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Deficit di vitamina D nelle malattie ginecologiche

L'impiego della vitamina D nella malattia renale cronica

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EDITORIALE

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Cari Colleghi
in questo numero troverete aggiornamenti sul possibile ruolo della vitamina D nella malattia renale cronica e in alcune malattie ginecologiche.
Noterete che in entrambi gli articoli gli esperti Autori partono dall'evidenziare quanto sia comune il deficit di vitamina D anche in queste condizioni patologiche.
Nel caso della malattia renale cronica si attribuisce questo deficit al ridotto apporto nutrizionale secondario alle tipiche restrizioni dietetiche, ai frequenti disturbi gastrointestinali associati e alla ridotta esposizione solare secondaria alla disabilità.
Si fa inoltre notare che in questa condizione al deficit di vitamina D nativa si aggiunge la compromissione della sintesi di calcitriolo, cui conseguono alterazioni del metabolismo minerale e osseo (*Chronic Kidney Disease-Mineral Bone Disorder*, CKD-MBD) caratterizzato da uno stato di iperparatiroidismo secondario, inizialmente "adattattivo" ma successivamente "maladattativo" se non corretto con un'adeguata supplementazione vitaminica D.
Interessante è l'osservazione che anche in pazienti con malattia renale cronica avanzata, tale da dover ricorrere alla dialisi, la somministrazione di colecalciferolo si associa a un incremento della sintesi di calcitriolo, a dimostrazione di una produzione anche extrarenale di quest'ultimo, pure a livello delle stesse paratiroidi.
Anche se l'argomento è dibattuto, attualmente le linee guida suggeriscono di usare la supplementazione con la vitamina D nativa (colecalciferolo o ergocalciferolo) specie per prevenire l'insorgenza o la progressione dell'iperparatiroidismo, magari raggiungendo preferibilmente in questi pazienti livelli di 25(OH)D sierici ben al di sopra dei 30 ng/ml.
I metaboliti attivi della vitamina D andrebbero riservati agli stadi più avanzati di malattia renale cronica, quando sono presenti elevati livelli sierici di paratormone nonostante adeguati livelli di 25(OH)D; non va dimenticato che l'uso di questi metaboliti si può associare a ipercalcemia, iperfosforemia, alterazione dei livelli di FGF-23 ed eccessiva riduzione dei livelli di PTH tale da aumentare il rischio di osso adinamico.
Anche quando si dovessero usare i metaboliti attivi della vitamina D è saggio garantire comunque una supplementazione con la vitamina D nativa considerati i suoi effetti fisiologici extrarenali e i presunti benefici extrascheletrici.

Che ne dite ad esempio dei recettori, dei geni modulati dalla vitamina D e degli enzimi attivanti la vitamina D in diversi tessuti, tra cui quelli del tratto riproduttivo?
Avete notato quanto letteratura nuova ci sia sempre nel nostro consueto aggiornamento bibliografico in ambito ostetrico-ginecologico ?
Gli Autori dell'altro articolo di questo numero ci fanno notare che polimorfismi genetici del recettore specifico per la vitamina D (VDR) sono stati associati a livelli differenti di ormoni sessuali e che l'aggiunta di vitamina D a cellule della granulosa è in grado di aumentarne la sintesi. Ciò potrebbe giustificare le correlazioni osservate tra deficit di vitamina D e disturbi del ciclo me-

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struale o la sindrome dell'ovaio policistico, caratterizzata da oligo-anovulazione, segni clinici e/o biochimici di iperandrogenismo e morfologia policistica dell'ovaio.
Potrebbe anche giustificare gli effetti positivi osservati con la supplementazione, specie se giornaliera, di pazienti affetti da policistosi ovarica, in termini di infertilità e di correzione di alcune tipiche alterazioni metaboliche associate, tra cui iperinsulinismo, dislipidemia e stato infiammatorio cronico.
Buoni motivi per non trascurare la valutazione dello stato vitaminico D e l'eventuale opportunità di supplementazione anche in questi pazienti.
Cosa ne pensate?
Buona lettura

