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EDITORIALE

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Cari Lettori
in questo numero torniamo a parlare di carenza di vitamina D in età pediatrica, la cui prevalenza è tornata ad aumentare in alcuni Paesi, come documentato in Inghilterra. Si, perché come scrive l'Autore dell'articolo a cui abbiamo affidato il compito di un aggiornamento in questo campo, sono cambiati e diventati più frequenti alcuni fattori di rischio come la composizione etnica e il sovrappeso. È aumentata in Europa la popolazione con colorito della pelle più scuro o con abitudini culturali quali la copertura estrema per motivi religiosi, fattori che com'è noto ostacolano la produzione endogena di vitamina D tramite l'esposizione solare. Ma anche la prevalenza del sovrappeso è arrivata al 30% della popolazione pediatrica, condizione anche questa che rappresenta un importante fattore di rischio per ipovitaminosi D e che richiede notoriamente una supplementazione con dosi maggiori, specie se si tratta di obesità. Al fine di assicurare un adeguato sviluppo scheletrico ma anche per probabili benefici extra-scheletrici, si ricorda quindi come sia importante assicurare un adeguato stato vitaminico D nelle varie fasi della crescita, necessariamente ricorrendo a supplementazione specie nei primi anni di vita e se la madre era in condizioni di ipovitaminosi D. In età pediatrica l'Autore sottolinea e documenta come le dosi di vitamina D debbano essere assunte quotidianamente e non in bolo mensile o settimanale, probabilmente perché alte dosi in bolo possono indurre l'espressione di enzimi del catabolismo inattivanti la vitamina D, come anche da noi ipotizzato sulla base dei risultati di un nostro recente studio sulla farmacocinetica [1]. In un precedente numero di questa rivista [2] avevamo anche proposto una giustificazione farmacocinetica e farmacodinamica a supporto degli esclusivi benefici, in particolare extrascheletrici, riportati con la somministrazione quotidiana di colecalciferolo.
Nell'altro articolo ho chiesto agli Autori di fare il punto su una possibile correlazione tra carenza di vitamina D e sindrome dolorosa regionale complessa, più praticamente definita algodistrofia, essendo recentemente stato riportato che pazienti con frattura di radio distale complicata da sindrome algodistrofica presentavano concentrazioni plasmatiche di vitamina D significativamente inferiori rispetto a coloro che non erano andati incontro a questa complicanza. Sulla base delle evidenze disponibili, come vedrete, gli Autori riconoscono che la carenza di vitamina D possa predisporre a un aumentato rischio di sindrome algodistrofica sostanzialmente per due motivi: il primo perché in grado di determinare, anche per il connesso rischio di caduta, un aumento di eventi fratturativi, specie di quelli intra-articolari, predisponenti la sindrome. Il secondo motivo potrebbe essere rappresentato dal fatto che l'ipovitaminosi D rappresenta una condizione predisponente la neuroflogosi e un assetto immunologico proflogogeno, entrambi coinvolti nella patogenesi della sindrome algodistrofica. A riprova di un possibile nesso causale tra livelli di vitamina D e di citochine proflogene, ricordano studi che dimostrano come la supplementazione vitaminica D sia in grado di ridurre la concentrazione sierica di TNFalfa e di IL-6, oltre che di IL-17, come da noi osservato in un recente studio [3]. Da ciò

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l'ipotesi che una supplementazione con vitamina D possa contribuire a determinare un beneficio aggiuntivo o più rapido del noto approccio terapeutico dell'algodistrofia con neridronato. Probabilmente contribuirebbe anche a ridurre l'incidenza o ad attenuare la manifestazione clinica dell'effetto collaterale degli aminobisfosfonati rappresentato dalla reazione di fase acuta, visto che i livelli sierici di 25(OH)D la modulano [4].
Cosa ne pensate?
Buona lettura

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Carenza di vitamina D e *Complex Regional Pain Syndrome*

