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 Editoriale

 Ruolo
immunomodulante
della vitamina D
nella malattia celiaca

 La vitamina D
e il COVID-19:
un raggio di sole
nella tempesta?

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bibliografica

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Carissimi,

ci mancava vero un contributo in ambito gastroenterologico che coinvolgesse la vitamina D e quindi eccolo in questo numero, in particolare sul tema della malattia celiaca, grazie alla disponibilità di esperti colleghi Gastroenterologi. Come vedrete il legame tra malattia celiaca e vitamina D è a doppio senso in quanto, se da una parte le lesioni intestinali possono portare a un malassorbimento di vitamina D con le note conseguenze negative sull'osso, dall'altra la carenza di vitamina D si associa a un'abnorme risposta infiammatoria che, almeno potenzialmente, potrebbe favorire l'esordio e il mantenimento dell'enteropatia stessa. In effetti gli Autori riportano evidenze dalla letteratura che inducono a ritenere che la vitamina D possa giocare un ruolo nella patogenesi della malattia celiaca, sia attraverso un effetto protettivo diretto sulla barriera intestinale, sia modulando la risposta immunitaria a favore dei meccanismi di tolleranza. Di nuovo in particolare anche in questo campo ritroviamo il razionale per un effetto protettivo della vitamina D nei confronti della "cascata citochinica", risposta infiammatoria che se eccessiva può fare gravi danni (da cosiddetto "fuoco amico"), come ipotizzato anche in corso di COVID-19¹. A questo proposito abbiamo recentemente ipotizzato² che anche il progressivo uso di aminobisfosfonati, riducendo nel lungo termine il livello di linfociti T $\gamma\delta$ circolanti e quindi la cascata citochinica responsabile della reazione di fase acuta, possa giustificare la riduzione osservata di polmoniti e di mortalità da polmoniti in pazienti trattati con questi farmaci³.

Anche considerata la seconda ondata in corso della pandemia da SARS-Cov-2 non potevamo esimerci dal fare il punto per i nostri lettori sulle conoscenze relative al possibile ruolo dello stato vitaminico D nei confronti della malattia COVID-19, essendo noti e riconosciuti i suoi molteplici effetti immunomodulatori e ricchissima la recente letteratura in merito, che ci ha costretto anche in questo numero a una selezione bibliografica dedicata. Mi pare che si possano condividere le fondate e prudenti conclusioni degli Autori ai quali abbiamo affidato questo compito, secondo i quali i dati a disposizione rendono credibile un legame tra carenza di vitamina D e suscettibilità e severità della infezione da SARS-Cov-2.

Nella recente esperienza da noi condotta in occasione della precedente ondata pandemica abbiamo studiato la prevalenza della carenza di 25(OH)D tra pazienti ricoverati per COVID-19, cercando in particolare le associazioni tra lo stato vitaminico D e la gravità della malattia⁴. Tra i 61 pazienti inclusi nello studio, il 72,1% era carente di 25(OH)D (<20 ng/mL) e il 57,4% aveva 25(OH)D <15 ng/mL. La carenza di vitamina D era associata a un rischio aumentato di avere PO₂ arteriosa < 60 mmHg, a un aumento di 3 volte del rischio di avere PCR alterata e a un incremento del rischio di presentare dispnea all'esordio della malattia. Tuttavia, come sapete, trattandosi di uno studio osservazionale retrospettivo non possiamo con questi risultati attribuire alla carenza di vitamina D un ruolo causale, anche in considerazione dei noti effetti della flogosi, specie se intensa, sui livelli sierici di 25(OH)D^{5,6}. Solo i risultati di trial clinici che prevedano la supplementazione con vitamina D potranno darci risposte certe a questo riguardo, ma credo che comunque nel frattempo sia saggio non trovarsi a rischio di carenza di vitamina D.

Cosa ne pensate?

Statemi bene

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Ruolo immunomodulante della vitamina D nella malattia celiaca

VITAMIN D

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Abstract

Del tutto recentemente stanno emergendo gli effetti extra-scheletrici della vitamina D soprattutto nel mantenimento dell'omeostasi immunologica e della barriera intestinale. Di conseguenza, appare plausibile l'ipotesi di un suo coinvolgimento nella patogenesi di molte condizioni immuno-mediate, tra cui la malattia celiaca. Questa è una malattia infiammatoria cronica che ha l'intestino tenue come organo bersaglio ed è scatenata dall'ingestione del glutine contenuto in alcuni cereali da parte di soggetti geneticamente predisposti. La malattia celiaca è la più frequente malattia non trasmissibile nel mondo in quanto si stima che la sua prevalenza oscilli tra 0,5-1%. Pur tuttavia, nonostante l'elevata se non assoluta attendibilità dei test diagnostici, la sua reale prevalenza è di gran lunga inferiore, circa 1‰, in quanto la poliedricità del quadro clinico e, soprattutto, la paucisintomaticità della gran parte dei casi, unitamente alla scarsa conoscenza di tale condizione, contribuiscono al fenomeno cosiddetto dell'"iceberg", in cui i casi diagnosticati rappresentano solo la punta del totale. Comunque, fino a oggi, la valutazione del tasso sierico di vitamina D è fortemente consigliata sia nelle forme pediatriche, sia in quelle dell'adulto di malattia celiaca, in quanto l'enteropatia di per sé, e l'eventuale contaminazione batterica del tenue che ne consegue, possono condurre a un malassorbimento di vitamina D, con ovvie ripercussioni a livello osseo. Da pochissimi anni, stanno crescendo le dimostrazioni di un suo ruolo immuno-modulante che si espletterebbe su tutte le popolazioni cellulari coinvolte nella risposta immunitaria. Inoltre, tale vitamina appare svolgere un'azione sia protettiva nei confronti della barriera intestinale, sia di regolazione dell'enterocinesi. Una sua carenza, pertanto, parrebbe rappresentare uno di quei fattori ambientali che, unitamente al glutine e alla suscettibilità genetica, sono necessari per l'innescamento e il mantenimento delle lesioni intestinali in questa condizione patologica.