Deficit di vitamina D nelle malattie ginecologiche

VITAMIN D
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Negli ultimi anni il ruolo della vitamina D (VitD) come elemento importante nella fisiopatologia di malattie ginecologiche è andato crescendo, con dati di laboratorio che si intersecano con dati clinici nell'indicare quale sia il ruolo, o i possibili ruoli, che questa vitamina può avere in campo ginecologico.
La produzione e il metabolismo della VitD origina dallo stimolo esercitato dai raggi ultravioletti a livello cutaneo, con trasformazione del 7-deidrocolestreolo in colecalciferolo, il quale, a sua volta, viene metabolizzato, a livello del fegato, da una 25-idrossilasi. La 25(OH)D, a livello renale, viene trasformata da una 1alfa-idrossilasi, in $1,25(OH)_2D$ o calcitriolo, il metabolita attivo. Ancora a livello renale, a opera di una 24-idrossilasi, si forma la $1,24,25(OH)_3D$, che è un composto biologicamente non attivo (Fig. 1).
La vitamina D, che più propriamente dovrebbe essere indicata come ormone D, attraverso il suo recettore specifico (*Vitamin D Receptor*, VDR), è in grado di modulare l'attività di circa 3.000 geni disposti in differenti aree dell'organismo umano, compresi i tessuti del tratto riproduttivo femminile (ovaio, utero, vagina); i polimorfismi genetici del VDR sono stati associati a livelli differenti di ormone luteinizzante (LH), *Sex Hormone Binding Globulin* (SHBG), testosterone e insulina [1].
In particolare, per ciò che riguarda il tratto riproduttivo, la VitD può esercitare un controllo sullo sviluppo dei follicoli ovarici e sulla fase luteale, grazie a un'interazione con le vie del *signaling* dell'ormone anti-mülleriano (AMH) e della sensibilità all'ormone follicolo-stimolante (FSH) [2].
In maniera interessante, inoltre, è stato dimostrato come l'aggiunta di VitD a cellule della granulosa umana nel mezzo di coltura sia in grado di aumentare la produzione di alcuni ormoni fondamentale per l'attività ovarica, rispetto alla non aggiunta di VitD, come progesterone (in misura del 13%; $p < 0,001$), estradiolo (in misura del 9%; $p < 0,02$), estrone (in misura del 21%; $p < 0,002$), ancora grazie alla presenza del VDR in queste cellule, dove media tale attività di stimolo della VitD sull'attività ovarica [3].
Uno studio del 2018 [4], che ha valutato la relazione tra lo stato vitaminico D e il ciclo mestruale in donne senza diagnosi di sindrome dell'ovaio policistico (*Polycystic Ovary Syndrome*, PCOS), 60 con bassi livelli di VitD (< 30 ng/ml) e 17 con livelli normali di VitD (> 30 ng/ml ≤ 80 ng/ml), ha riportato come nel gruppo con bassi livelli di VitD ci fosse il 40% dei soggetti con cicli irregolari, il 27% con oligomenorrea e il 13% con amenorrea. Viceversa, nel Gruppo con livelli normali di VitD, solo il 12% delle donne presentavano disturbi del ciclo mestruale, il 6% con oligomenorrea e il 6% con amenorrea; inoltre, il fatto di appartenere al gruppo con VitD bassa aumentava di 5 volte la probabilità di avere un'irregolarità del ciclo mestruale rispetto all'appartenenza al gruppo con livelli normali di VitD [OR = 5; (IC 95%:

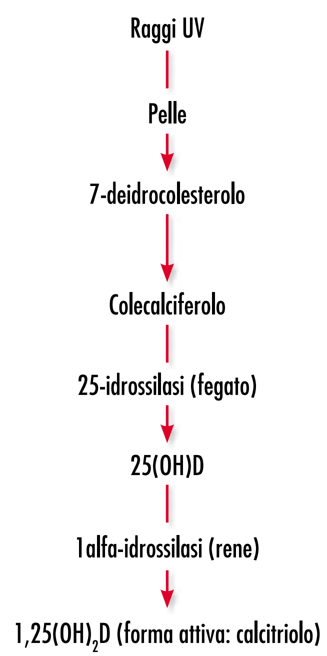

FIGURA 1.
Vitamina D: sintesi e metabolismo.

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Gli Autori dichiarano nessun conflitto di interessi.

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1,047-23,87), p = 0,04]. Quindi, anche nelle donne senza disturbi ormonali, la VitD può contribuire alla regolarità del ritmo del ciclo mestruale, attraverso la modulazione dell'attività dell'ovaio. Inoltre, è opportuno ricordare come sia presente il VDR a livello dell'endometrio [5] e come, sempre a livello endometriale, siano presenti atttvità enzimatiche, come la 1alfa-idrossilasi, fondamentali per il metabolismo della VitD e la produzione del suo metabolita attivo, la $1,25(OH)_2D$ o calcitriolo [6].

VITAMINA D E SINDROME DELL'OVAIO POLICISTICO

La PCOS è la patologia ormonale più frequente nel sesso femminile, ed è presente in circa le seguenti percentuali in associazione a differenti condizioni: 20% delle donne fertili sane, 75% delle donne con infertilità ovulatoria, 80% delle donne con oligomenorrea, 80% delle donne con ipertricosi e ciclo mestruale regolare, 30% delle donne con amenorrea secondaria, 80% delle donne con acne severa [7].

Volendo considerare le implicazioni della VitD in alcune condizioni fisiopatologiche della ginecologia, non si può non considerare la PCOS, che è il disturbo ormonale più frequente nel sesso femminile e la cui diagnosi si basa sul reperto di due dei seguenti tre parametri: oligo-anovulazione, segni clinici e/o biochimici di iperandrogenismo, morfologia policistica dell'ovaio all'esame ecografico. Oltre a ciò, deve essere ricordato, dal punto di vista fisiopatologico, il ruolo dell'iperinsulinemia come fattore di disregolazione dell'attività dell'ovaio e della produzione e azione degli ormoni androgeni [8]. Clinicamente, oltre ai disturbi del ciclo mestruale, le manifestazioni dermatologiche che si associano alla PCOS sono frequentemente il motivo di richiesta di consulto medico da parte delle pazienti affette. In particolare, la PCOS si può associare a seborrea (pelle grassa), acne, irsutismo, alopecia androgenetica. Oltre a ciò, l'iperinsulinemia, frequentemente associata alla PCOS, provoca una manifestazione caratteristica a livello della pelle come l'*Acanthosis Nigricans*, cioè la presenza, soprattutto a livello delle pieghe cutanee, di una pelle ispessita, vellutata, con un colorito tendente allo scuro.

Il deficit di VitD può influenzare la fertiltà nelle donne con PCOS; come ricordato precedentememte, i VDR sono presenti a vari livelli, come le cellule della granulosa dei follicoli ovarici, l'ipofisi e l'endometrio. Anche il gene *promoter* dell'AMH contiene degli elementi di risposta alla VitD [9].

Per ciò che riguarda l'associazione tra VitD e PCOS, è importante ricordare come livelli inferiori di VitD sono spesso riscontrati in pazienti con PCOS e come sia stata descritta un'associazione tra bassi livelli di VitD e insulino-resistenza (con conseguente iperinsulinemia) nella PCOS; infine, bassi livelli di VitD si riscontrano di frequente in pazienti PCOS obese [10,11].

