糖尿病以外にも多くの疾患を持つ者や，すでに多剤を併用している者が多くなるため，できるだけ薬物治療に頼りすぎないようなセルフケアを指導する必要がある．

一方で，高齢になると，長年慣れ親しんできた生活習慣の変更を受け入れにくくなりがちになり，セルフケアの指導に困難を伴うこともまれではない．また，糖尿病は認知症のリスク因子でもあり，高齢の糖尿病患者では認知機能障害を合併することも多いため，指導の内容や指導法に考慮が必要となったり，家族の支援を求めたりしなければならないことも多い．医療者と家族が協力しあって，高齢者のセルフケアを支えていく必要がある(図1)．

高齢者の生活指導における注意点

高齢者では，糖尿病患者に限らず，身体機能・認知機能・生活機能に個人差が大きいことが特徴である．また，家族背景・家族関係・経済状況などの個人を取り巻く状況にも個人差が非常に大きく，それぞれの事情に応じた個別の対応が求められる．こうした個人の状況を包括的に把握するためには，高齢者総合的機能評価(comprehensive geriatric assessment；CGA)が有用である．CGAは，高齢者を身体機能（基本的日常生活機能＝basic activities of daily living；BADL，手段的日常生活機能＝instrumental activities of daily living；IADL），精神心理面（認知機能，抑うつ度），社会的背景，などの面から包括的に評価して把握するものである(図2)．代表的な評価尺度として，BADLの評価であるBarthel index(表1)[1)]，IADLの評価であるLawton

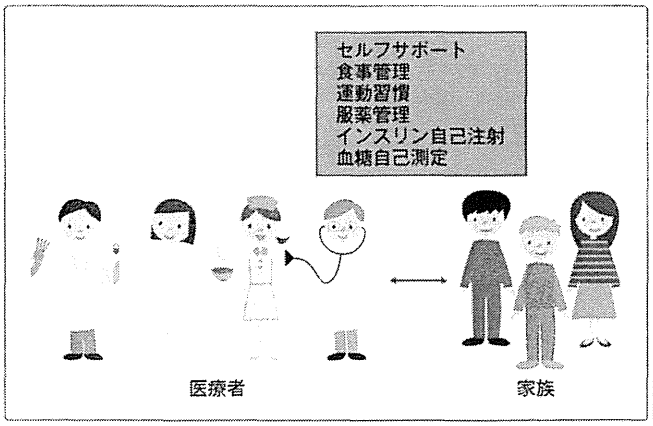

図1 セルフケアの援助
医療者と家族が協力しあい，高齢者のセルフケアを支える必要がある．

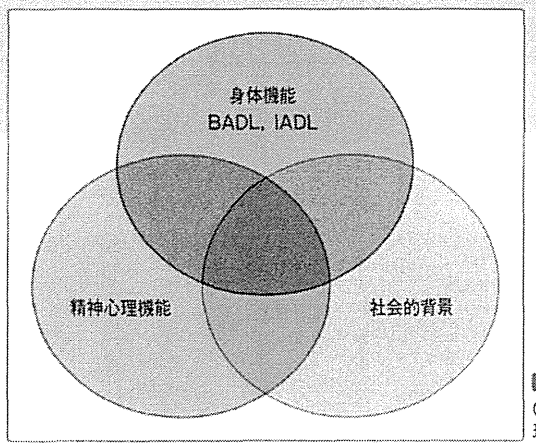

図2 CGAの概念
CGAとは，高齢者を身体機能・精神心理機能・社会的背景の面から総合的・包括的に評価し，理解するものである．

表1 BADLの評価(Barthel index)（文献1，飯島訳）

機能	内容	スコア
食事	自立．必要に応じて自助具を使用して，食物を切ったり，調味料をかけたりできる	10
	食物を切ってもらう必要があるなど，ある程度介助を要する	5
	上記以外	0
車いすとベッド間の移動*	移動のすべての段階が自立している(ブレーキやフットレストの操作を含む)	15
	移動の動作のいずれかの段階で，最小限の介助や，安全のための声かけ，監視を要する	10
	移動に多くの介助を要する	5
	上記以外	0
整容	手洗い，洗顔，髪梳き，歯磨き，ひげ剃りができる	5
	上記以外	0
用便動作	用便動作(便器への移動，衣服の始末，拭き取り，水洗操作)が介助なしにできる	10
	安定な姿勢保持や衣服の着脱，トイレットペーパーの使用などに介助を要する	5
	上記以外	0
入浴	すべての動作を他人の存在なしに遂行できる(浴槽使用でもシャワーでもよい)	5
	上記以外	0
平地歩行	少なくとも45 m，介助や監視なしに歩ける(補助具や杖の使用は可．車輪つき歩行器は不可)	15
	最小限の介助や監視下で少なくとも45 m歩ける	10
	歩行不可能だが，自力で車いすを駆動して少なくとも45 m進める	5
	上記以外	0
階段昇降	1階分の階段を介助や監視なしに安全に昇り降りできる(手すりや杖の使用は可)	10
	介助や監視を要する	5
	上記以外	0
更衣	すべての衣服（靴の紐結びやファスナーの上げ下ろしも含む)の着脱ができる（治療用の補装具の着脱も含む）	10
	介助を要するが，少なくとも半分以上は自分で，標準的な時間内にできる	5
	上記以外	0
排便コントロール	随意的に排便でき，失敗することはない．坐薬の使用や浣腸も自分でできる	10
	ときに失敗する，もしくは坐薬の使用や浣腸は介助を要する	5
	上記以外	0
排尿コントロール	随意的に排尿できる．必要な場合は尿器も使える	10
	ときに失敗する．もしくは尿器の使用などに介助を要する	5
	上記以外	0

*車いすを使用していない場合には，ベッド脇に設置した肘かけいすとベッドとの間の移動が安全にできるかどうかを評価する．

表2 IADLの評価(Lawton index)（文献3）

	項目	得点
A. 電話の使い方	1. 自由に電話をかけることができる.	1
	2. いくつかのよく知っている番号であればかけることができる.	1
	3. 電話で対応できるが電話をかけることはできない.	1
	4. まったく電話を使うことができない.	0
B. 買い物	1. ひとりで買い物ができる.	1
	2. 少額の買い物であればひとりでできる.	0
	3. だれかつきそっていれば買い物ができる.	0
	4. まったく買い物ができない.	0
C. 食事の支度（男性の場合は「もしできれば」で，参考扱いとする）	1. 人数にあった支度をして必要十分な用意ができる.	1
	2. 材料が用意してあれば食事の支度ができる.	0
	3. 食事をつくることはできるが，人数にあった用意ができない.	0
	4. 他人に支度をしてもらう.	0
D. 家事（男性の場合は「もしできれば」で，参考扱いとする）	1. 力仕事など以外はひとりで家事をすることができる.	1
	2. 食事のあとの食器を洗ったり布団を敷いたりするなどの簡単なことはできる.	1
	3. 簡単な家事はできるが，きちんとあるいは清潔に維持できない.	1
	4. 他人の助けがなければ家事をすることができない.	1
	5. まったく家事をすることができない.	0
E. 洗濯（男性の場合は「もしできれば」で，参考扱いとする）	1. ひとりで洗濯できる.	1
	2. 靴下などの小さなものは洗濯できる.	1
	3. 他人に洗濯してもらう.	0
F. 移動・外出	1. 自動車を運転したり，電車・バスを利用して出かけたりすることができる.	1
	2. タクシーを自分で頼んで出かけられるが，電車やバスは利用できない.	1
	3. つきそいがあれば電車やバスを利用することができる.	1
	4. つきそわれてタクシーや自動車で出かけることができる.	1
	5. まったく出かけることができない.	0
G. 服薬の管理	1. きちんとできる.	1
	2. 前もって飲む薬が用意されていれば自分で服用できる.	0
	3. 自分ではまったく服用できない.	0
H. 金銭の管理	1. 自分でできる（家計費，家賃，請求書の支払，銀行での用事など）.	1
	2. 日常の買い物は管理できるが，大きな買い物や銀行へはつきそいが必要.	1
	3. 金銭を扱うことができない.	0
		合計： 点

