

<学 会 賞>

研究題目:(和)	糖尿病性骨減少症の病態生理学的研究		
(英)	Studies on Pathophysiological Aspects of Diabetic Osteopenia		
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研究業績要旨(1,000字以内)

全身性の代謝異常を生じる糖尿病では、慢性合併症の存在が患者の予後を決定する。そこで糖尿病に合併する可能性が指摘されていた骨・ミネラル系の代謝異常の病態ならびに成因について、基礎的および臨床的検討を加えた。

まず種々の糖尿病モデル動物を用いて、糖尿病状態下でのミネラル・ビタミンD代謝異常の存在を確認した。次いで日本人の糖尿病の大多数を占める2型糖尿病症例について検討を進め、骨塩量の低下とともにビタミンD代謝に異常が生じている事実を明らかにした(文献8-10)。そこでビタミンD代謝異常の成因を明らかにするために、2型糖尿病モデルのdb/dbマウスを用いて小腸ならびに腎のビタミンD受容体について検討したところ、これらの受容体数の減少と腎におけるビタミンD代謝の異常を認めた(文献7)。一方で骨代謝異常について、骨芽細胞機能の鋭敏なマーカーである血中osteocalcin(OC)を1型糖尿病モデルのSTZラットで測定したところ、対照に比し明らかな低下を認めた(文献6)。さらに2型糖尿病モデルのWistar fattyならびにNSZラットにも同様の血中OCの低下を認めた(文献5)ことから、骨形成の低下は1型と2型糖尿病に共通していることが示された。

次に破骨細胞機能に関して検討を進めたところ、Wistar fattyラットで骨吸収の亢進を示唆する成績を得た(文献4)。そこで2型糖尿病症例を対象に、骨芽ならびに破骨細胞機能を種々のマーカーを用いて検討することとした(文献1, 3)。その結果、骨芽細胞機能の低下と同時に、一方で破骨細胞機能の亢進を認めた。またこれらの細胞機能のuncoupling機構を追究するために、coupling factorの1つで破骨細胞形成に対して抑制的な作用を有するosteoprotegerin(OPG)に関する検討を加えたところ、血中OPGは糖尿病症例においてむしろ高値の傾向を示すとともに、破骨細胞機能との間に正の相関を、そして骨塩量との間に負の相関を認めた(文献1)。したがって糖尿病状態下では破骨細胞機能の亢進が骨量の減少に重要な役割を果たすとともに、OPGは二次的に上昇し骨吸収の亢進とさらなる骨量の減少に対して抑制的に作用することが示された。

国民の高齢化に伴い骨量の減少を有する糖尿病患者の増加が容易に予想されることから、本研究を基盤とした骨減少症の病因の解明や、治療(文献2)ならびに予防法の確立が求められている。

報文等リスト

(1) この研究に直接関連するもの(10編以内)

1. 10編を列挙致しました(文献1-10)。
2. そのうち、1編がJNSV掲載論文です(文献9)。

(2) その他の論文(編数制限なし)

1. 134編を列挙致しました(文献11-144)。
2. そのうち、3編がJNSV掲載論文です(文献66、75、125)。
3. 印刷中の1編については、掲載証明書を添付しました(文献11)。

(3) 過去5年間の本学会での活動状況

1. 2002年~2004年:理事、学会誌編集委員長
2. 2004年~現在 :各種授賞等選考委員会委員
3. 2006年~現在 :副会長

(4) 特記事項

なし

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