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Al fine di esplorare quelli che sono i possibili rapporti tra vitamina D e sindrome algodistrofica, ovvero la *Complex Regional Pain Syndrome* (CRPS) secondo l'attuale nosografia internazionale, è indispensabile porre attenzione ai reciproci rapporti che individuano nel tessuto osseo un attore fondamentale nelle dinamiche patogenetiche dell'algodistrofia [1].
Oltre ai consolidati riscontri terapeutici ottenuti con farmaci il cui meccanismo d'azione prevede il tessuto osseo quale bersaglio principale, esistono tutta una serie di evidenze che depongono per un ruolo fondamentale dell'osso nell'innesco e nel mantenimento della malattia. A prescindere dai riscontri strumentali (osteoporosi alla radiologia standard, ipercaptazione all'esame scintigrafico, edema osseo alla risonanza magnetica), gli studi epidemiologici riportano la frattura quale evento predisponente più frequente e, di conseguenza, tutte le patologie che comportano un aumento della fragilità scheletrica e quindi dell'incidenza di fratture (osteoporosi postmenopausale e senile, *Osteogenesis imperfecta*), sono gravate da un parallelo aumento di incidenza della malattia algodistrofica. A ulteriore riprova di tale nesso patogenetico, vi sono segnalazioni che dimostrano come la patologia osteoporotica sia presente nei pazienti affetti da CRPS con una prevalenza significativamente maggiore rispetto alla popolazione generale [2].
Il modello animale che più fedelmente riproduce la malattia umana si ottiene inducendo una frattura della tibia distale nell'animale da esperimento. Va infine citato il riscontro di un incremento dell'osteoprotegerina (OPG), molecola coinvolta nella regolazione del sistema RANK/RANKL, nelle fasi iniziali di malattia.
Come precedentemente riportato, la CRPS trova in un evento traumatico che nella maggior parte dei casi comporta una frattura l'evento predisponente più frequente. Le più attendibili rilevazioni epidemiologiche [3] evidenziano come il picco di incidenza nel sesso femminile e nella decade successiva alla menopausa è verosimilmente l'epifenomeno di un analogo andamento nella popolazione generale della frattura di radio distale, ovvero l'evento fratturativo che più frequentemente si complica con una CRPS [4]. I dati di incidenza a seguito di una frattura del radio distale riportati in letteratura presentano risultati ampiamente dispersi (dall'1 al 37%) e tale variabilità è sicuramente da riferirsi ai diversi criteri diagnostici impiegati per censire tale evento. Gli studi più recenti, ovvero quelli che impiegano i criteri diagnostici adottati dall'*International Association for the Study of Pain* (IASP), ovvero i criteri di Budapest, e riconosciuti come quelli dotati delle migliori caratteristiche in termini di sensibilità e specificità, riportano un'incidenza di CRPS nel 14% dei pazienti che subiscono una frattura di radio distale [4].
È ampiamente noto come tale tipo di frattura rappresenti l'evento clinico più precoce correlato all'osteoporosi, verificandosi mediamente 15 anni prima della frattura di femore prossimale e come rappresenti un evento predittivo di altre fratture da fragilità, segnatamente la frattura vertebrale e la frattura di femore prossimale [5]. Tra le diverse variabili cliniche individuate come predittive della frattura di radio distale va annoverata la carenza di vitamina D [6].
Partendo quindi dalla premessa che adeguati livelli di vitamina D siano indispensabili per una buona salute scheletrica, la ricerca si è rivolta a indagare se un'ipovitaminosi D possa rappresentare il tramite attraverso cui soggetti carenti e quindi maggiormente predisposti alla frattura da fragilità, siano in qualche misura maggiormente predisposti a essere affetti da CRPS. Un ulteriore aspetto oggetto di indagine è stato quello di verificare se un'ipovitaminosi D sia, in presenza di un evento fratturativo, una condizione che, indipendentemente da altre variabili, sia in grado di favorire la comparsa della sindrome algodistrofica. Sulla base dei riscontri epidemiologici sopra citati, la frattura di radio distale (frattura di Colles)

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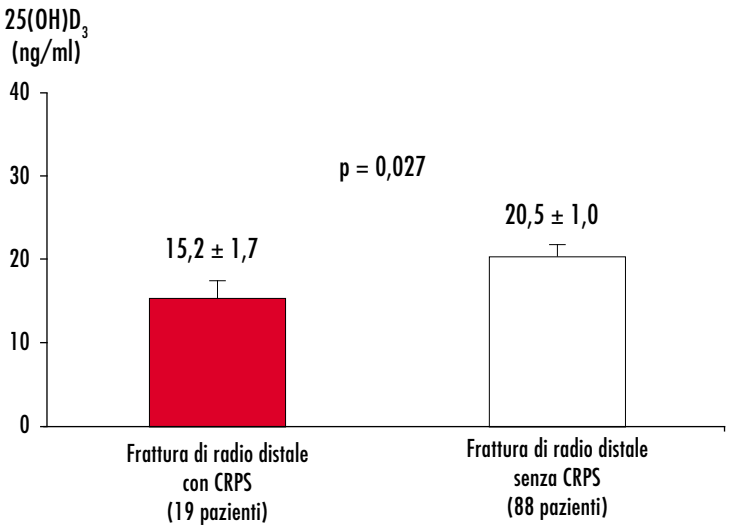

FIGURA 1.
Confronto dei valori di 25(OH)D_3 tra 19 pazienti che hanno sviluppato un'algodistrofia dopo frattura del radio distale e 88 pazienti senza algodistrofia.