男性はA，B，F，G，Hを対象にして5点満点，女性はA～Hすべてを対象に8点満点とする．それぞれの項目（A～H）は0点か1点である．

index（表2）[2,3]，認知機能の評価であるMini-Mental State Examination（表3）[4]，うつの評価尺度であるGeriatric Depression Scale-15（表4）[5]などが用いられる．これらによって，総合的かつ包括的に評価を行い，看護師，栄養士，介護士などの医療者間で情報を共有することが，有効な生活指導につながる．たとえば，荒木らの報告では，高齢糖尿病患者ではIADLに障害を持つ者がまれではなく，80歳以上になるとIADLに障害を持つ者が著しく増加することが報告されている（表3）[6]．

食事指導

高齢者では，普段の食事の内容に個人差が大き
低栄養と栄養過剰の両方の場合がありうる．したが
て，まずは食事内容の把握が重要である．日本のコ
ロールの比較的不良な高齢糖尿病患者およそ1000名
対象とした臨床研究であるJapanese Elderly Diabe

表3 認知機能の評価(MMSE)(文献4)

配点	質問内容	回答	得点	得点法
1. 5点	今年は何年ですか？	年		正答1点，誤答0点
	今の季節は何ですか？			
	今日は何曜日ですか？	曜日		
	今日は何月何日ですか？	月		
		日		
2. 5点	ここは何県ですか？	県		正答1点，誤答0点
	ここは何市ですか？	市		
	ここは何病院ですか？	病院		
	ここは何階ですか？	階		
	ここは何地方ですか？	地方		
3. 3点	記憶力の検査をして良いか尋ねた後，1秒間に1つ程度の速さで互いに関係のないものの名前を3個言う．その後被検者が3つとも言えるまで最高6回までくりかえさせる．			初回の復唱のみ得点(0～3点)
4. 5点	100から順に7を引くよう指示する．5つの引き算を終えたところで(93, 86, 79, 72, 65)止める．これができない場合は「フジノヤマ」を逆から言わせる．			正しい回答のみ得点．途中で「86～7」などの指示をしない
5. 3点	「先程くりかえして言って覚えていただいた3つのものの名前を思い出して言ってください」			正答ごとに1点
6. 2点	(腕時計を見せながら)これはなんですか？			正答ごとに1点
	(鉛筆を見せながら)これはなんですか？			
7. 1点	次の文章をくりかえして下さい．『みんなで力を合わせて綱を引きます．』			1回のみで評価，正答すれば1点
8. 3点	白紙を1枚出し，3段階の命令を与える．『この紙を右手に持って，それを半分に折り，床の上に置いて下さい』			各段階の指示が正しくできるごとに1点
9. 1点	次の文章を読み，その指示に従って下さい．『目を閉じなさい．の文字を見せる』			文章を読み，両目を閉じたら1点
10. 1点	「なにか文章を書いて下さい」 例文を書き取らせるのではなく，自発的に書かれた文章でなくてはならない．			意味が通じる文章のみ得点を与える
11. 1点	次の図形を描いて下さい．			10個の角があり，その2つが交差してしていれば可．線の震えは可
		合計		30点

表4 うつの評価(GDS-15)(文献5)

1.	毎日の生活に満足していますか	(はい・いいえ)
2.	毎日の活動力や周囲に対する興味が低下したと思いますか	(はい・いいえ)
3.	生活が空虚だと思いますか	(はい・いいえ)
4.	毎日が退屈だと思うことが多いですか	(はい・いいえ)
5.	たいていは機嫌よく過ごすことが多いですか	(はい・いいえ)
6.	将来への漠然とした不安にかられることがありますか	(はい・いいえ)
7.	多くの場合は自分が幸福だと思いますか	(はい・いいえ)
8.	自分が無力だなあと思うことが多いですか	(はい・いいえ)
9.	外出したりなにか新しいことをするよりも，家にいたいと思いますか	(はい・いいえ)
10.	なによりもまず，物忘れが気になりますか	(はい・いいえ)
11.	いま生きていることが素晴らしいと思いますか	(はい・いいえ)
12.	生きていても仕方がないという気持ちになることがありますか	(はい・いいえ)
13.	自分が活気にあふれていると思いますか	(はい・いいえ)
14.	希望がないと思うことがありますか	(はい・いいえ)
15.	周りの人が，あなたよりも幸せそうにみえますか	(はい・いいえ)
	合計	15点

1，5，7，11，13には「はい」に0点，「いいえ」に1点を，2，3，4，6，8，9，10，12，14，15には「はい」に1点，「いいえ」に0点を配点し合計する．5点以上がうつ状態とされている．

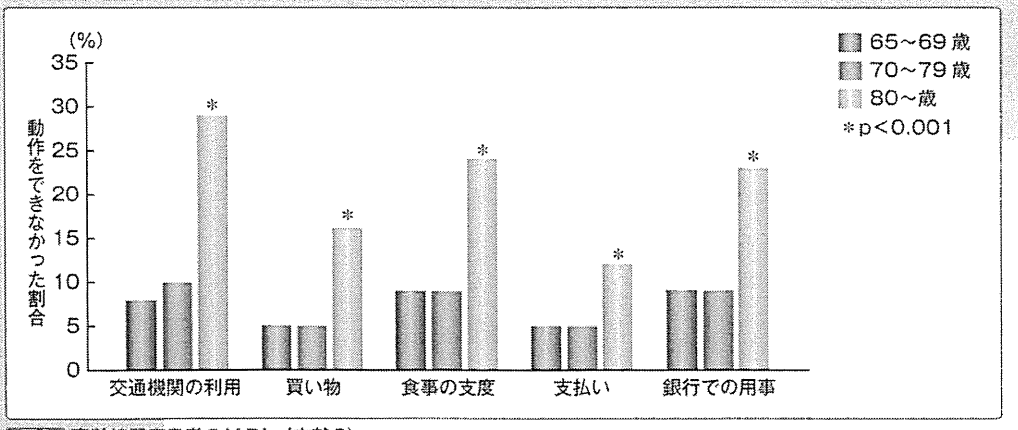

図3 高齢糖尿病患者のIADL（文献6）
80歳以上になるとIADLの障害が多くなる．

表5 簡易栄養指導（文献8）

1. 毎日3食，主食，主菜，副菜のある食事
2. 主食は定量
3. 主菜（蛋白質，魚，肉，大豆製品，卵）は毎食1品～1品半くらい
4. 副菜（野菜）は生なら両手1杯（半分は緑黄色野菜）：1食に2皿
5. 油脂料理は1日2品以下で朝食か昼食で
6. 牛乳，ヨーグルトはコップ1杯，ヨーグルトは低糖，プレーンを
7. 果物の量を確認 ●1日2個のもの（みかん，キウイフルーツ） ●1日1個のもの（バナナ，オレンジ） ●1日1/2個のもの（グレープフルーツ，りんご）
8. 菓子は多くても週1～2回程度，清涼飲料水はお茶に代える（はちみつ，みりん，飴に注意）
9. アルコールは1日2単位以内（ビールなら中瓶1本，日本酒1合，焼酎100 cc）