INTRODUZIONE

Del tutto recentemente si sta facendo strada l'affascinante idea di un ruolo della vitamina D diverso e non meno importante di quello svolto nel metabolismo osseo e legato alla regolazione della risposta immunitaria e della barriera intestinale¹. Sta emergendo, infatti, il legame tra patrimonio di vitamina D e malattie immuno-mediate, quali la sclerosi multipla, il diabete tipo 1, l'artrite reumatoide, e il lupus eritematoso sistemico^{2,3}, per citarne alcune. Queste rappresentano la vera emergenza sanitaria dei Paesi Occidentali e di quelli in via di sviluppo in quanto la loro prevalenza è in continuo aumento ed è responsabile di elevati costi diretti, ma anche indiretti legati alla ridu-

zione della qualità di vita e all'invalidità di una porzione rilevante della popolazione, soprattutto in età produttiva⁴. Tra queste, le malattie infiammatorie croniche intestinali sono scatenate dalla complessa interazione tra fattori genetici, immunologici e ambientali che rende anche ragione della loro variabilità clinica⁵. La malattia celiaca (MC) è la più frequente enteropatia nel mondo, in quanto la sua prevalenza si aggira intorno a 0,5-1%⁶, anche se una larga proporzione di casi rimane non diagnosticata⁷. Anche la sua incidenza sta criticamente aumentando non solo per una migliore capacità diagnostica, ma anche per la pressione data da fattori ambientali⁸. Il legame tra MC e vitamina D è a doppio senso in quanto, se da una

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Conflitto di interessi

Rachele Ciccocioppo e Luca Frulloni dichiarano nessun conflitto di interesse.

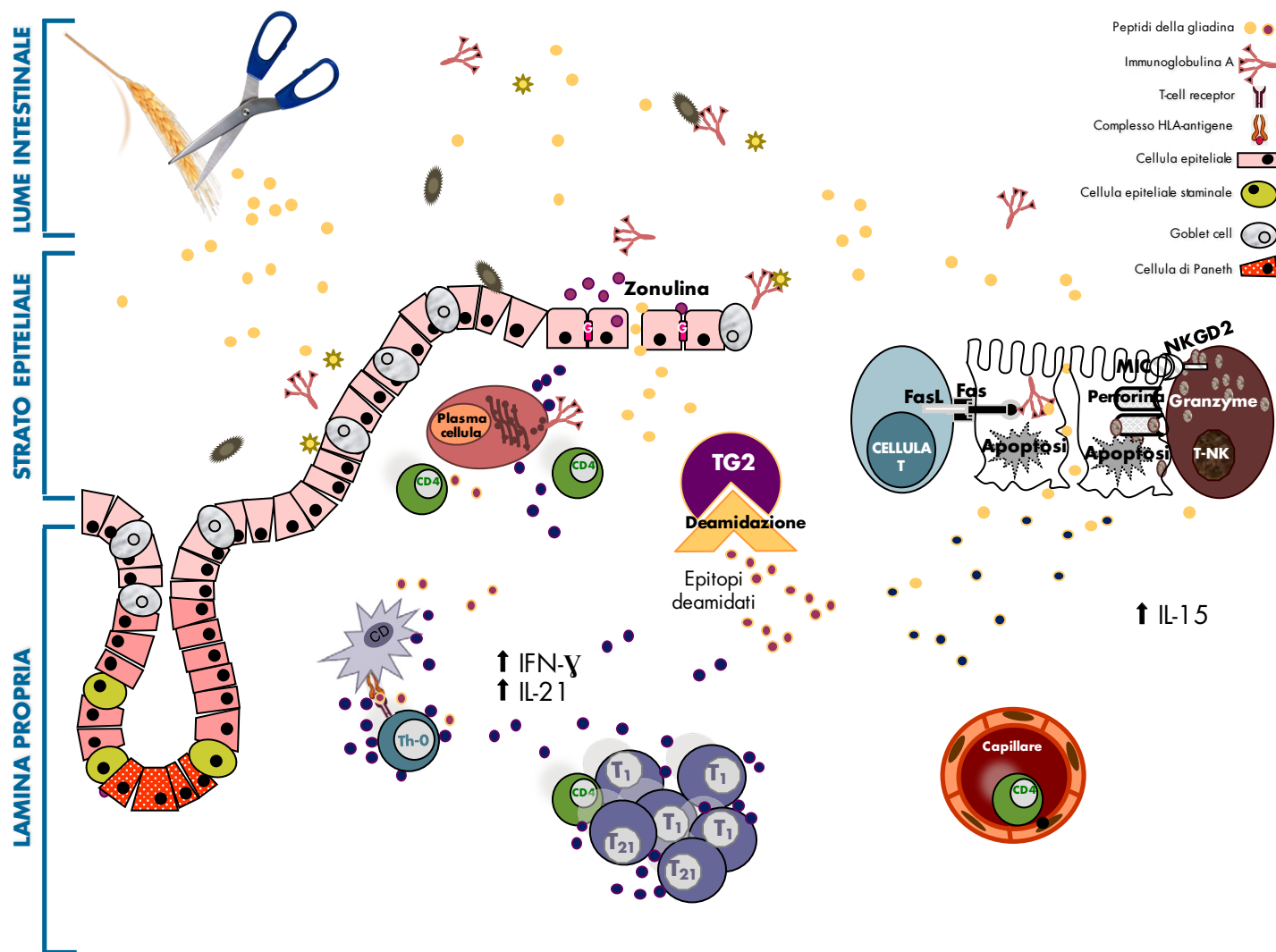
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CD: cellula dendritica; Th-0: T helper-0; IFN: interferone; IL: interleuchina; TG2: transglutaminasi tissutale di tipo 2; G: complesso giunzionale inter-enterocitario; NK: natural killer; FasL: ligando del recettore Fas; Fas: recettore di morte; NKG2D: natural killer group 2-member D; MIC: Major histocompatibility complex (MHC) class I-related chain molecules; T₂₁: T helper-21; T₁: T helper-1; T-NK: linfocita T-natural killer.