In effetti, diversi studi hanno mostrato bassi livelli di VitD nella popolazione con PCOS, con un livello medio di 25(OH)D compresi tra 11 e 31 ng/ml, anche se la maggioranza delle pazienti presenta livelli < 20 ng/ml (67-85%) [10].

Un aspetto particolarmente interessante risulta essere il rapporto tra VitD e l'omeostasi glico-insulinica, che risulta essere basata sulla presenza di VDR a livello delle cellule beta del pancreas e del muscolo scheletrico, cellule nelle quali è presente l'enzima 1alfa-idrossilasi, che catalizza la conversione di 25(OH)D in $1,25(OH)_2D$; inoltre, sono presenti elementi di risposta per la VitD nel *promoter* del gene dell'insulina nel genere umano [12]. Innanzitutto, bisogna ricordare come livelli di calcio elevati a livello intracellulare possono alterare gli effetti post-recettoriali del legame dell'insulina al suo recettore, come la defosforilazione della glicogeno-sintetasi e l'attivazione del *Glucose Transporter-4* (GLUT-4). Quindi, il deficit di VitD potrebbe comportare un aumento secondario dei livelli di paratormone (iperparatiroidismo secondario), con aumento dei livelli intracellulari di calcio, così riducendo la risposta delle cellule bersaglio dell'azione dell'insulina (trasporto del glucosio). La prevalenza del deficit di VitD nelle pazienti PCOS è circa del 67-85%, con livelli sierici di 25(OH)D < 20 ng/ml [13]; in tal senso, le conseguenze endocrino-metaboliche del deficit di VitD possono essere importanti nella patogenesi della PCOS, così come nella sua espressività clinica (Fig. 2).

Per ciò che riguarda il rapporto tra livelli di VitD e profilo metabolico nella PCOS, livelli ridotti di VitD si associano a insulino-resistenza, indipendentemente dall'indice di massa corporea (BMI) o dal rapporto vita/fianchi (*Wiast/Hip Ratio*, WHR) nelle donne con PCOS; interessante ricordare come via sia un aumento dei livelli di insulina in donne senza PCOS ma con deficit di VitD; il colesterolo HDL (lipoproteine ad alta densità) si correla positivamente con i livelli di VitD indipendentemente dal BMI o dal WHR [14].

Uno studio cross-sezionale ha esplorato l'associazione tra stato della VitD e la diagnosi di disturbi ovulatori/PCOS in una popolazione di 67 donne infertili in buona salute generale; come risultato, valori ridotti di VitD (normalizzati per altri fattori confondenti) sono stati evidenziati in donne con disturbo ovulatorio e PCOS in confronto alle donne con infertiltià da altra causa; peraltro, ogni unità di aumento dei livelli di VitD (normalizzata per il BMI) riduceva la probabilità di avere una diagnosi di PCOS del 96% (p = 0,015); nessuna paziente sia con disturbo ovulatorio sia con PCOS aveva livelli normali di VitD: il 39% delle donne con disturbi ovulatori e il 38% con PCOS avevano livelli sierici <15 ng/ml che indicavano deficit della vitamina [15].

Dal punto di vista dell'impatto metabolico, lo stato della VitD si correla con i marker di insulino-resistenza nella PCOS (correlazione tra deficit di VitD e HOMA-IR con p = 0,0001 e con glicemia a digiuno p = 0,047) [16].

Uno studio cross-sezionale cinese [17] condotto su 169 donne con PCOS e 114 controlli, ha riscontrato livelli inferiori di VitD nelle pazienti PCOS rispetto ai controlli (11,6 ± 7,2 *vs* 18,9 ± 8,4 ng/mL; p < 0,05) e livelli inferiori di VitD in pazienti PCOS con obesità o insulino-resistenza rispetto alle donne senza obesità o insulino-rersistenza (8,9 ± 3,7 *vs* 13,6 ± 5,3 ng/mL, p < 0,05; 7,2 ± 2,9 *vs* 15,8 ± 4,9 ng/mL, p < 0,01); anche altri parametri metabolici e infiammatori aveno una correlazione importante con i livelli di VitD basali (Tab. I).

Il primo studio, che ha valutato l'effetto della supplementazione di VitD nella gestione della PCOS, è stato effettuato da Thys-Jacobs et al. nel 1999; in questo studio, 13 donne con PCOS sono state trattate con 50.000 UI di ergocalciferolo a settimana o ogni 2 settimane per ottenere un livello di VitD sierico di 75-100 nmol/l; entro 2 mesi venne riportato un miglioramento della regolarità mestruale [18].

In uno studio del 2012 [19], 12 donne con PCOS in sovrappeso e con deficit di VitD sono state supplementate per 3 mesi con VitD (dose giornaliera di 3.533 unità, aumentata a 8.533 unità dopo le prime 5 partecipanti) e 530 mg di calcio; dopo 3 mesi si è riscontrata una riduzione dei livelli di testosterone totale (p = 0,036) e androstene-