高齢者にも理解しやすい栄養指導を心がける必要がある．

Intervention Study（J-EDIT）の登録時の調査では，全体の4.7 %がBMI 18.5未満で，30.7 %がBMI 25以上であった．日本人の高齢糖尿病患者は肥満者も多いが，「やせ」の者もまれではないことがわかる[7]．また，J-EDITの食事調査では，日本人の高齢糖尿病患者の平均摂取カロリーは，男性で1802 ± 396 kcal，女性で1661 ± 337 kcalであった．標準偏差が大きいことから，摂取カロリーの個人差が大きいことがうかがえる．したがって，個人の食事摂取状況を正確に把握したうえで，的確な指導を行うことが求められる．

高齢者では，食べることが生きがいになっていることも多く，一律に高圧的な食事摂取制限にならないような配慮も必要とされるであろう．また，エネルギー制限にあまりこだわりすぎず，栄養素をバランスよく摂取できるような指導が有効な場合もあろう．

また，認知機能の低下がある場合も多いため，あま複雑になりすぎず，わかりやすい指導を心掛けるべきであ
たとえば，荒木らの推奨する簡易な栄養指導法も参考なる（表5）[8]．

高齢者では，糖質（菓子，果物）の過剰摂取が比較的い傾向にある．J-EDITのデータでも，肥満傾向の者に菓子類やソフトドリンク類の摂取が多い傾向が認められいる．そうした場合には，おやつ・間食の減量を指導ることが簡便かつ比較的効果的である．

J-EDITの解析では，野菜の摂取が多いこととHbAの有意な低下との間に関連を認めている[9]．たとえば，食あたり生野菜なら両手に1杯分，もしくは2皿の野菜理を摂取するように指導するとよい．

運動指導

高齢糖尿病患者における運動療法の意義は，もちろ血糖コントロールが大きな目的ではあるが，それだけにどまらず，健康づくり・体力づくりによってQOLの維向上を図ることが重要である．また，場合によって運動を通じて社会参加を促し，生きがいの形成にもつがるような指導が望まれる．

高齢者では，多くの疾患が併存していることが多

表6 メディカルチェック

1. 問診*	自覚症状，既往歴，家族歴，日常生活状態(食生活・運動など)
2. 診察*	身長，体重，血圧，脈拍数
	内科診察
	整形外科的診察(骨，関節など)
	眼科診察(眼底検査，白内障の有無)
3. 胸部X線*	立位正面像および側面像
4. 心電図	安静時12誘導心電図*
	運動負荷試験**（マスター法負荷，トレッドミル負荷，自転車エルゴメーター）
5. 血圧検査	白血球，赤血球，Ht，Hb，血小板，GOT，GPT，γ-GTP，LDH
	BUN*，クレアチニン*，尿酸*，Na，K，CT，血糖値*，グリコヘモグロビン(HbA1c)*，総コレステロール*，TG*，HDL-C*，リポ蛋白プロフィール
6. 尿検査	糖*，ケトン体*，蛋白*，潜血*，沈渣，微量アルブミン*
7. その他	腹部超音波検査（腹部エコー），心臓超音波検査（心エコー）**，心筋シンチグラフィー，肺機能検査，動脈血ガス分析など

* 必須項目：糖尿病合併症や他の慢性疾患(虚血性心疾患)の有無の確認のために必要な検査．
** 高齢者には実施が望ましい検査．
高齢糖尿病患者では，心血管疾患や整形外科的疾患を持つ者も多く，運動の指導の前にはメディカルチェックが欠かせない．

表7 Borg Scale

	英訳	日本語訳
19	very very hard	非常にきつい
18		
17	very hard	かなりきつい
16		
15	hard	きつい
14		
13	somewhat hard	ややきつい
12		
11	light	楽である
10		
9	very light	かなり楽である
8		
7	very very light	非常に楽である
6		

主観的な運動強度の目安としてはBorg Scaleが用いられる．9～12程度の強度を目安とするとよい．

糖尿病の合併症も進行していることが多い．また，恒常性維持の機構の機能低下もあり，水・電解質代謝などの異常をきたしやすい．そのため，運動指導の前には，メディカルチェックが必要である．一般的なメディカルチェックの項目を表に掲げる(表6)．

運動の強度が強すぎると無酸素的な代謝となり，かえって脂肪分解が抑制されるだけでなく，血圧上昇などの危険も伴いやすい．したがって，中等度以下の強度の運動を指導する．具体的には，最大酸素消費量の40～50％(60～70歳代では，脈拍数100/分以下程度を目安）の運動を1回10～30分，週3～5回程度実施することを勧めるのがよい．主観的な運動強度の指標であるBorg Scaleでは，9（かなり楽である）～12（ややきつい）程度とする(表7)．運動の種類としては，ウォーキング，ジョギング，自転車，水泳，体操など，全身の筋肉を使う有酸素運動が勧められる．また，筋力が低下した高齢者に対しては，軽いダンベル，タイヤチューブや，自重を用いた軽いレジスタンストレーニングなども併せるとより有効である．

運動前の準備体操や運動後のストレッチなども欠かさず実施するように指導する．運動中の低血糖に備えたグルコースの携帯，さらに脱水やこむらがえりを予防するための水分摂取についても説明すべきである．

介護保険を利用している患者の場合には，デイケアへの参加や，ヘルパーつきそいの散歩なども有効であろう．

薬物療法

高齢者では，服薬やインスリンの自己注射においても若年者とは異なる問題を抱える者が多く，高齢者に独特のさまざまな病態・背景因子を理解したうえで指導にあたる必要がある(表2)．

高齢者に独特な因子としては，①身体機能の低下，②認知機能の低下，③多病による多剤併用，④心理的特性，などが挙げられる．

身体機能の低下

加齢に伴って，手指の巧緻性の低下とともに，視力も低下する．また，脳血管障害による麻痺や感覚障害を持つ患者も多くなる．そのために，小さな錠剤などをつまみにくくなり，落としやすくなる．インスリンの自己注射には，単位数のみにくさや注射器の押しにくさなどの問題が生じやすい．より患者の使いやすいデバイスの選択も重要であろう．さらに，嚥下に問題を持つ者も増えてくることに注意を要する．

認知機能の低下

糖尿病は認知機能の低下および認知症のリスク因子で

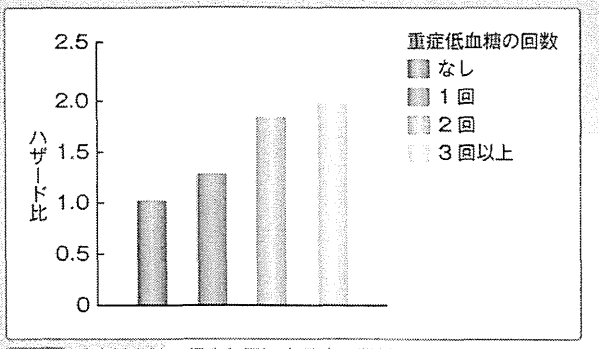

図4 重症低血糖の頻度と認知症発症の関係
重症低血糖の経験回数が多いほど，認知症発症のリスクが上昇する．

あり[10]，認知機能が低下している高齢糖尿病患者は多い．J-EDITでは多くの調査項目があるために，認知機能が比較的保たれていると主治医が判断した患者を登録したと思われるが，それでも，全体の6.5 %がMMSE 24点未満であった[11]．また，筆者らの外来通院患者を対象とした調査では，65～74歳の糖尿病患者の24 %，75歳以上の患者の47.4 %が，MMSE 24点未満であった[10]．したがって，服薬・インスリン自己注射・血糖自己測定などを指導する際は，患者の認知機能低下の有無を評価したうえで，その患者にあわせた指導が必要である．認知機能の低下した患者には，できるだけ一包化した服薬，服薬カレンダーや服薬ボックスの利用，家族や介護・看護サービスによる服薬確認，などを提案・指導する．