FIGURA 1.

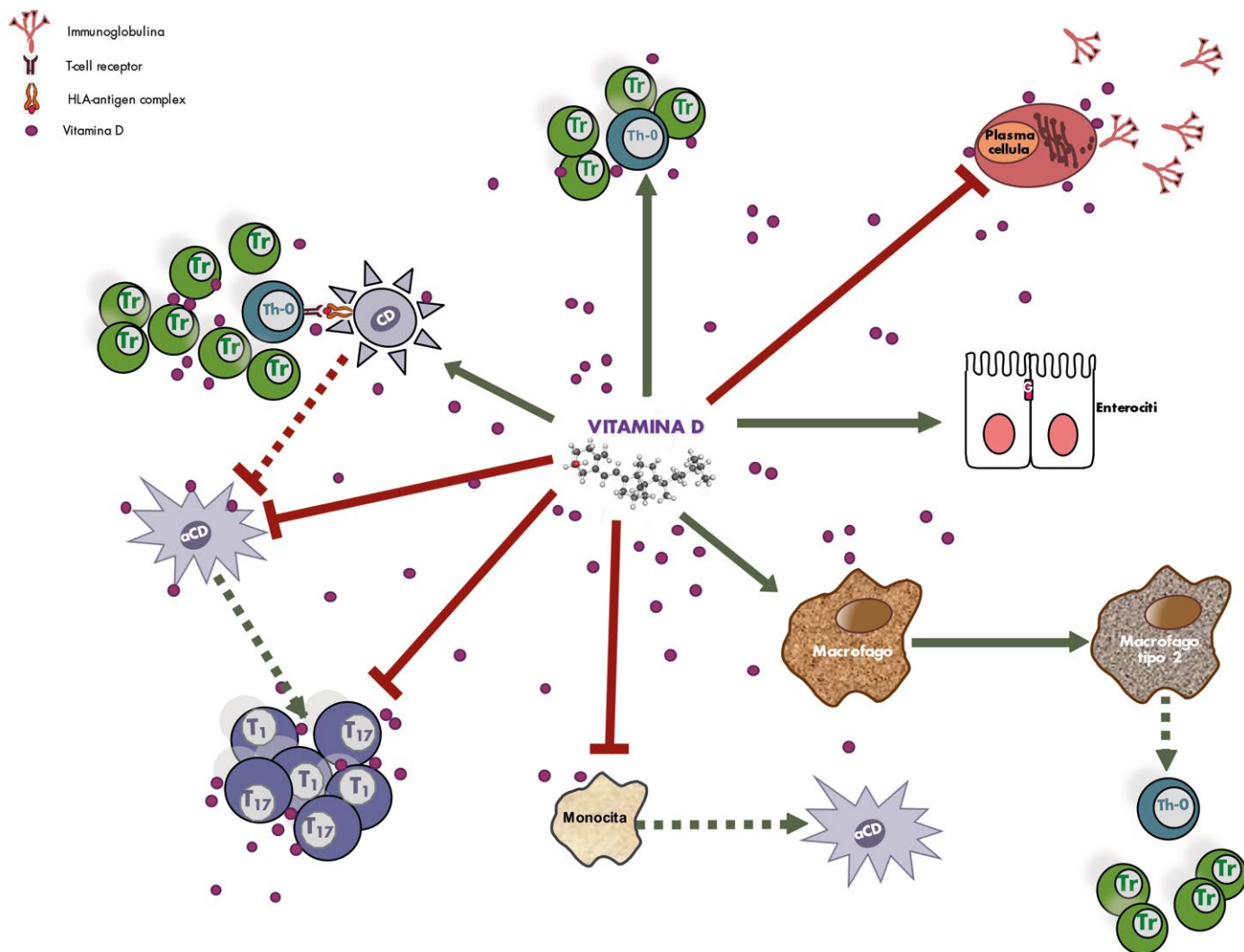
Immunopatogenesi delle lesioni intestinali nella malattia celiaca. Vedere testo.

parte le lesioni intestinali possono portare a un malassorbimento di vitamina D con le negative conseguenze sull'osso ⁹, una carenza di vitamina D si associa a un'abnorme risposta infiammatoria ¹⁰ che, almeno potenzialmente, potrebbe favorire l'esordio e il mantenimento dell'enteropatia stessa. In questa mini-review, partendo dall'immunopatogenesi della MC (Fig. 1), passeremo a trattare le evidenze sinora raccolte sugli effetti della vitamina D (e della sua carenza) sull'immunità innata e adattativa, e sulla barriera intestinale (Fig. 2), per poi citare i dati sinora disponibili nella MC.