ha rappresentato l'evento fratturativo che è stato più estensivamente indagato.
Nel 2020 uno studio retrospettivo condotto in ambito ortopedico su oltre un centinaio di donne postmenopausali, ha dimostrato come i soggetti che dopo una frattura di radio distale andavano incontro allo sviluppo della sindrome algodistrofica presentavano concentrazioni plasmatiche di vitamina D significativamente inferiori rispetto a coloro che non presentavano tale tipo di complicanza (Fig. 1) [7]. È interessante sottolineare come il dosaggio di alcuni marcatori biochimici del turnover scheletrico (osteocalcina, fosfatasi alcalina), così come la valutazione densitometrica eseguita sia a livello lombare che a livello del femore prossimale, non mostravano differenze significative tra i soggetti che sviluppavano una CRPS e i soggetti che presentavano la stessa frattura e il medesimo trattamento chirurgico ma non presentavano nessun segno in senso algodistrofico.
A livello speculativo i risultati di tale studio aprono le porte a una serie di possibili ipotesi di ordine patogenetico in grado di rappresentare un tramite tra bassi valori di vitamina D e insorgenza di una CRPS. In primo luogo, l'indagine densitometrica non mostra differenze significative in ragione della comparsa o meno della sindrome algodistrofica. Tale risultato consente di ipotizzare come la malattia osteoporotica definita per il tramite dell'indagine densitometrica non rappresenti per sé un fattore di rischio per la comparsa di un'algodistrofia. Le considerazioni epidemiologiche sopra riportate sono viceversa coerenti con un ruolo indiretto dell'osteoporosi: la presenza di bassi valori di massa ossea possono essere considerati il tramite per il quale i soggetti affetti da osteoporosi andrebbero più frequentemente incontro a un evento predisponente come la frattura di radio distale, senza che la presenza di osteoporosi rappresenti un moltiplicatore della probabilità di andare incontro ad algodistrofia. Analoghe considerazioni possono essere fatte per i marcatori biochimici del metabolismo osseo: la sovrapponibilità dei risultati tra soggetti affetti da algodistrofia e i soggetti che non sviluppano la malattia tenderebbe a escludere che i livelli di turnover scheletrico rappresentino un fattore di rischio per lo sviluppo della malattia algodistrofica.
Una possibile chiave interpretativa dei risultati di tale studio provengono dall'esplorazione di alcune altre segnalazioni presenti in letteratura. Un recente studio su soggetti che presentavano una frattura di radio distale, ha dimostrato che i pazienti che presentavano una frattura intra-articolare (con coinvolgimento della corticale distale del radio) presentavano al momento della frattura valori sierici di 25(OH)D_3 significativamente inferiori rispetto ai soggetti che presentavano una frattura metafisaria extra-articolare [8]. È sufficiente incrociare questo dato con i risultati di uno studio osservazionale su circa 600 pazienti fratturati che ha esplorato i fattori predittivi della comparsa della sindrome algodistrofica [4] per acquisire un riscontro suggestivo in tal senso: quale fattore di rischio per la comparsa della sindrome algodistrofica a seguito di un evento fratturativo va annoverata proprio la frattura intra-articolare e pluriframmentaria. Anche in questo caso, quindi, l'ipovitaminosi D potrebbe avere un ruolo indiretto: una situazione carenziale potrebbe essere predittiva di una frattura intra-articolare che a sua volta correla con una maggiore probabilità di sviluppare algodistrofia.
Discorso analogo potrebbe valere per l'artrite reumatoide. La presenza di tale malattia rappresenta un fattore di rischio per la comparsa di algodistrofia a seguito di un evento fratturativo [4] ed è ben nota l'elevata prevalenza di ipovitaminosi D nei soggetti affetti da artrite reumatoide [9]. Infine, quale ulteriore nesso causale in grado di porre in relazione algodistrofia, frattura e ipovitaminosi D va citata la propensione alla caduta e quindi alla frattura del soggetto anziano che presenta valori di vitamina D inadeguati [10].
La malattia algodistrofica è caratterizzata da un'intensa sintomatologia dolorosa, alterazioni sensitive e vasomotorie, edema locale e deficit funzionale. Negli ultimi anni, importanti acquisizioni sono state ottenute circa i meccanismi patogenetici di malattia (Fig. 2). L'increzione locale di citochine proflogogene e la liberazione retrodromica da parte di afferenti nocicettivi di mediatori neuropeptidici in grado di interferire con la regolazione del microcircolo locale, rappresentano gli eventi che innescano e mantengono la malattia, causando iperalgesia e allodinia, ovvero una percezione dolorosa sproporzionata rispetto all'intensità dello stimolo, e una percezione dolorosa a seguito di uno stimolo normalmente non in grado di evocare dolore. Successivamente, l'alterata permeabilità capillare, l'edema interstiziale e la conseguente ipossia e acidosi locale costituiscono le successive dinamiche patogenetiche in grado di mantenere le tipiche manifestazioni cliniche, ovvero l'intensa sintomatologia dolorosa, l'edema, le alterazioni del termotatto e del colorito locale [11] (Fig. 3).
L'utilizzo di metodiche biochimiche estremamente sensibili e lo studio di modelli animali hanno consentito di identificare i processi di neuroflogosi quali eventi patogeneticamente connessi con le manifestazioni cliniche iniziali di malattia. In modelli murini di CRPS-1 è stata dimostrata un'elevata concentrazione locale di *Nerve Growth Factor*,