多病による多剤併用

高齢者では，糖尿病だけにとどまらず多くの疾患を持つ者が多く，薬剤も多種になる．糖尿病治療薬は服薬法が複雑になりがちであるが，できるだけ単純で遵守しやすい処方を考慮して指導にあたるべきである．

多剤併用は，薬物相互作用による副作用を引き起こしやすい．そのため，処方が変更になった際には副作用の可能性を十分に説明するだけでなく，その対応法の説明も求められる．

心理的特性

高齢者には，医療者に対して遠慮してしまう者もいるため，医療者になかなか本音をいえないことがある．たとえば，α-グルコシダーゼ阻害薬（α-GI）の内服により腹満が強くなり服用を中止したとしても，医療者への遠慮からなかなかいいだせず，服用を中止したまま処方を受けつづけるようなケースも見受けられる．また，家族にも遠慮してしまい食事の内容を変更するように医療者から指導を受けても食事の用意をしている家族に対して，自分のために特別配慮を求めることをいいだせないようなケースもある．こうした心理的な背景に気づき，配慮をすることが重要である．

また，糖尿病患者には抑うつを伴うことがまれでない[12]．抑うつはセルフケアの阻害因子であり，高齢者の生活指導においては常に配慮が求められる．

こうした高齢者に特有な因子を十分に理解したうえで患者のQOLに配慮した薬物治療の指導が必要であろう．

低血糖対策

高齢者では，多剤併用や腎機能などの低下による薬物代謝の遅延による効果の増強・遷延などのために，糖尿病治療薬による低血糖を起こしやすい．低血糖は，転倒骨折の原因となり，認知症発症（図4）[13]，心血管イベント発症[14]のリスクを上昇させる．また，低血糖の症状が非典型的になり，発見が遅れることも多い．重症低血糖のリスク因子は，腎不全，多剤併用，インスリン使用，食事摂取の低下，認知症，退院直後などであり，これらの因子を持つ患者には一層の注意が必要である．患者自身が低血糖を起こしやすい時間帯や行動などを理解してもらいブドウ糖などによる対処の仕方を十分に説明する必要がある．

口腔ケア

糖尿病では歯周病が多くなるため，口腔ケアも重要である．高齢者にとって，残存歯を維持することは咀

力を保つうえでも重要であり，栄養の摂取にも関連する問題である．

また，糖尿病患者は免疫力の低下をきたすため，肺炎などの感染症のリスクも高い．口腔ケアは，誤嚥などをもとにした高齢者の肺炎の発症を減らす効果もあり[15]，口腔ケアの指導も重要である．

フットケア

高齢者では，身体の柔軟性がなくなるため前屈みの姿勢がとりづらくなるうえに，視力の低下などによって自分の足の観察がしづらくなり，足の変化がみすごされる．できるだけ毎日足を観察するように指導するとともに，家族にもフットケアの重要性の指導が必要となる場合が多い．

高齢者では白癬の合併も多く，とくに爪白癬患者が多いために，爪切り法の指導も必要である．また，加齢とともに皮膚が乾燥しやすくなるうえに，神経障害を合併すると発汗機能が低下してさらに乾燥しやすくなり，踵周囲などの皮膚のひび割れなどが増えてくるため，保湿の指導も必要となる．足にあった靴を選ぶこと，清潔を保持するために毎日靴下を交換するなどの指導が必要であろう．

まとめ

高齢糖尿病患者の生活指導においては，糖尿病の状態のみでなく，身体機能(BADL, IADL)の評価，認知機能，抑うつなどを含む精神心理面の評価，家族状況などの評価を包括的に行う高齢者総合機能評価（CGA）が有用である．患者本人の状況にあわせ，QOLを維持・向上させることを目標とした生活指導を行うことが重要である．

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REVIEW ARTICLE

Cognitive dysfunction: An emerging concept of a new diabetic complication in the elderly

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The incidence of type 2 diabetes mellitus (T2DM) has risen, and this trend is likely to continue. Recent advances suggest that T2DM is a risk factor for cognitive decline. We are now encountering novel complications of T2DM, namely cognitive dysfunction and dementia. Although the treatment strategy for diabetic patients with neurocognitive dysfunction has received a great deal of attention, the appropriate level of glycemic control for the prevention of the development and/or progression of cognitive decline in elderly diabetic patients remains to be elucidated. Another issue in diabetic treatment in patients with cognitive dysfunction is the selection of medicines. The best choice and combination of antidiabetic medications for the preservation of cognition should also be studied. Ample studies suggest that exercise helps to preserve cognitive function, although existing evidence does not necessarily indicate its effectiveness exclusively in diabetic patients. Exercise is a helpful non-pharmacological therapy. Considering the progressive aging of the worldwide population, more research to investigate the best way to manage this population is important. **Geriatr Gerontol Int 2013; 13: 28–34.**

Keywords: Alzheimer's disease type dementia, hypoglycemia, insulin resistance, neurocognitive assessment, vascular dementia.

Introduction

The incidence of type 2 diabetes mellitus(T2DM) has risen, and this trend is expected to continue.[1] Recent remarkable advances in pharmacological therapy in T2DM have resulted in a wide variety of treatments. Many large clinical trials have been carried out, and a variety of interventions are now available to prevent and treat the classic microvascular and macrovascular complications that occur with DM, so that people are living longer with the condition.[2] Recent studies suggested that T2DM is a risk factor for cognitive dysfunction and dementia in the elderly. With the increase in the number of elderly individuals with DM, the number of diabetic patients with cognitive dysfunction has been increasing. We are now encountering novel complications of T2DM that are not targeted by the current management strategies. As one of these new targets, cognitive impairment and dementia in patients with T2DM has generated a great deal of interest, and diabetic treatment in this population that takes brain protection into consideration should be provided.

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Cognitive impact of T2DM

Large epidemiological studies have shown the cognitive impacts of T2DM. In the Rotterdam Study,[3] T2DM patients showed an increased risk of developing dementia. The study also showed that patients treated with insulin were at a 4.3-fold higher relative risk for dementia. The Hisayama Study showed that the incidence of all-cause dementia, Alzheimer's disease (AD) and vascular dementia were significantly higher in patients with diabetes than in those with normal glucose tolerance[4] The same study showed that systemic insulin resistance was associated with the pathogenic process of AD, neuritic plaques formation.[5] The Religious Orders Study, which observed some 800 nuns and priests longitudinally for 9 years, showed that diabetic people had a 65% increased risk of developing AD.[6] The Honolulu Asia Aging Study, a cohort of Japanese Americans in Hawaii, showed that the diabetic population had a 1.8-fold higher risk of developing AD and a 2.3-fold risk of vascular dementia.[7,8]

Prospective trials also suggested that T2DM caused cognitive function to deteriorate in the elderly.