LA MALATTIA CELIACA

Tale patologia può svilupparsi in soggetti geneticamente predisposti a seguito dell'ingestione del glutine e, d'altro canto, l'eliminazione del glutine dalla dieta è l'unica terapia ad oggi disponibile ¹¹. Si tratta di una malattia autoimmune che ha l'intestino tenue come organo bersaglio anche se, in una certa proporzione di casi, la malattia può colpire la cute (dermatite erpetiforme) o il cervello (ataxia glutine-relata) ¹². Dal punto di vista dei meccanismi patogenetici che conducono alle lesioni localizzate nella mucosa intestinale e caratterizzate da: au-

mento dei linfociti intra-epiteliali, iperplasia delle cripte, atrofia di vario grado dei villi e infiltrato infiammatorio polimorfo della lamina propria ¹³, la MC rappresenta una condizione privilegiata in quanto sono ben noti gli aplotipi di suscettibilità genetica (HLA-DQ2/8), il trigger esterno (il glutine) e l'autoantigene (l'enzima transglutaminasi tissutale) ¹¹. Purtroppo, la predisposizione genetica è presente in oltre il 30% della popolazione e il glutine è un alimento base della dieta pressoché di tutta la popolazione mondiale. È chiaro, pertanto, che sono necessari fattori aggiuntivi affinché le lesioni si realizzino



αCD: cellula dendritica attivata; CD: cellula dendritica; G: complesso giunzionale inter-enterocitario; Tr: linfocita T regolatorio; Th-0: T helper-0; T₁: T helper-1; T₁₇: T helper-17.

FIGURA 2.

Effetti immuno-modulanti ed epitelio-protettrici della vitamina D. Vedere testo.

e si mantengano e che ci aiutino anche a spiegare la variabilità dell'età di insorgenza della malattia stessa. Si parla, infatti, di "missing environmental factors" ¹⁴ e, tra questi, il microbiota, la tipologia del parto e dell'allattamento, l'epoca dello svezzamento, le infezioni virali e, recentemente, anche il tasso di vitamina D, sono stati chiamati in causa e sono l'oggetto delle attuali ricerche.

Immunopatogenesi

Il glutine rappresenta la componente proteica contenuta in alcuni cereali quali il grano,

l'orzo, la segale e l'avena, e che rimane a seguito dell'eliminazione della crusca con la macinazione e dell'amido con la centrifugazione ¹⁵. In realtà, si tratta di una miscela di proteine tra cui quelle solubili in alcol sono le gliadine nel grano, le ordeine nell'orzo, le secaline nella segale e le avenine nell'avena. La loro peculiarità risiede nel fatto che sono ricche di prolina e glutamina, il che conferisce alle rispettive farine le proprietà necessarie per la lievitazione e la panificazione. L'intestino umano non produce enzimi (prolil-endopeptidasi) in grado

di scindere il legame tra questi aminoacidi per cui, dopo la digestione chimica operata dall'acidità gastrica e quella enzimatica da parte delle peptidasi intestinali, rimangono degli oligopeptidi non ulteriormente scindibili ¹⁶. Nel caso in cui la barriera intestinale sia alterata a seguito, ad esempio, di un'infezione virale o una disbiosi ¹⁷, tali oligopeptidi attraversano l'epitelio e giungono nella lamina propria ove risiedono le cellule immuno-competenti che hanno il compito di mantenere la tolleranza immunologica nei confronti della miriade di antigeni batterici

e dietetici presenti nel lume intestinale. Inoltre, è stato invocato anche un meccanismo diretto di tali oligopeptidi nell'indurre un danno di barriera e, quindi, determinare un'aumentata permeabilità intestinale attraverso il rilascio della zonulina¹⁸, a seguito del loro legame con il recettore delle chemochine CXCR3 espresso sugli enterociti, con conseguente disassemblaggio delle molecole che compongono le giunzioni strette¹⁹. Queste, unitamente alle giunzioni aderenti e al complesso basale, sigillano gli enterociti tra di loro in modo da garantire il passaggio estremamente selettivo delle molecole per via trans- o para-cellulare e contribuiscono all'integrità di quell'unità anatomico-funzionale chiamata barriera intestinale²⁰. Altri fattori che la compongono sono le immunoglobuline secrete (sIgA), lo strato di muco che riveste gli enterociti, i linfociti intraepiteliali, nonché tutte le popolazioni cellulari presenti nella lamina propria e che formano il cosiddetto "gut-associated lymphoid tissue", da cui dipende il mantenimento dell'omeostasi immunologica²¹. È proprio dallo stato di quiescenza o attivazione delle cellule antigene-presentanti residenti nella lamina propria che dipende il destino della risposta immunologica in senso tollerogenico o infiammatorio²². Infatti, recenti studi hanno dimostrato che la presenza di specie patobionte nel microbiota intestinale, unitamente ai peptidi della gliadina, determina un'attivazione delle cellule dendritiche che quindi percepiscono tali oligopeptidi come antigeni, li complessano con le molecole HLA-DQ2/8 e li presentano ai linfociti T CD4⁺, con innesco di una cascata infiammatoria piuttosto che di una risposta tollerogenica²³. La presenza di un microambiente infiammatorio determina anche l'attivazione dell'enzima transglutaminasi tissutale che, se da una parte rappresenta l'autoantigene della MC²⁴, dall'altra opera una deamidazione selettiva di tali oligopeptidi, in particolare il 33mer, con sostituzione dei residui di glutamina con acido glutamico, che li rende immunodominanti, e quindi in grado di amplificare la risposta proliferativa e secretoria dei linfociti T-specifici²⁵. Questi ultimi, così stimolati, producono una cascata di citochine pro-infiammatorie, largamente dominata dall'interferone- γ e dall'interleuchina-15, con conseguente attivazione dei linfociti CD8⁺ cito-tossici, macrofagi, e cellule natural-killer responsabili, infine, dell'atrofia dei villi²⁶. Questa si produce, infatti, a seguito di un'esagerata apoptosi enterocitaria legata sia a

meccanismi citolitici Fas/ligando di Fas e perforina/granzyme, sia al distacco dalla membrana basale, che non viene compensata dall'aumentata proliferazione a livello delle cripte²⁷. Tale enteropoiesi inefficace contribuisce alla perdita della competenza funzionale della barriera intestinale, in quanto la superficie mucosa si trova ad essere rivestita da cellule in ritardo maturativo e, quindi, dotate di un complesso giunzionale inefficace. Infine, i linfociti T-helper presentano gli epitopi ai linfociti B che, differenziandosi in plasmacellule, producono gli anticorpi specifici²⁸ che possiamo dosare nel siero dei pazienti e sono dotati di elevata accuratezza diagnostica²⁹.