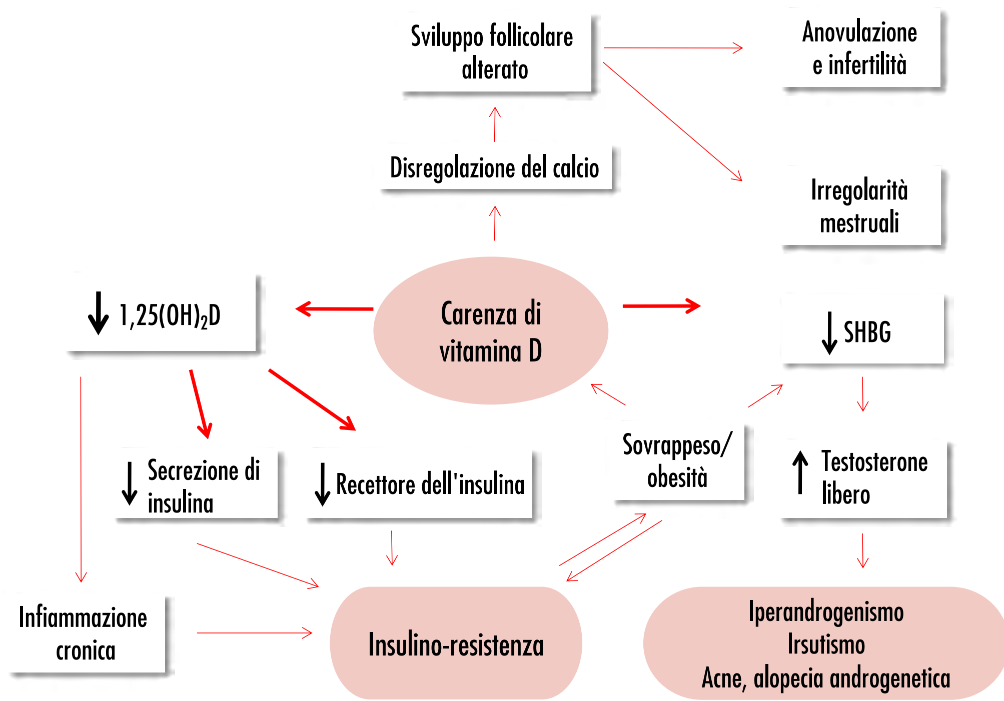

FIGURA 2.
Possibile ruolo della vitamina D nella patogenesi della PCOS (da Thomson et al., 2012, mod.) [13].

dione (seppure non in maniera significativa). Un trial randomizzato e controllato con placebo [20] condotto su 70 donne con PCOS e deficit di VitD (< 20 ng/ml) (età compresa tra 18 e 40 anni), ha studiato 2 gruppi di pazienti: uno trattato con 50.000 unità di VitD ogni 2 settimane per 3 mesi e uno con placebo; i risultati hanno mostrato una differenza statisticamente significativa nei livelli di glicemia a digiuno (-3,1 ± 7,3 *vs* +0,5 ± 6,3 mg/dl, p = 0,02), nei livelli di insulina basali (-1,4 ± 3,6 *vs* +2,6 ± 7,0 µIU/ml, p = 0,004) e nei livelli di HOMA-IR (-0,3 ± 0,8 *vs* + 0,6 ± 1,6, p = 0,003); inoltre, anche i livelli di hs-CRP risultavano significativamente più bassi (-0,7 ± 1,4 *vs* +0,5 ± 2,1 µg/ml; p = 0,009), così come i livelli di malondialdeide (-0,1 ± 0,5 *vs* +0,9 ± 2,1 µmol/l, p = 0,01).

Una metanalisi, del 2020, pubblicata da Miao et al. [21], ha preso in considerazione 11 studi (= 483 soggetti); degli 11 studi presi in considerazione, 7 riportavano tra i criteri di inclusione diagnosi di PCOS e deficit di VitD. Tale metanalisi ha mostrato come la supplementazione con VitD sia associata a riduzione del testosterone totale (differenza media: -0,10; IC 95%: -0,18, -0,02; p = 0,02), riduzione dell'HOMA-IR (differenza media: -0,44, IC 95%: -0,86, -0,03, p = 0,04), riduzione dei livelli del colesterolo totale (differenza media:-11,90, IC 95%: -15,67, 8,13, p < 0,01), riduzione dei livelli di colesterolo LDL (differenza media: -4,54, IC 95%: -7,29, -1,80, p = 0,001).

In una metanalisi pubblicata nel 2021, condotta prendendo in considerazione 18 trial randomizzati e controllati con placebo (= 1.060 soggetti, tutti con valori medi di VitD al baseline < 30 ng/ml), Zhao et al. [22] hanno mostrato come la supplementazione con VitD aveva un impatto positivo sul profilo di tipo ormonale, ossidativo e infiammatorio nella PCOS; infatti, si riscontrava una riduzione dei livelli di testosterone totale (IC 95%: -0,40, -0,07; p = 0,006), riduzione dei livelli di proteina C-reattiva ad alta sensibilità (hs-CRP) (IC 95%: -0,73, -0,38; p < 0,00001), riduzione dei livelli di malondialdeide (IC 95%: -0,90, -0,54; p < 0,0001), aumen-

TABELLA I.
Status della vitamina D e fattori metabolici in PCOS (Wang et al., 2020, mod.) [17].

	25(OH)D < 20 ng/ml (deficienza)	25(OH)D ≥ 20 ≤ 30 ng/ml (insufficienza)	25(OH)D > 30 ng/ml (valore normale)	p*
BMI	27,3 ± 9,2	25,4 ± 8,1	23,5 ± 9,3	0,029
WHR	1,0 ± 0,4	0,9 ± 0,5	0,8 ± 0,3	0,036
Insulina (mIU/L)	39,6 ± 10,7	33,5 ± 9,9	26,8 ± 8,5	0,012
HOMA-IR	8,9 ± 3,7	7,3 ± 2,8	5,7 ± 2,1	0,009
Colesterolo totale (mmol/L)	6,1 ± 1,7	5,5 ± 1,6	4,2 ± 1,4	0,03
hs-CRP (mg/L)	2,4 ± 0,9	1,9 ± 0,6	1,4 ± 0,3	0,017
HDL (mmol/L)	1,3 ± 0,6	1,4 ± 0,7	1,8 ± 0,6	0,03

* Analisi della varianza. BMI: indice di massa corporea; WHR: *Wiast/Hip Ratio*; HOMA-IR: *Homeostatic Model Assessment for Insulin Resistance*; hs-CRP: proteina C-reattiva ad alta sensibilità; HDL: lipoproteine ad alta densità.

to dei livelli di capacità antiossidante totale (IC 95%: 0,01, 0,83; p = 0,04). Ancora in questa metanalisi, si dimostra come lo schema di supplementazione più adeguata per ottenere questi risultati sia quello giornaliero con dosi ≤ 1.000 U/die, che risulta essere migliore della somministrazione settimanale, con una durata adeguata che appare essere di almeno 12 settimane.