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A diagnosis of diabetes increased the odds of cognitive decline 1.2-fold to 1.7-fold (95% CI 1.3–2.3) in several neurocognitive assessments.[9] A recent systematic review of large prospective trials reported that T2DM increased the risk of AD by a factor of 1.59 (range 1.15–2.7).[10] Another systematic review reported that T2DM has a risk of vascular dementia of 2.0–4.2.[9,11]

The advances in the research in this field strongly suggest that T2DM is a risk factor for cognitive dysfunction or dementia.[12,13]

Assessment of diabetes-associated cognitive dysfunction

To screen patients with cognitive impairment, several neuropsychological assessment tools might be applied. The Mini-Mental State Examination (MMSE) is an assessment scale for global cognition including orientation, memory, calculation, verbal ability and constructional disability.[14] A full score is 30, and a cut-off point of 23 out of 24 is usually used for the screening of dementia. The MMSE subset analysis identified impaired attention and calculation as specific characteristics of DM patients,[15] whereas patients with AD had lower scores in temporal orientation and recall.[16]

As a part of a large cohort study of older DM patients (Japanese Research of Cholesterol and Diabetes Mellitus, UMIN000000516 Japan CDM), we carried out MMSE on diabetic patients aged older than 65 years in a diabetic outpatient clinic (52 males, 61 females; mean age 74.7 ± 4.6 years). Of these patients, 75 were aged less than 75 years (younger-old mean age 69.9 ± 4.7 years) and 38 patients were aged older than 75 years (older-old mean age 80.7 ± 4.4). In the younger-old group, 76.0% of patients (57/75) had a MMSE score of more than 24 (mean score 25.3 ± 4.7), and in the older-old group, 52.6% (20/38) had a MMSE score of more than 24 (mean score 24.2 ± 4.6). This small assessment showed that many diabetic patients had lower cognitive scores indicative of dementia, especially in the older-old.

Diabetes affects a wide range of cognitive domains.[17] Among the domains affected by T2DM, cognitive speed might provide early detection of diabetes-related cognitive decline[18,19] The digit symbol substitution test (DSST) is a test of cognitive speed that can be carried out relatively easily. It consists of a number (e.g. nine) of digit-symbol pairs (followed by a list of digits). Under each digit, the patient is asked to write down the corresponding symbol as quickly as possible. The number of correct symbols written within the allowed time (e.g. 90 or 120 s) is measured.

In clinical settings, the diagnosis of dementia is generally made based on the Diagnostic and Statistical Manual of Mental Disorders III revised criteria in patients with or without DM.[20] The disturbance in memory impairment with at least one of the following is required for the diagnosis of dementia: abstract thinking, judgement, higher cortical function and personality changes interferes with work or social activities. The leading cause of dementia in diabetic patients is AD, as is those without DM. DM patients often have cerebrovascular disease, and clinical-pathological studies support the notion that vascular lesions aggravate the deleterious effects of AD pathology by reducing the threshold for cognitive impairment.[21]

Pathogenesis of diabetes-associated cognitive dysfunction

The precise mechanisms underlying T2DM-related cognitive dysfunction or the development of dementia, especially AD-type dementia, remain to be elucidated; however, several hypothetical mechanisms have been proposed (Fig. 1). To develop pharmacological and non-pharmacological strategies for treating the diabetic elderly with cognitive impairment, elucidating the pathogenesis of this complication might be essential.

High glucose concentration, a major pathological characteristic of diabetes, might have toxic effects on neurons in the brain through osmotic insults and oxidative stress, and the maintenance of chronic high glucose also leads to the enhanced formation of advanced glycation end-products (AGE).[22] AGE couple with free radicals and create oxidative damage, which in turn leads to neuronal injury,[23] and they also reactivate microglia, the resident innate immune cells in the brain. A wealth of evidence shows that activated microglia can become deleterious and damage neurons.[24]

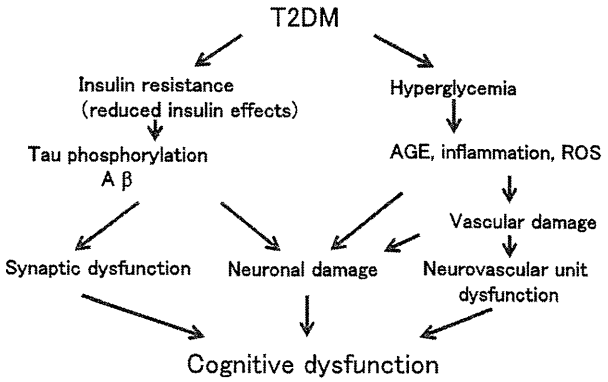

Figure 1 Pathogenesis of type 2 diabetes mellitus (T2DM)-associated cognitive dysfunction. Cognitive dysfunction in T2DM is induced by multiple pathways. Insulin resistance might be associated with Alzheimer's disease pathology, and hyperglycemia induces advanced glycation end-products (AGE) formation, inflammation and reactive oxygen species (ROS) production, which might lead to neuronal damage and neurovascular dysfunction.

T2DM, especially in conjunction with obesity, is characterized by insulin resistance and/or hyperinsulinemia. Insulin degrading enzyme (IDE) catabolizes insulin in the liver, kidneys and muscles.[25,26]

It is generally agreed that insulin located within the brain is mostly of pancreatic origin, having passed through the blood–brain barrier, although there is debate about the amount of insulin that is produced de novo within the central nervous system.[27] Major known actions of insulin in the brain include control of food intake (through insulin receptors located in the olfactory bulb and thalamus) and effects on cognitive functions, including memory.[28,29] Insulin also regulates acetylcholine transferase expression, which is an enzyme responsible for acetylcholine (ACh) synthesis. ACh is a critical neurotransmitter in cognitive function, and it might be relevant to neurocognitive disorders in diabetics.[30] Recent basic research showed that insulin signaling in the central nervous system prevents the pathological binding of amyloid beta (Aβ) oligomers.[31] Aβ oligomers are soluble molecules that attach with specificity to particular synapses, acting as pathogenic ligands.[32]

Insulin has multiple important functions in the brain, as aforementioned. These functions are disrupted in insulin-resistant states. The transport of insulin into the brain across the blood–brain barrier is reduced in insulin-resistance-associated hyperinsulinemia, and insulin levels in the brain are subsequently lowered.[33,34] Intranasal insulin showed some benefits in early AD patients.[35] With intranasal administration, insulin bypasses the periphery and the blood–brain barrier, reaching the brain and cerebrospinal fluid within minutes through extracellular bulk flow transport along olfactory and trigeminal perivascular channels, as well as through more traditional axonal transport pathways.[36,37]

Some basic research suggests that insulin signaling is involved in AD-related pathology through its effects on the Aβ metabolism and tau phosphorylation.[38] Insulin signaling activates PI3K/Akt pathway, which leads to inactivation of glycogen synthase kinase-3β (GSK-3β). GSK-3β regulates tau phosphorylation, one of the main pathological components in AD. Less insulin signaling might also induce increased activity of GSK-3β, which leads to the enhanced phosphorylation of tau protein and the formation of neurofibrillary tangles.[39] Decreased insulin signaling reduces the synthesis of several proteins, including IDE. IDE degrades Aβ as well as insulin, and reduced amounts of IDE might result in greater amyloid deposition. The results of pathological assessments in AD with or without DM, however, are highly controversial.[40,41] More research would be warranted to elucidate the relevance of insulin and insulin resistance in the underlying mechanism of T2DM-assocaiated cognitive dysfunction.

Diabetic patients often have ischemic brain lesions.[42] Even asymptomatic cerebral infarctions have affects on the cognition in elderly diabetic patients.[18,43] On cerebral magnetic resonance imaging, white matter hyperintensities and lacunae, both of which are frequently observed in the elderly, are generally viewed as evidence of small vessel disease in the brain (white matter lesions and lacunae). Small vessel diseases affect cognitive function in older diabetics.[18,44] DM also affects the function of microvascular endothelial cells. The deterioration of the endothelial cell function leads to the disruption of blood–brain barrier function, which might induce neuroinflammatory reactions and neurodegeneration.[45] The endothelial cells play a critical role in the control of hemodynamic coupling among neuronal, glial and vascular components; that is, "neurovascular units". Dysfunction of "neurovascular units" might have some impact on cognition in diabetic patients.[46]

Treatment of vascular risk factors including T2DM was reportedly associated with a lower conversion rate from mild cognitive impairment to AD[47] or slower cognitive decline in AD patients.[48] Comprehensive management in DM patients should be warranted.