EFFETTI IMMUNOMODULANTI DELLA VITAMINA D

Il recettore della vitamina D (VDR), localizzato a livello nucleare e responsabile degli effetti biologici della vitamina D, è codificato da un gene altamente polimorfico che fa parte della superfamiglia dei recettori per gli steroidi³⁰.

Recentemente, l'espressione di tale recettore è stata identificata anche in tessuti non coinvolti nel metabolismo osseo e minerale e, in particolare, in cellule del sistema immunitario, come quelle presentanti l'antigene³¹. Pertanto, alcuni polimorfismi del VDR potrebbero aumentare o diminuire la suscettibilità a malattie immuno-mediate, tra cui la stessa MC³². Ciò ha dato il via a una serie di studi che hanno dimostrato come la vitamina D sia implicata nella risposta immunitaria sia innata che adattativa³³. Infatti, l'enzima che converte la 25-idrossi-vitamina D nella sua forma attiva è espresso anche nei monociti-macrofagi e viene attivato a seguito del legame dei recettori Toll-like sulla loro superficie con i rispettivi antigeni virali e batterici, con il risultato di potenziare le difese nei confronti delle infezioni³⁴. Inoltre, la vitamina D inibisce la differenziazione dei monociti in cellule dendritiche riducendo, pertanto, la capacità di presentare l'antigene e di innescare una risposta infiammatoria³⁵.

La vitamina D possiede anche un'azione diretta sulle cellule dendritiche, come dimostrato da studi *in vitro*, inibendo la loro maturazione e quindi il loro potere antigen-presentante, e favorendo l'acquisizione di un profilo tollerogenico^{36,37}.

Per quanto riguarda la risposta adattativa, la vitamina D riduce la differenziazione dei linfociti T verso un profilo pro-infiammato-

rio, mentre promuove l'espansione della quota ad attività regolatoria³⁸.

Inoltre, i linfociti T che esprimono elevati livelli di VDR sulla loro superficie sarebbero sensibili a un'azione immunomodulante della vitamina D, in senso anti-infiammatorio. E ciò è di grande interesse nell'ambito della patogenesi della MC in quanto i linfociti T gliadina-specifici sono i principali responsabili del danno mucosale e del processo linfomagenico legato a questo tipo di infiammazione cronica³⁹.

Per quanto riguarda gli effetti sui linfociti B, sempre studi *in vitro* hanno dimostrato che la vitamina D è in grado di ridurre la loro differenziazione in plasmacellule e di aumentarne l'apoptosi esitando, in ultima analisi, in una riduzione della produzione delle immunoglobuline e quindi, anche degli auto-anticorpi⁴⁰.

Inoltre, il VDR è localizzato anche sugli enterociti, ove ne regola la proliferazione, la differenziazione e l'apoptosi, in pratica governa l'enterocinetica, giocando quindi un ruolo di primo piano nei meccanismi di difesa e nella funzionalità della barriera intestinale⁴¹. A tal proposito, occorre menzionare i lavori di Chen et al. in cui si dimostra che la vitamina D svolge un effetto protettivo sulla barriera epiteliale *in vitro* e *in vivo* agendo sulla via *myosin light-chain kinase-dipendente* che, a sua volta, è attivata dall'aumento del fattore nucleare-kB indotto da uno stimolo infiammatorio^{42,43}. In particolare, la via *myosin light-chain kinase-dipendente* agisce direttamente sull'assemblaggio dei filamenti di actina, determinando una contrazione del citoscheletro e quindi una distruzione delle giunzioni strette. Ciò è di estrema importanza se consideriamo che il disassemblaggio di queste ultime è già stato dimostrato nella MC⁴⁴.

In seguito, il gruppo di Dong ha confermato l'effetto protettivo della vitamina D nei confronti delle giunzioni strette enterocitarie sia utilizzando un modello *in vitro* di monostato di cellule CaCo₂ in cui la rottura della barriera epiteliale era indotta dai peptidi della gliadina, sia in un modello *in vivo* glutine-sensibile⁴⁵. In particolare, in entrambi i modelli, la vitamina D era capace di inibire il rilascio di zonulina indotto dalla gliadina, proteggere l'integrità delle giunzioni strette e, quindi, di mantenere la competenza della barriera. Ma il primo lavoro che ha messo in relazione un possibile ruolo extra-osseo della vitamina D nella

MC del bambino è quello di Tanpowpong et al.⁴⁶. Gli autori hanno dimostrato come la carenza di vitamina D potrebbe contribuire ad alterare la barriera intestinale, rendendo i soggetti più suscettibili alle infezioni enteriche e, di conseguenza, al rischio di sviluppare un'abnorme risposta immunitaria nei confronti degli antigeni presenti nel lume intestinale. Ciò ha completamente rivoluzionato il modo in cui noi clinici pensiamo alla vitamina D nella MC ove una sua carenza era considerata solo un effetto dell'enteropatia e non una possibile causa. Infine, in una coorte di celiaci adulti, la carenza di vitamina D correlava con l'aumentata frequenza di psoriasi, sebbene non di altre malattie autoimmuni⁴⁷. Tutto ciò ci conduce a pensare che la semplice determinazione dei livelli sierici di tale vitamina nella MC sia riduttiva, pertanto auspichiamo che in futuro vengano condotti studi volti ad approfondire la globalità dei suoi effetti nel determinare il rischio di sviluppare tale patologia, di perpetuare il danno d'organo e, forse, anche di sviluppare le complicanze.