Una recente *review* sistematica con metanalisi [23], condotta considerando 9 RCT (studi controllati randomizzati) (n = 1677) e 3 studi di coorte (n= 675), su pazienti infertili con deficit di VitD, ha valutato l'influenza della supplementazione con VitD sull'*outcome* riproduttivo, partendo dal dato che un basso livello di VitD si associa a un aumentato rischio di infertilità; ebbene, il trattamento con VitD ha aumentato significativamente il tasso di gravidanza clinica in confronto al gruppo di controllo (OR: 1,70, IC 95%: 1,24-2,34; p = 0,001); Il miglioramento del tasso di gravidanza era influenzato dal livello di VitD delle pazienti, dal tipo di preparazione somministrata, dal dosaggio totale somministrato, dalla durata del trattamento, dalla frequenza di somministrazione, e dalla somministrazione giornaliera della supplementazione di VitD. Le donne infertili (con livelli di VitD < 30 ng/ml) trattate con preparazioni mutlicomponenti con VitD o con VitD 1.000-10.000 unità al giorno per 30-60 giorni potevano avere un migliore outcome gravidico.

CONCLUSIONI

La VitD svolge un ruolo fisiologico per la funzione riproduttiva femminile. In particolare, è importante mantenere uno stato vitaminico D adeguato, sia in condizioni fisiologiche normali, sia nelle donne affette da patologie ginecologiche (ad es., nella PCOS). La valutazione dello stato vitaminico D nella salute della donna e, se necessaria, la supplementazione possono essere molto importanti nella pratica clinica.

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L'impiego della vitamina D nella malattia renale cronica

VITAMIN D
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INTRODUZIONE

Il termine "vitamina D" indica un gruppo di composti steroidei, liposolubili, fondamentali per la regolazione del metabolismo del calcio e del fosforo, mediato principalmente attraverso l'assorbimento intestinale [1].

Le due isoforme più importanti, indicate cumulativamente come "vitamine D native", sono: l'ergocalciferolo (vitamina D_2) e il colecalciferolo (vitamina D_3). L'ergocalciferolo viene introdotto attraverso la dieta ed è sintetizzato solo dalle piante e dai funghi, il colecalciferolo, invece, è una molecola di sintesi sia esogena che endogena e deriva dalla fotolisi del 7-deidrocolesterolo, mediata dalle radiazioni UVB che colpiscono la pelle [1].

L'ergocalciferolo e il colecalciferolo rappresentano le due forme inattive di vitamina D; la loro trasformazione nella forma biologicamente attiva, il calcitriolo [$1,25(OH)_2D$], richiede un processo di idrossilazione che ha luogo in due fasi successive. Il primo step avviene a livello epatico: qui le vitamine D_2 e D_3 vengono idrossilate a livello della posizione C25 da parte della vitamina D 25-idrossilasi e convertite in 25-idrossi-vitamina D [25(OH)D o calcifediolo], la forma quantificabile utilizzata principalmente per determinare i livelli di vitamina D nel siero. Il secondo step avviene a livello del tubulo prossimale renale mediante l'1alfa-idrossilasi; qui la 25(OH)D viene idrossilata a livello di C1 formando la 1,25-diidrossi-vitamina D, nota anche come $1,25(OH)_2D$ o calcitriolo [1]. Tuttavia è anche noto che l'attività 1alfa-idrossilasica (che rappresenta la capacità di produrre 1,25-diidrossivitamina D) è presente anche nei macrofagi attivati, negli osteoblasti, nei cheratinociti ed è stata documentata anche a livello prostatico, nel colon e nella mammella ed è in grado di attivare forme nutrizionali e proormonali della vitamina D.

La $1,25(OH)_2D$ è la forma "attiva" di vitamina D. La sua quantificazione sierica, seppur importante in alcune patologie, fornisce poche informazioni sullo stato della vitamina D ed è solitamente normale o addirittura elevata quando l'iperparatiroidismo si associa all'ipovitaminosi D [1].

L'$1,25(OH)_2D$ viene veicolata in circolo tramite una proteina circolante legante la vitamina D (VDBP) e, raggiunti gli organi bersaglio, si lega al recettore della vitamina D (VDR). Il VDR appartiene a un ampio gruppo di fattori di trascrizione nucleare attivati dal ligando, e può vantare un'espressione pressoché ubiquitaria e tessuto-dipendente nelle cellule nucleate. Questo spiega come la vitamina D, oltre a regolare l'assorbimento intestinale e la mobilizzazione del calcio e del fosforo, esercita anche diverse funzioni che esulano dagli effetti propriamente osteogenici e inerenti al metabolismo minerale. I suoi effetti sono mediati dagli elementi responsivi alla vitamina D (VDRE) e portano a cambiamenti nell'espressione di diversi geni [2] (Fig. 1).

L'integrità morfologica e funzionale del tessuto osseo riflette la regolazione e il mantenimento del rimodellamento osseo. Quest'ultimo è l'espressione dell'attività sia degli osteoblasti, che controllano la neoformazione ossea, che degli osteoclasti, i quali hanno la capacità di riassorbire l'osso mineralizzato: tale attività è modulata dagli osteoblasti attraverso il sistema RANK-RANKL-OPG.