Treatment and management of diabetic patients with cognitive impairment

T2DM is associated with cognitive dysfunction; however, it has not yet been made clear whether glycemic control leads to the preservation or improvement of cognitive function. Several prospective studies[19,49,50] have shown that higher glycated hemoglobin (HbA1c) levels at baseline are associated with cognitive decline. A recent prospective study by Christman *et al.*, however, showed that HbA1c levels at baseline had no effects on cognitive function.[51] A large cohort study, the Action to Control Cardiovascular Risk in Diabetes–Memory in Diabetes (ACCORD-MIND) trial, has found that HbA1c levels were cross-sectionally associated with worse performance on several cognitive functional tests.[52] However, the results of the interventional study were rather disappointing.[53] Although total brain volume in the intensive glycemic control group was significantly greater than in the standard treatment group after 40 months, there was no significant difference in cognitive assessment. The results of the study, however, should be interpreted cautiously because of the early drop-outs in the intervention group.

In the ACCORD-MIND study, the intensive control group achieved a HbA1c level of 6.6% compared with 7.5% in the standard treatment group. Several smaller studies involving less intensive glycemic treatment, however, indicated that modest cognitive decrements in patients with T2DM are partially reversible with the improvement of glycemic control,[54–59] although not invariably.[60] Postprandial hyperglycemia is associated

with atherosclerosis and diabetic complications,[61] and a control of postprandial hyperglycemia might prevent cognitive decline in older diabetic individuals.[59] These studies suggested that metabolic control might have beneficial effects in terms of cognitive function; however, the appropriate levels of blood glucose control remain unclear. In contrast, a recent report has suggested that a history of severe hypoglycemic episodes is associated with a greater risk of dementia.[62] The diabetic control in this population should be balanced between the merits of treatment and the risk of hypoglycemia.

Another issue related to the treatment that pertains to cognitive dysfunction is the selection and combination of antidiabetic medicines. The Rotterdam Study reported that insulin use increased the incidence of dementia.[3] However, many confounding factors must be considered when interpreting the results of that study. The patients who used insulin might have had worse diabetic control, a longer history and more complications, and these factors might have some impact on the incidence of dementia. Greater insulin resistance means that a greater amount of insulin is required to control the blood glucose level. The association of the use of an excessive amount of insulin with insulin resistance status might be undesirable, the appropriate prescription of insulin for maintaining a desirable blood glucose level has not yet been determined for individuals with insulin resistance. A small study reported that pioglitazone, an insulin sensitizer, has some beneficial effects on cognition in AD.[63] Comprehensive management in combination with insulin use would be necessary to achieve appropriate glycemic control, and efforts to reduce insulin resistance would be warranted.

Recently, a new class of diabetic pharmacological treatments known as incretin-related medicines has emerged. Glucagon-like peptide 1 (GLP-1) and glucose-dependent insulinotropic peptide (GIP), whose activity is reduced in insulin resistance, have been implicated in central nervous system function, including cognition, synaptic plasticity and neurogenesis.[64] An animal study showed that GLP-1 prevented the neurodegenerative developments in AD model mice.[65] Further clinical investigation from the perspective of brain protection is warranted.

Many studies suggested that exercise has the potential to protect brain function. A systematic review of the Cochran database by Angevaren *et al.* reported the effects in elderly individuals without known cognitive impairment, and another systematic review of a prospective cohort study by Hamer *et al.* reported that exercise reduces the risk of incidence of dementia by 28% and of AD by 45%.[66,67]

Exercise also has effects on patients with mild cognitive impairment and dementia.[68] Although existing evidence does not indicate the effects of exercise on the protection of brain function exclusively in the diabetic population, exercise has multiple established effects on diabetic patients, including the improvement of insulin resistance. Studies to investigate the effects of exercise on diabetic cognitive dysfunction are warranted.

Cognitive dysfunction is associated with poor ability of self-care in elderly diabetics, and the use of both health and social services.[69] In addition, physical function is often more compromised in those with cognitive impairment. Individuals with DM with cognitive impairment might have difficulty carrying out the daily tasks of DM self-care effectively,[70] which might result in worse glycemic control than in individuals without cognitive impairment. A study reported that cognitively impaired DM patients were at increased risk of mortality and functional disability.[71] The relationship between cognition and self-management ability might be bidirectional. While it could be that poor self-management practices lead to poorer metabolic control and therefore brain dysfunction, cognitive deterioration would lead to changes in self-management ability.

A depressive mood is often comorbid with dementia,[72] especially in diabetics.[73] Depressed mood might also be associated with cognitive impairment and might interfere with effective self-management.[74–77]

People with dementia often experience behavioral and psychological symptoms of dementia (BPSD) during the course of their illness. The management of dementia is complicated by BPSD, such as psychosis, depression, agitation, aggression and disinhibition. BPSD also disrupts the daily diabetes care routine, with "denial" of having diabetes or memory loss (anosognosia) being the most disruptive.[78] Caregivers often report that caring for both diabetes and dementia is highly burdensome, that they feel overwhelmed by BPSD, and that they want more support from family and from the patients' healthcare providers.

To control BPSD, antipsychotic medication is sometimes prescribed. Antipsychotic drugs, especially second-generation drugs including olanzapine and quetiapine, have the potential to induce weight gain and elevate plasma glucose levels.[79] The use of these drugs in demented diabetic patients should be avoided.

Conclusion

Cognitive dysfunction might be a novel class of diabetic complication in the elderly. The management of diabetic patients with this complication is challenging and presents many unresolved problems. Considering the progressive aging of the worldwide population, it will be important to carry out investigations to improve our understanding of the association between T2DM and cognitive dysfunction, and to determine the best way to manage these populations.

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Disclosure statement

Nothing to declare.

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ORIGINAL ARTICLE: EPIDEMIOLOGY, CLINICAL PRACTICE AND HEALTH

Cognitive impairments and functional declines in older adults at high risk for care needs

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Aim: Functional status of those who have very mild cognitive impairment have not been sufficiently investigated. In the current study, we analyzed the characteristics of functional awareness in older adults who had cognitive impairment and were at high risk of requiring support/care (termed as specified elderly at high risk for care needs in the long-term care insurance scheme).

Methods: The answers of a health check, which is provided by the local municipal government for those aged 75 years or older who have not been certified as eligible for care services, were analyzed. The differences of the variables between the two groups regarding yes/no answers to each of three cognition-related questions were analyzed. Then, a multiple logistic analysis was carried out to investigate the association of yes/no answers of the three cognition-related questions and the awareness of functional decline.

Results: The participants who had cognitive impairment had greater awareness of functional declines. Multiple logistic regression analysis showed that subjective memory impairment and disorientation were significantly associated with a wider range of awareness of functional decline.

Conclusions: Subjective cognitive impairment was associated with a wide range of awareness of functional decline in older adults at high risk for care need. **Geriatr Gerontol Int 2013; 13: 77–82.**

Keywords: depressive mood, dysphagia, instrumental activities of daily life, memory impairment, physical activity, vitality.

Introduction

Screening for cognitive impairment is essential for better health outcomes. Early identification and intervention holds the promise of improving overall care for affected persons through the use of chronic disease management strategies. In general, the existing literature does not support screening of unselected older adults for cognitive impairment;[1] however, screening in a high-risk population might be valid.