CONCLUSIONI

Da quanto esposto, appare evidente che la vitamina D possa giocare un ruolo di primo piano nella patogenesi della MC sia attraverso un effetto protettivo diretto sulla barriera intestinale, sia modulando la risposta immunitaria a favore dei meccanismi di tolleranza. Ciò conduce a ipotizzare che programmi di prevenzione della sua carenza possano contribuire ad arginare l'aumento dell'incidenza non solo di questa patologia, ma anche di molte altre malattie infiammatorie croniche. In tal senso, vale la pena ricordare che una carenza di vitamina D della madre durante la gravidanza sembra legata a un'aumentato rischio di sviluppare malattie autoimmuni, tra cui la MC, soprattutto nei primi due anni di vita⁴⁸. Ovviamente, non è pensabile di supplementare tutta la popolazione, soprattutto chi ha un adeguato introito giornaliero di vitamina D, in quanto un sovradosaggio non solo è dannoso per l'osso, ma anche per l'omeostasi immunologica in quanto favorisce una polarizzazione in senso Thelper 2^{49,50}. È fondamentale, pertanto, portare avanti programmi di prevenzione e di screening sulla popolazione volti a identificare un'eventuale carenza della vitamina D al fine di limitare le conseguenze su larga scala.

Ringraziamenti

Un sentito ringraziamento ai celiaci e ai loro familiari per la disponibilità a partecipare a ricerche volte a migliorare la nostra conoscenza sulla malattia e, quindi, la loro condizione.

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La vitamina D e il COVID-19: un raggio di sole nella tempesta?

VITAMIN D

UpDates

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La patologia COVID-19 (COroNaVirus Disease-2019), come in molte patologie emergenti, presenta caratteristiche biologiche, manifestazioni cliniche e di imaging strumentale assolutamente uniche. COVID-19 è la conseguenza, in alcuni soggetti, dell'infezione da Severe Acute Respiratory Syndrome CoronaVirus-2 (SARS-CoV-2), a oggi il settimo tipo di coronavirus in grado di infettare l'uomo¹. L'infezione si è rapidamente diffusa a tutto il pianeta partendo dalla Cina². Il virus ha un'elevata trasmissibilità, principalmente attraverso *droplets* emesse con la fonazione e il respiro, o tramite contatto diretto (sebbene nelle fasi più avanzate dell'infezione il virus sia riscontrabile anche nelle feci e questo non esclude una possibile via di infezione oro-fecale)¹. COVID-19 ha un'incubazione che va da 1 giorno a 2 settimane, con un picco tra il 3° e 7° giorno. Lo spettro di espressione clinica varia da forme del tutto asintomatiche fino a quadri drammatici come l'Acute Respiratory Distress Syndrome (ARDS). I sintomi più comuni nelle forme lievi-moderate sono febbre, astenia e la tosse secca, alle quali possono seguire o accompagnare cefalea, congestione nasale, faringodinia, mialgie e artralgie. Raramente vi è in coinvolgimento gastro-intestinale (specie nei bambini) con nausea, vomito e diarrea¹. Una proporzione variabile di soggetti infettati, spesso durante la seconda settimana di malattia, sviluppa difficoltà respiratoria, ipossia, desaturazione e tachipnea. Questi sono i segni tipici del coinvolgimento polmonare severo che può arrivare fino alla polmonite bilaterale di tipo interstiziale e che a sua volta può purtroppo evolvere in ARDS, caratterizzata da morbidità e mortalità considerevoli¹. Frequente è anche la presenza di disturbi del sistema coagulativo e una trombocitopenia con conseguente aumentato rischio di emorragia, associata o meno ad altri disordini ematologici, come trombosi periferica, trombosi venosa profonda, tromboembolia polmonare e coagulazione intravasale disseminata (DIC)¹.

Considerate le attuali limitazioni dei trattamenti antivirali a oggi disponibili, la gestione clinica si basa essenzialmente sul controllo della risposta infiammatoria abnorme e sul supporto respiratorio in ambiente ospedaliero. Questo spiega perché la pandemia abbia finito per sconvolgere anche le economie e i sistemi sanitari più consolidati, imponendo un ripensamento sull'allocazione degli sforzi e delle risorse. Sembra scontato in un momento di crisi abbassare l'attenzione da tutte quelle condizioni che non sono considerate "essenziali". Tuttavia, siamo davvero sicuri che questa strategia sia corretta? Su questo tema (in particolare per quanto riguarda il problema osteoporosi) è stato pubblicato un interessante editoriale³ dove viene riportata una massima di Jawaharlal Neru (erede spirituale di Gandhi), che affermava: "in una crisi, ogni piccola cosa conta". Questa citazione dovrebbe far riflettere, soprattutto perché, a oggi, sono ancora pochi i dati a disposizione non solo relativamente ai trattamenti concretamente efficaci, ma anche riguardo i fattori in grado di condizionare la suscettibilità all'infezione e la sua gravità.