Il RANK ligando, secreto dagli osteoblasti, si lega a un recettore (RANK) presente sulla superficie dei pre-osteoclasti, stimolandone la differenziazione in osteoclasti attivi (maturi), mentre l'OPG, anch'essa secreta dagli osteoblasti, impedisce il legame di RANK ligando al suo recettore, inibendo quindi l'attivazione osteoclastica.

Queste complesse interazioni vengono regolate da ormoni locali e sistemici come PTH, vie di segnalazione del sistema di Wnt, FGF23 e appunto $1,25(OH)_2D$, la quale svolge un ruolo fondamentale nella regolazione del *bone remodelling* [3].

Corrispondenza
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Conflitto di interessi
Gli Autori dichiarano nessun conflitto di interessi.

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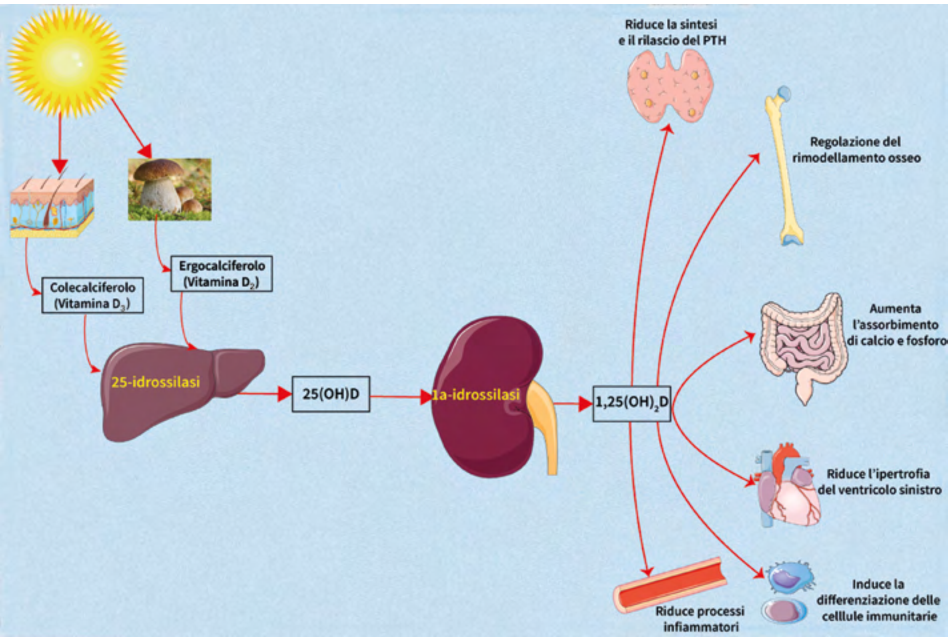

FIGURA 1.
Metabolismo della vitamina D e suoi effetti.

Il principale effetto "endocrino" che fa seguito all'attivazione del recettore della vitamina D (VDR) è la regolazione dell'omeostasi minerale e ossea. L'attivazione del VDR controlla l'assorbimento di calcio e fosfato a livello intestinale, il riassorbimento tubulare del calcio a livello renale e l'attività e la vitalità delle cellule ossee. A livello degli osteoblasti la $1,25(OH)_2D$ è in grado di aumentare l'espressione del fattore 2 di trascrizione correlato a Runt (RUNX2), di osterix (OSX) e della fosfatasi alcalina, molecole coinvolte a vario titolo nella differenziazione osteoblastica e nel processo di mineralizzazione. Inoltre, il *Wingless-type* (*Wnt-beta-catenin pathway*) è un importante regolatore della differenziazione e della funzione degli osteoblasti, la cui espressione è aumentata dall'$1,25(OH)_2D$ [4].
Il calcitriolo, oltre a stimolare la formazione ossea, promuove anche il riassorbimento osseo aumentando il numero e l'attività degli osteoclasti. Gli effetti possono essere mediati dai VDR e dall'alfa-idrossilasi, espressi anche a livello degli osteoclasti, dal *macrophage colony-stimulating factor* (m-CSF) e dell'attivatore del recettore del ligando del fattore nucleare-κB (RANKL) [5].

LA VITAMINA D NELLA MALATTIA RENALE CRONICA

Il deficit di vitamina D nativa è estremamente comune nei pazienti affetti da malattia renale cronica (MRC), ed è riconducibile a diverse condizioni, come il ridotto apporto nutrizionale secondario alle restrizioni dietetiche cui è sottoposto il paziente nefropatico (dieta a basso contenuto proteico e a basso contenuto di fosfati), riduzione dell'appetito e sintomi gastrointestinali, ridotta esposizione agli UVB legata alla ridotta mobilità e alle ospedalizzazioni frequenti [6].Il progressivo declino dell'eGFR è associato a un aumento nella prevalenza del deficit di vitamina D. Uno studio *cross-sectional* su 825 pazienti in dialisi ha mostrato che il 78% dei pazienti aveva un deficit di vitamina D con valori < 30 ng/ml e che il 18% dei pazienti aveva un deficit severo con valori < 10 ng/ml. Lo studio ha inoltre dimostrato che i bassi valori di vitamina D si associavano a un aumentato rischio di mortalità precoce [7]. Nella MRC, oltre a un deficit di vitamina D nativa, è presente anche una ridotta sintesi di calcitriolo; infatti, la progressiva perdita della funzione renale si associa a una ridotta attività dell'1alfa-idrossilasi e a una conseguente ridotta produzione di $1,25(OH)_2D$ [2]. Nella MRC l'ipovitaminosi D deve essere inquadrata in un contesto più ampio in quanto è alla base (sebbene non rappresenti l'unico fattore causale) delle alterazioni del calcio, del fosforo e del PTH. All'insorgenza di tali alterazioni fa seguito lo sviluppo dell'iperparatiroidismo secondario, quadro clinico e laboratoristico peculiare della MRC. Inoltre, in questi pazienti l'alterata omeostasi del metabolismo minerale non ha solo un impatto sul sistema scheletrico, ma è anche strettamente associata ad altre alterazioni importanti, come lo sviluppo di calcificazioni vascolari e, soprattutto, la progressione delle malattie cardiovascolari [4].