Several factors are closely associated with mild cognitive impairment (MCI) and very early dementia. Depressive mood might be a risk factor or an early manifestation of dementia.[2–4] Subtle impairments of instrumental activities of daily living (IADL) might also be very early manifestations.[5,6]

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In Japan, the public long-term care insurance system provides services to older adults who have been certified as requiring support (level 1 and 2) or care (levels ranging from 1 to 5 depending on their care needs). Uncertified, but not quite healthy, older adults who are considered at high risk of requiring support/care are categorized as specified elderly at high risk of care needs (specified elderly are provided with preventive care services by the municipalities in which they reside). The specified elderly are community-dwelling and have neither basic activities of daily living (B-ADL) impairments nor dementia. The specified elderly, however, is supposed to be the transitional stage to requiring care. Elucidating the characteristics of this group and developing some adequate intervention on this population to prevent the transition to requiring care are warranted. The local governments provide a health check of the uncertified elderly annually, in which all examined subjects complete a basic yes/no questionnaire that consists of simple assessments of their instrumental activities of daily living (7 items), memory problems (3 items), walking status (5 items), dysphagia (3 items), nutritional

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status (2 items) and depressive mood (5 items).[7] Subjective memory complaint might be an easy method to screen cognitive impairment, and a report showed that subjective memory complaint was associated with depressive mood and difficulties of activities of daily living (ADL).[8] In this assessment, subjective cognitive dysfunction was evaluated by three questions, and in the same assessment awareness of functional declines were also evaluated.

However, the functional characteristics of those who have subjective cognitive impairment by this assessment in the specified elderly at high need for requiring care have been unclear. Elucidating the characteristics of this population might lead to the development of intervention for the prevention of the transition to dementia and/or the status of requiring care.

In order to portray the characteristics of awareness of functional decline in those who are considered to have subjective cognitive impairment by this assessment, we examined the associations between non-cognitive items and cognitive items of the questionnaire in older adults at high risk of requiring support/care.

Methods

Measurements

To screen the elderly at high risk for care, a health check is provided by the local municipal government for those elderly aged 75 years or older who have not been certified as eligible for care services.

The health check includes a yes/no questionnaire that consists of simple assessments of their IADL (7 items), subjective cognitive problems (3 items), walking status (5 items), dysphagia (3 items), nutritional status (2 items) and depressive mood (5 items). In the current study, we calculated the scores for each of these six domains, with higher scores indicating worse functioning. The data for 1163 men and 2651 women who were determined to be specified elderly were obtained from annual health checks implemented in one of the urban municipalities in central Japan during October and November in 2009.

Continuous variables (age, blood pressure, hemoglobin, serum albumin and body mass index) were compared by Student's *t*-test, and others were compared by χ^2 analysis.

The questionnaire was as follows;

1) IADL

1. Do you go out alone using transportation? 2. Do you shop for daily necessaries by yourself? 3. Do you manage your bank account on your own? 4. Do you visit your friends alone? 5. Are you consulted by your family or friends?

2) Waking status

6. Do you climb up the stairs without holding onto handrails or walls? 7. Do you stand up without assistance? 8. Can you walk for more than 15 min without rest? 9. Have you fallen within a year? 10. Are you anxious about falls?

3) Nutrition

11. Have you lost more than 2–3 kg in weight in the recent 6 months? 12. BMI < 18.5 kg/m^2

4) Dysphagia

13. Do you have difficulty in eating hard food? 14. Do you choke with liquid? 15. Do you care about dry mouth?

5) Vitality

16. Do you go out more than once a week? 17. Do you go out less frequently than last year?

6) Cognition

18. Are you told that you repeatedly ask the same things? 19. Do you look up the numbers, dial and make phone calls without help? 20. Do you sometimes forget the date?

7) Depressive mood

21. Do you feel unfulfilled with daily life? 22. I do not enjoy my life as I used to (recent 2 weeks). 23. I feel more bothered to do everyday things than before (recent 2 weeks). 24. I do not feel that I am useful (recent 2 weeks). 25. I feel tired for no reason (recent 2 weeks).

The differences of the variables between the two groups regarding yes/no answers to each of the three cognition-related questions (Are you told that you always ask the same things? [memory]; Do you look up numbers, dial and make calls without help? [telephone]; Do you sometime forget what day it is? [orientation]) were analyzed. In the analysis, answers for related questions were scored as follows: IADL, 0–5; walking status, 0–5; depressive mood, 0–5; dysphagia, 0–3; vitality, 0–2; and nutritional status, 0–2. The difference of the distribution was analyzed by Student's *t*-test, Mann–Whitney *U*-test, or χ^2 analysis. Then, a multiple logistic analysis was carried out to investigate the association of yes/no answers of these three cognition-related questions and the awareness of functional decline.

Results

The characteristics of the participants are shown in Table 1.

IADL, walking status, depressive mood, vitality, and nutrition were all associated with subjective memory impairment and disorientation in univariate analysis (Tables 2 and 4). IADL, walking status, depressive mood and vitality were associated with an inability to call by themselves, but dysphagia and nutritional status were not significantly associated (Table 3).

Multiple logistic regression analysis showed that vitality was not associated with each of the three

cognition-related items (Table 5), although it was associated in univariate analysis (Tables 2–4). Nutritional status was not associated with subjective memory impairment and disorientation by multiple logistic regression analysis either (Table 5).

Discussion

The present study showed that self-claiming memory impairment was associated with a wide range of awareness of functional decline. The results also showed that depressive mood was significantly associated with subjective cognitive impairment. Community studies in normally-aging populations suggest that depression is associated with cognitive decline.[9–18] Older adults with depression often present with signs and symptoms indicative of functional or cognitive impairment. These somatic symptoms make evaluating and treating depression in older adults more complex. Depression in late life is more frequently associated with cognitive changes. Cognitive impairment in late-life depression might be a result of a depressive disorder or an underlying dementing condition. Memory complaints are also common in older adults with depression. There is a wide range of cognitive impairment in late-life depression, including decreased central processing speed, executive dysfunction and impaired short-term memory. The etiology of cognitive impairment might include cerebrovascular disease, which likely interrupts key pathways between frontal white matter and subcortical structures important in mood regulation and structural changes, such as hippocampal atrophy.[19] Depressive symptoms often coexist with dementia or MCI.[4] In the current survey, the questionnaire asked for subjective answers regarding cognitive function. Hence, one cannot deny the possibility that depressive mood might have interfered with the self-assessment of one's own cognition.

Memory impairment and disorientation was associated with lower walking status. The association of physical activity and memory is well recognized.[20,21] Also, an association between physical frailty and cognitive dysfunction has been reported.[22,23] Physical frailty is associated with the risk of MCI and a rapid rate of cognitive decline in aging.[24] A lower level of fitness was associated with hippocampal atrophy,[25] and exercise training increased the hippocampal volume.[26] The current results were in agreement with these previous findings.

Table 1 Participants' backgrounds

n	3814
Age (years)	75.1 (6.2)
Sex (male/female)	1163/2651
Body mass index	22.5 (4.5)
Systolic BP (mmHg)	134.0 (17.8)
Diastolic BP (mmHg)	74.4 (11.0)
Hemoglobin (g/dL)	12.8 (1.4)
Albumin (g/dL)	4.2 (0.3)

Mean (SD). BP, blood pressure.