Certamente, le vitamine sono "una piccola cosa", ma la vitamina D in particolare (o meglio, la sua carenza) potrebbe avere un ruolo non irrilevante, anche in relazione ai suoi dibattuti effetti extrascheletrici. Sebbene siamo perfettamente d'accordo con l'European Society for Clinical and Osteoarthritis (ESCEO) sul fatto che non vi siano al momento evidenze sufficienti per raccomandare l'uso della supplementazione vitaminica D per la prevenzione e/o trattamento di patologie extrascheletriche⁴, dobbiamo anche ricordare come sia stato proprio questo *position paper* a sottolineare la crescente mole di dati disponibili (soprattutto indiretti ma anche diretti) a supporto degli effetti extrascheletrici della vitamina D⁴. Fino a oggi, l'infezione da SARS-CoV-2 pare essere risultata più frequente e particolarmente aggressiva anche in termini di mortalità nei paesi del Sud Europa (specie Italia e

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Numero di pazienti che devono essere supplementati (NNT) con vitamina D per evitare un'infezione respiratoria acuta (da metanalisi di 25 studi con oltre 11.300 soggetti)





	INTERA CASISTICA SENZA SUBANALISI	NNT = 33
	Solo supplementazione quotidiana o settimanale	NNT = 20
	Solo soggetti con livelli di vitamina D < 10 ng/ml	NNT = 8
	Solo soggetti con livelli di vitamina D < 10 ng/ml + solo supplementazione quotidiana o settimanale	NNT = 4

FIGURA 1.

Dai dati di questa vasta metanalisi emerge come la supplementazione vitaminica D sia in grado di ridurre significativamente il rischio di infezioni respiratorie acute. La protezione (in termini di NNT) è più marcata nei soggetti gravemente carenti e appare particolarmente rilevante con l'uso della somministrazione quotidiana o settimanale (da Martineau et al., 2017, mod) ⁶.

Spagna), che sono gli stessi maggiormente coinvolti dalla condizione di ipovitaminosi D ⁵. In Italia le regioni più colpite sono quelle del Nord, rispetto alle più soleggiate Regioni Meridionali e, soprattutto, durante il periodo invernale. I soggetti anziani, e soprattutto quelli nelle lungodegenze, nei quali l'ipovitaminosi D è sostanzialmente endemica, sono quelli che hanno pagato le conseguenze più drammatiche della malattia e lo stesso dicesi per gli obesi, anch'essi a elevato rischio di deficit vitaminico D.

Come abbiamo già detto, COVID-19 è prevalentemente una malattia infettiva respiratoria e la mole di dati a favore di un significativo effetto della vitamina D nel prevenire e mitigare le infezioni respiratorie è divenuta in questi ultimi anni sempre più consistente. Un'interessante metanalisi pubblicata nel 2019 ha analizzato i dati di oltre 11.300 pazienti provenienti da 25 studi randomizzati, dimostrando un effetto protettivo della supplementazione vitaminica D verso le infezioni respiratorie acute, effetto non solo statisticamente ma anche clinicamente significativo: *Number Needed to Treat* (NNT) = 33. Ovviamente, l'efficacia è risultata maggiore nei soggetti affetti da una severa carenza (NNT = 8) (Fig. 1). Questa azione protet-

tiva della vitamina D non è tuttavia risultata evidente nella subanalisi dei 15 studi che prevedevano l'uso della somministrazione con boli e, pertanto, l'effetto protettivo parrebbe quindi in gran parte sostenuto dall'effetto della supplementazione quotidiana o settimanale (10 studi), dove il risultato è stato particolarmente interessante (Fig. 1) ⁶. Un'altra metanalisi si è invece concentrata sui pazienti con BPCO (broncopneumopatia cronica ostruttiva) dimostrando ancora una volta come la supplementazione vitaminica D sia in grado sostanzialmente di dimezzare il numero delle riacutizzazioni respiratorie moderate/severe nei pazienti con stato carenziale al baseline (<10 ng/ml) ⁷. Questa ultima osservazione appare fondamentale visto il gran numero di studi clinici negativi sulla vitamina D recentemente pubblicati, che purtroppo condividono proprio questo limite, ovvero l'arruolamento di una maggioranza di soggetti non solo non carenti ma spesso addirittura con valori ben al di sopra del limite ideale ⁸. La vitamina D, infatti, non andrebbe considerata un agente farmacologico, bensì un micronutriente. Conseguentemente, la sua supplementazione risulta indicata ed efficace strettamente in condizioni di deficit.

Il ruolo immunomodulatore della vitamina D è noto da tempo. Essa è in grado di sostenere l'immunità innata mediante la produzione di peptidi antimicrobici, quali le catelicidine, le defensine e l'IL-37. Inoltre, mediante la modulazione delle principali citochine proinfiammatorie, quali IL-6, TNF- α e interferone-gamma, è in grado di agire sull'immunità adattativa controllando la risposta mediata dai linfociti Th1 ⁹. Questo controllo chiaramente inizia a zoppiare in condizioni di deficit vitaminico D, il quale tuttavia si ripristina dopo un'adeguata supplementazione. Un recente studio su cellule dell'epitelio respiratorio ha dimostrato che il pretrattamento con concentrazioni fisiologiche di metaboliti della vitamina D (calcifediolo o calcitriolo) sia in grado di produrre una transitoria resistenza all'infezione da Rinovirus (Rv-16) e di attenuare la produzione indotta dal virus delle molecole di adesione necessarie sia al Rinovirus che allo *Streptococcus pneumoniae*. Tutto questo si accompagna all'attivazione del gene per la catelicidina e alla modulazione di NF- κ B, ulteriori possibili meccanismi alla base degli effetti protettivi della vitamina D sul rischio di infezione da Rinovirus e di sovra-infezione batterica ¹⁰. Per quanto riguarda il virus SARS-CoV-2, i dati per il momento sono ancora limitati, ma un report preliminare di uno studio che ha valutato il potenziale antivirale di diverse molecole ha documentato l'effetto inibitorio del calcitriolo sull'epitelio nasale infettato dal virus ¹¹. Questo dato appare di particolare interesse se consideriamo uno studio israeliano su 14.000 soggetti testati per infezione da SARS-CoV-2 che avevano eseguito almeno un precedente dosaggio sierico della 25-idrossi-vitamina D [25(OH)D]. Dai risultati emerge come avere livelli di vitamina D sub-ottimali (< 30 ng/ml) rappresenti un potenziale fattore di rischio di infezione da SARS-CoV-2, di sviluppo conseguentemente di COVID-19 e, in particolare, di necessità di ospedalizzazione ¹². Questi risultati appaiono in linea con quelli di uno studio americano su un campione di 489 soggetti che avevano una pregressa valutazione dello stato vitaminico D nell'anno precedente. Di questa coorte, 71 soggetti sono in seguito risultati positivi per infezione da SARS-CoV-2. Lo studio ha documentato come l'essere "probabilmente" carenti (livelli circolanti di 25(OH)D < 20 ng/ml oppure di 1,25(OH)₂D < 18 pg/ml) fosse associato a un rischio 1,77 volte maggiore di essere positivi al test ¹³.