LA VITAMINA D NEI DISORDINI DEL METABOLISMO MINERALE INDOTTI DALLA MALATTIA RENALE CRONICA

La MRC è strettamente associata alla presenza di alterazioni del metabolismo osseo che comprendono una disregolazione del metabolismo del calcio, del fosforo, nonché dell'asse fisiopatologico rappresentato da vitamina D-PTH-FGF23. Nel 2006, le linee guida KDIGO (*Kidney Disease Improving Global Outcomes*) hanno coniato la definizione di CKD-MBD (*Chronic Kidney Disease-Mineral Bone Disorder*) per descrivere le alterazioni del metabolismo minerale e le patologie che ne conseguono, come i disordini a livello osseo e cardiovascolare, associati a un maggior rischio fratturativo e cardiovascolare [8]. Queste alterazioni sono già presenti in circa il 40-80% dei pazienti con CKD negli stadi 3 o 4 [9].
Sebbene l'esatta sequenza cronologica degli step fisiopatologici non sia completamente nota, si ritiene che l'incremento dei livelli di fosfato sierico, conseguente alla ridotta funzione renale, stimoli la sintesi e il rilascio da parte degli osteoblasti e degli osteociti del *fibroblast growth factor 23* (FGF23) che, se da un lato inibisce la sintesi di PTH, dall'altro inibisce anche l'1alfa-idrossilasi renale con conseguente riduzione dei valori di calcitriolo e aumento della sintesi di PTH. La costante stimolazione delle cellule paratiroide e la mancata correzione dei fattori modificabili, come la carenza di vitamina D e l'iperfosfatemia, induce una risposta inizialmente "adattativa" e successivamente "maladattativa", se non corretta da un adeguato intervento dietetico e farmacologico, che si caratterizza per un'iperplasia policlonale delle cellule paratiroidee. La transizione dell'iperplasia policlonale alla forma "nodulare" dell'iperplasia, determina un'ulteriore progressione dell'iperparatiroidismo secondario, caratterizzata a livello paratiroideo da una serie di adattamenti morfologici e funzionali (ridotta espressione del VDR), che rendono il quadro scarsamente responsivo alla terapia farmacologica e per il quale si rende necessario il ricorso alla terapia chirurgica (paratiroidectomia) [10].

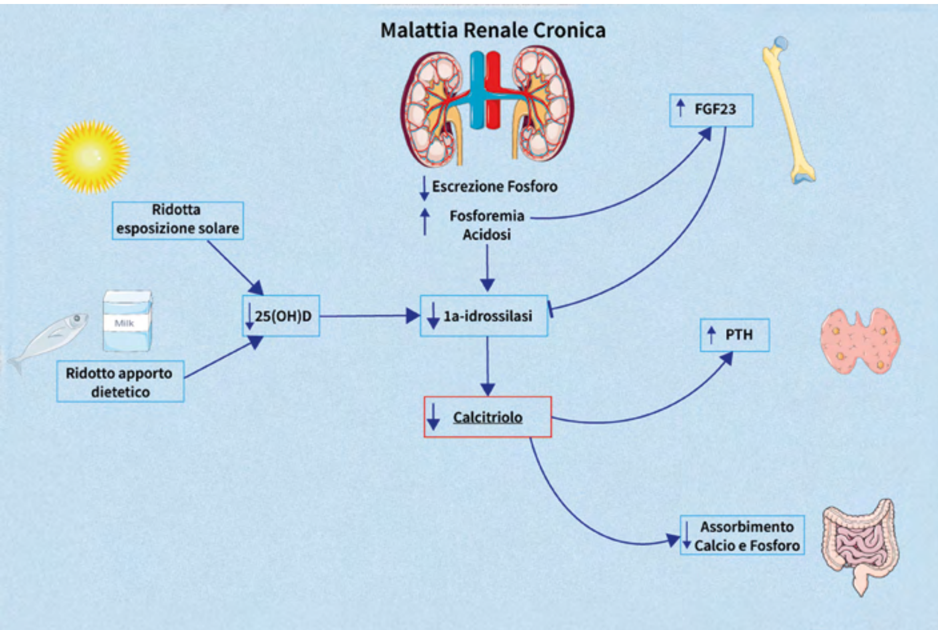

FIGURA 2.
Vitamina D e disordini del metabolismo minerale nella malattia renale cronica.