Table 2 Differences between participants with or without memory impairment

	No memory impairment	Memory impairment	*P*-value
n	2654	1160	
Age (years)	74.6 ± 6.0	76.2 ± 6.4	<0.01
Male (% of male)	799 (30.1)	364 (31.4)	0.45
Body mass index(kg/m^2)	22.6 ± 4.7	22.4 ± 4.1	0.10
Systolic BP (mmHg)	134.2 ± 18.0	133.6 ± 17.4	0.33
Diastolic BP (mmHg)	74.5 ± 11.0	73.9 ± 10.9	0.12
Hemoglobin (g/dl)	12.8 ± 1.4	12.7 ± 1.4	<0.01
Albumin (g/dl)	4.3 ± 0.3	4.2 ± 0.3	0.02
IADL (0–7)	5.8 ± 1.5	5.1 ± 1.8	<0.01
Walking status (0–5)	2.8 ± 1.4	2.5 ± 1.3	<0.01
Depressive mood (0–5)	1.3 ± 1.5	2.3 ± 1.7	<0.01
Dysphagia (0–3)	1.5 ± 1.0	1.8 ± 1.0	<0.01
Vitality (0–2)	1.6 ± 0.6	1.3 ± 0.7	<0.01
Nutrition (0–2)	16 ± 0.6	1.5 ± 0.6	0.01

Mean ± SD. Age, body mass index, systolic and diastolic blood pressure (BP), hemoglobin and albumin were analyzed by Student's *t*-test. Sex was analyzed by χ^2-test. Instrumental activities of daily living (IADL), walking status, depressive mood, dysphagia, vitality and nutrition were analyzed by Mann–Whitney *U*-test.

Table 3 Differences between participants with or without impairment in telephone function

	No impairment	Impairment	*P*-value
n	3350	464	
Age (years)	74.9 ± 6.0	76.5 ± 7.2	<0.01
Male (% of male)	981 (29.3)	182 (39.2)	<0.01
Body mass index (kg/m^2)	22.5 ± 4.5	22.6 ± 4.8	0.88
Systolic BP (mmHg)	133.8 ± 17.8	135.7 ± 17.9	0.03
Diastolic BP (mmHg)	74.2 ± 10.9	75.21 ± 1.0	0.07
Hemoglobin (g/dL)	12.8 ± 1.4	12.9 ± 1.5	0.23
Albumin (g/dL)	4.2 ± 0.3	4.3 ± 0.4	0.85
IADL (0–7)	5.8 ± 1.4	4.1 ± 2.0	<0.01
Walking status (0–5)	2.8 ± 1.4	2.4 ± 1.4	<0.01
Depressive mood (0–5)	1.6 ± 1.6	2.2 ± 1.8	<0.01
Dysphagia (0–3)	1.6 ± 1.0	16 ± 1.0	0.73
Vitality (0–2)	1.5 ± 0.6	13 ± 0.7	<0.01
Nutrition (0–2)	1.6 ± 0.6	1.6 ± 0.6	0.72

Mean ± SD. Age, body mass index, systolic and diastolic blood pressure (BP), hemoglobin and albumin were analyzed by Student's *t*-test. Sex was analyzed by χ^2-test. Instrumental activities of daily living (IADL), walking status, depressive mood, dysphagia, vitality and nutrition were analyzed by Mann–Whitney *U*-test.

Table 4 Differences between participants with or without disorientation

	No impairment	Impairment	*P*-value
n	2550	1264	
Age (years)	74.7 ± 5.9	76.0 ± 6.7	<0.01
Male (% of male)	743 (29.1)	420 (33.2)	0.01
Body mass index (kg/m^2)	22.7 ± 4.7	22.3 ± 4.1	0.01
Systolic BP (mmHg)	134.2 ± 17.7	133.7 ± 18.0	0.49
Diastolic BP (mmHg)	74.6 ± 10.7	73.9 ± 11.4	0.09
Hemoglobin (g/dL)	12.8 ± 1.4	12.8 ± 1.4	0.84
Albumin (g/dL)	4.3 ± 0.3	4.2 ± 0.3	0.02
IADL (0–7)	5.8 ± 1.5	5.1 ± 18	<0.01
Walking status (0–5)	2.8 ± 1.4	2.6 ± 1.4	<0.01
Depressive mood (0–5)	1.3 ± 1.5	2.3 ± 1.7	<0.01
Dysphagia (0–3)	1.5 ± 1.0	1.8 ± 1.0	<0.01
Vitality (0–2)	1.5 ± 0.6	1.3 ± 0.7	<0.01
Nutrition (0–2)	1.6 ± 0.6	1.5 ± 0.6	0.02

Mean ± SD. Age, body mass index, systolic and diastolic blood pressure (BP), hemoglobin and albumin were analyzed by Student's *t*-test. Sex was analyzed by χ^2-test. Instrumental activities of daily living (IADL), walking status, depressive mood, dysphagia, vitality and nutrition were analyzed by Mann–Whitney *U*-test.

Awareness of lower IADL was significantly associated with subjective cognitive impairment. This finding is conceivable, given that IADL requires complex cognitive function, and becomes vulnerable in early stages of cognitive decline.[27–29]

Univariate analysis showed that vitality was associated with awareness of subjective cognitive declines; however, multiple logistic analysis did not show a significant association with subjective cognitive dysfunction in the current study. The exclusion of depressive mood from the multiple regression analysis models made both vitality and nutrition significantly associated with cognition-related items (data not shown). The association of vitality with subjective cognitive declines might be at least partly through depressive mood. Toba *et al.* reported that vitality was impaired in the elderly with cognitive impairment.[30] That study involved more severely

Table 5 Results of multiple logistic regression analysis

	Memory Odds ratio	95% CI	*P*-value	Telephone Odds ratio	95% CI	*P*-value	Orientation Odds ratio	95% CI	*P*-value
Age	1.021**	1.009–1.034	<0.01	0.994	0.997–1.011	0.48	1.011	0.999–1.023	0.08
Sex	1.013	0.860–1.193	0.88	0.769*	0.612–0.965	0.02	0.888	0.758–1.042	0.15
IADL	1.125**	1.060–1.194	<0.01	1.824**	1.693–1.966	<0.01	1.154**	1.088–1.224	<0.01
Walking status	1.072*	1.008–1.140	0.03	1.043	0.954–1.140	0.36	1.065*	1.003–1.131	0.04
Depressive mood	1.283**	1.222–1.347	<0.01	1.075*	1.005–1.151	0.04	1.298**	1.237–1.361	<0.01
Dysphagia	1.342**	1.284–1.458	<0.01	1.027	0.914–1.153	0.66	1.300**	1.199–1.410	<0.01
Vitality	1.061	0.913–1.235	0.44	1.048	0.880–1.248	0.60	1.005	0.866 1.166	0.95
Nutrition	1.050	0.932–1.182	0.43	0.929	0.782 1.104	0.41	1.095	0.975 1.229	0.13

$^{**}P < 0.01$; $^{*}P < 0.05$. IADL, Instrumental activities of daily living.

cognitively impaired participants than the current study, which might be a reason of the discrepancy with the current study.

Univariate analysis showed an association between nutritional status and awareness of cognitive declines (memory and orientation); however, multiple regression analysis did not. This might also be a result of adjustment for depressive mood.

The present finding that dysphagia was associated with memory impairment and disorientation is not in agreement with a recent study showing that memory was not associated with dysphagia.[31] In the current study, we could not obtain information about the comorbidity of the interviewees. Therefore, one can speculate that the difference in the rate of stroke prevalence might explain the discrepancy. The observed discrepancy requires further substantiation.

The association of subjective cognitive impairment and a wide range of awareness of functional declines might suggest that these functional impairments may share a common pathology, which leads to a construction of complex interactions among symptoms of geriatric syndrome or frailty syndrome.

The current study suggested that subjective cognitive impairment assessed by a relatively simple questionnaire was associated with a wide range of functional decline in older adults at high risk for care need. Therefore, screening for subjective cognitive impairment in this population might be valid for the early detection of dementia and other functional declines.

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Disclosure statement

None of the authors have personal or financial conflicts of interest with regard to this manuscript.

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