Al contrario, un simile studio inglese non ha replicato tale riscontro ¹⁴, sebbene valori "anamnestici" di metaboliti della vitamina D potrebbero non essere rappresentativi della reale condizione al momento dell'infezione e quindi apportare un limite non indifferente di questo tipo di esperienze scientifiche.

Un rilevamento interessante dello studio americano già citato ¹³ è che nei 48 soggetti inizialmente carenti e che grazie alla supplementazione vitaminica D sono arrivati a livelli adeguati, il rischio di essere positivi test per SARS-CoV-2 sarebbe risultato sovrapponibile a quello dei soggetti che erano fin dall'inizio repleti di vitamina D. Questo sembrerebbe appunto supportare l'effetto protettivo della supplementazione vitaminica D quando in grado di normalizzare lo stato vitaminico del soggetto. Il numero limitato dei casi, tuttavia, si associa a un intervallo di confidenza troppo grande per poter sostenere questa ipotesi in maniera solida.

Differente è invece il disegno di uno studio svizzero che ha valutato i livelli circolanti di 25(OH)D in una coorte di pazienti con sintomi sospetti per COVID-19 dopo qualche settimana dall'esecuzione del tampone, dimostrando come nei soggetti poi risultati positivi i livelli fossero significativamente più bassi (mediana circa 11 ng/ml) rispetto ai negativi (mediana circa 25 ng/ml) ¹⁵. Il dato appare interessante anche se non si può escludere che proprio l'infezione virale di per sé possa essere stata la causa del deficit vitaminico D. La relativamente breve durata dell'infezione (poche settimane), tuttavia, rende comunque questa ipotesi tutto sommato meno probabile.

La vitamina D, oltre a fornire una potenziale protezione dall'infezione, potrebbe anche condizionare l'evoluzione e la gravità della stessa, come del resto suggerisce anche il dato relativo alle ospedalizzazioni dello studio israeliano sopra citato ¹². La modulazione/soppressione da parte della vitamina D di un'eventuale eccessiva risposta Th1 potrebbe infatti contribuire a contrastare la tempesta citochinica alla base del danno polmonare e della progressione verso l'ARDS ⁹. Infatti, la carenza vitaminica D è dimostrata essere associata a un maggior rischio di sviluppare ARDS ¹⁶. Inoltre, la sua correzione, parrebbe essere in grado di ridurre il danno capillare alveolare prodottosi nei soggetti carenti ¹⁶. Questa capacità protettiva della vitamina D sembra essere secondaria all'azione locale

del metabolita attivo calcitriolo sul sistema renina-angiotensina mediante un effetto diretto sull'espressione degli enzimi ACE ¹⁷. Ciò risulta particolarmente interessante se consideriamo che l'ACE-2 è ritenuto essere il recettore chiave per le infezioni da SARS-CoV-2. Come infatti noto, il virus si legerebbe tramite la proteina *spike* proprio al recettore ACE-2 per poter penetrare nelle cellule polmonari e in seguito agirebbe downregolando sia l'attività che l'espressione dell'enzima stesso ⁹.

In conclusione, i dati a nostra disposizione rendono a nostro avviso credibile un legame tra carenza di vitamina D e suscettibilità e severità della infezione da SARS-CoV-2. Sulla base di questo razionale, diversi studi clinici interventistici sono stati avviati in pazienti con quadri di infezione severa. Si tratta di una sfida che molti esperti, noi compresi, riteniamo sarà difficile da vincere ¹⁸. In questa tipologia di pazienti, infatti, l'abnorme risposta infiammatoria è probabilmente già troppo sostenuta per ipotizzare un significativo beneficio da parte della supplementazione vitaminica D, anche in soggetti molto carenti. Inoltre, i farmaci steroidei o immunosoppressori utilizzati in questi casi hanno effetti tali da mascherare quelli potenziali della vitamina D, che rimane pur sempre un micronutriente. In ogni caso, vista la prevalenza del deficit vitaminico D, questi soggetti andrebbero a nostro avviso comunque supplementati.

Più promettente potrebbe essere invece lo studio sul beneficio della supplementazione (quotidiana o settimanale) nella riduzione della suscettibilità all'infezione e progressione verso forme più severe.

Rimane infine un fondato timore sull'impatto che la nota AIFA 96 potrebbe aver avuto sulla prevalenza del deficit vitaminico D, non solo nei soggetti più fragili dal punto di vista osseo, ma anche in quelli più a rischio di COVID-19.

Sottolineamo quindi la necessità di una forte presa di posizione in tempi rapidi, prima dell'inizio dei mesi invernali e di un'eventuale ripresa della forza della pandemia, soprattutto se davvero convinti che "in una crisi, ogni piccola cosa conta".

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