La vitamina D svolge quindi un ruolo fondamentale nella genesi e nella progressione dell'iperparatiroidismo secondario; infatti, concentrazioni fisiologiche di 1,25(OH)D hanno effetti inibitori sulla trascrizione del PTH. Inoltre, a fronte di una bassa affinità per il VDR, è stato dimostrato che elevati livelli sierici di 25(OH)D sono in grado di attivare il VDR, imitando così l'effetto della 1,25(OH)$_2$D. Inoltre, l'1alfa-idrossilasi, enzima chiave nella conversione del calcifediolo in calcitriolo, è presente nelle ghiandole paratiroidee e in molti altri tessuti extrarenali, presumibilmente per la produzione locale dell'ormone.
A questo proposito, è stato dimostrato che i livelli sierici di 25(OH)D e 1,25(OH)$_2$D aumentano in risposta alla somministrazione di vitamina D nutrizionale (colecalciferolo ed ergocalciferolo) nei pazienti dializzati, il che suggerisce che anche nella MRC sia presente un'attività della 1alfa-idrossilasi in tessuti extrarenali che, in presenza di livelli elevati di 25-idrossivitamina D, risulta in grado di consentire una produzione extrarenale sufficiente di 1,25-diidrossivitamina D per il controllo del PTH.
Poiché le ghiandole paratiroidi esprimono 1alfa-idrossilasi, dovrebbe essere preso in considerazione un possibile meccanismo autocrino mediante il quale la supplementazione di vitamina D nutrizionale sia in grado di ridurre la produzione di PTH.
Le più recenti linee guida KDIGO (2017) per la gestione della CKD-MBD affermano l'importanza di monitorare i livelli sierici di calcio, fosfato e PTH all'inizio della CKD stadio G3a e di valutarne l'andamento nel tempo, oltre a suggerire la misurazione livelli di 25(OH)D per diagnosticare la carenza di vitamina D (Fig. 2) [8].
In merito ai valori di vitamina D, nella popolazione generale, viene fatto riferimento alle raccomandazioni dell'*Endocrine Society* che stabiliscono la carenza con concentrazioni di 25(OH)D < 20 ng/mL, l'insufficienza con concentrazioni comprese tra 21 e 29 ng/mL e la normalità o sufficienza con livelli sierici > 30 ng/mL [12]. Per quanto riguarda la popolazione con MRC, negli anni sono state formulate diverse linee guida che hanno posto diverse indicazioni in merito alla diagnosi e al trattamento dell'ipovitaminosi D.
Le indicazioni più recenti, effettuate dalla *National Kidney Foundation*, hanno stabilito che concentrazioni di 25(OH)D > 20 ng/mL possono essere considerate "adeguate", mentre concentrazioni <15 ng/ml dovrebbero essere trattate. Per livelli di 25(OH)D compresi tra 15 e 20 ng/mL bisognerebbe considerare anche i livelli di PTH e l'attività controregolatoria della vitamina D su questo ormone [13].
La supplementazione della vitamina D è ancora un argomento dibattuto nei pazienti con MRC. Le linee guida KDIGO suggeriscono di effettuare la supplementazione con la vitamina D nutrizionale (colecalciferolo ed ergocalciferolo) come per la popolazione generale, al fine di migliorare lo stato carenziale e prevenire l'insorgenza e la progressione dell'iperparatiroidismo secondario [8]. Attualmente non sono però disponibili studi conclusivi sull'effetto della supplementazione di vitamina D nativa sui valori di PTH, sebbene quelli attualmente disponibili non mostrino alterazioni dei valori di calcio e fosforo o eventi avversi. È stato ipotizzato che l'integrazione con la vitamina D nutrizionale tenda a essere più efficace nel prevenire l'insorgenza/progressione dell'iperparatiroidismo piuttosto che a ridurre effettivamente i valori di PTH quando questi sono già elevati nelle fasi avanzate di malattia.
L'iperparatiroidismo secondario è un processo che si instaura lentamente fin dalle fasi più precoci della MRC (fase conservativa), prevenirne l'insorgenza e/o la progressione, correggendo il deficit di vitamina D con una precoce e congrua supplementazione di vitamina D nutrizionale, potrebbe ridurre gli effetti negativi dell'iperparatiroidismo secondario sul *bone remodelling* [14] e può ridurre il rischio di avere livelli di PTH al di sopra degli intervalli target raccomandati dal KDIGO e la necessità di maggiori prescrizioni di farmaci durante la successiva fase dialitica [15].
Inoltre, è verosimile, alla luce di studi pre-clinici e clinici, che nel contesto fisiopatologico della MRC l'azione antagonizzante della vitamina nutrizionale sull'insorgenza dell'iperparatiroidismo secondario si esplichi in presenza di livelli sierici più elevati di 25(OH) [> 40 ng/ml] rispetto a quelli ritenuti "efficaci" nella popolazione generale [16]. Il che suggerisce che in un contesto fisiopatologico specifico, quale è quello della MRC, i livelli di 25(OH)D attualmente raccomandati (> 30 ng/mL) potrebbero essere inefficaci/insufficienti per il trattamento dell'SPHT.
Sia le linee guida KDIGO che le raccomandazioni della *National Kidney Foundation* suggeriscono che bisogna preferire prima l'integrazione con vitamina D nutrizionale (ergocalciferolo, colecalciferolo) e solo successivamente introdurre i composti attivi della vitamina D (attivatori del recettore della vitamina D: VDRA), riservando quest'ultimi agli stadi più avanzati di MRC e a casi di iperparatiroidismo severo non controllabili dalla sola vitamina D nutrizionale. La tera-

pia con i VDRA, inoltre, dovrebbe essere intrapresa quando lo stadio di MRC è avanzato, quando sono presenti elevati valori di PTH associati ad adeguati livelli di 25(OH)D e in assenza di elevati valori di calcemia o fosforemia [8,17]. Infatti, i VDRA dovrebbero essere utilizzati con cautela in quanto sono stati segnalati casi di ipercalcemia e iperfosforemia, inoltre la loro capacità di indurre un'eccessiva riduzione di PTH può aumentare il rischio di malattia ossea adinamica e bisogna sempre tenere in considerazione l'aumento, che di per sé rappresenta un effetto negativo dei livelli di FGF-23 [17].

In conclusione, la vitamina D svolge un ruolo fondamentale nella MRC considerando che, alla luce dell'ubiquità dei recettori della vitamina D, il suo ruolo è fondamentale per l'omeostasi dell'organismo in generale e la sua azione non può essere ridotta al solo metabolismo osseo. L'ipovitaminosi D deve quindi essere prontamente diagnosticata e trattata ancora di più nei pazienti con MRC alla luce dell'importante impatto sull'iperparatiroidismo e sulla regolazione del metabolismo osseo.

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