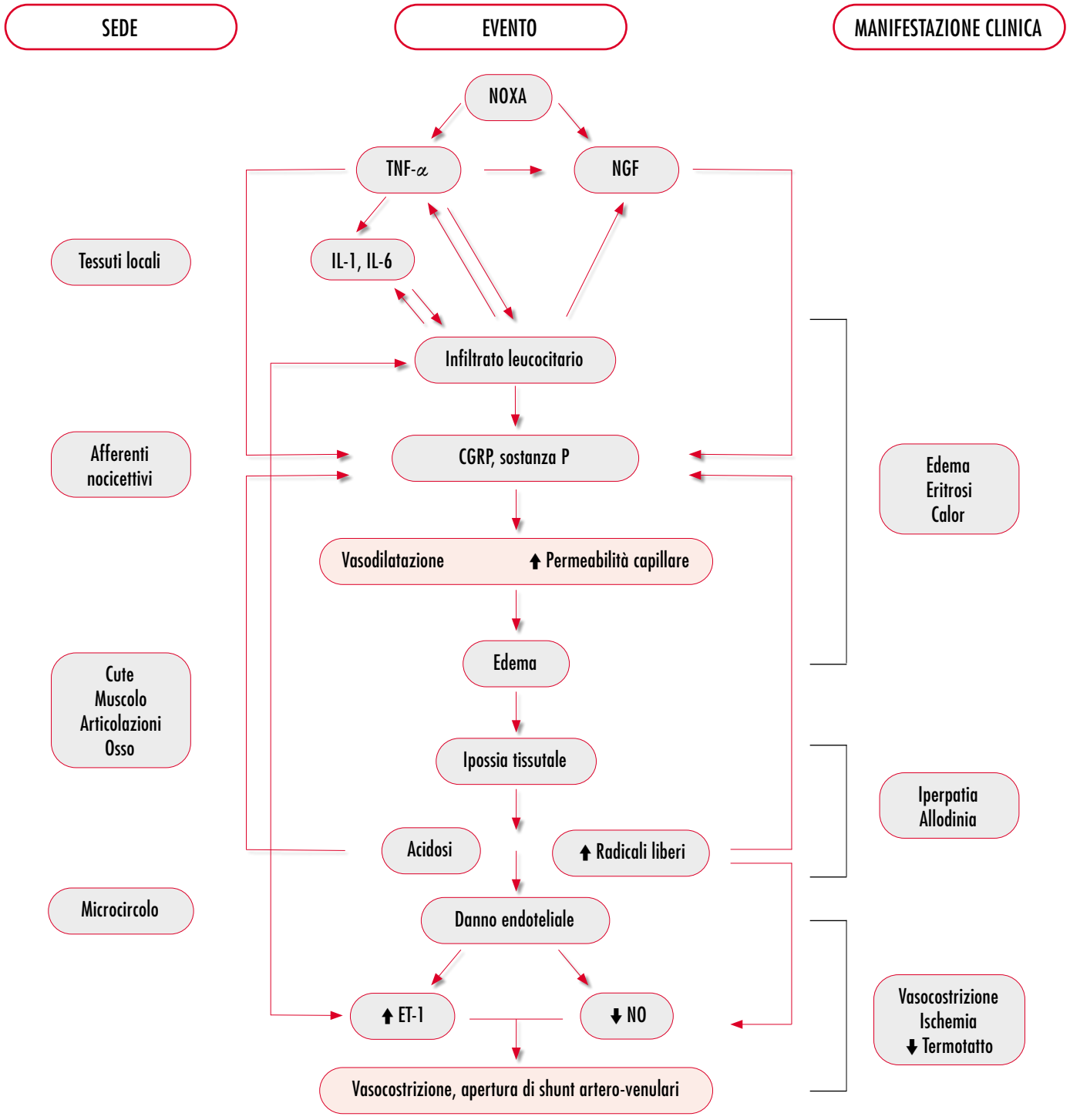

FIGURA 2.
Diagramma dei meccanismi patogenetici coinvolti nell'esordio e nel mantenimento della CRPS-I e delle conseguenti manifestazioni cliniche.

potenzialmente implicato nei meccanismi di genesi e trasmissione del dolore e nell'induzione della produzione citochinica locale. È un dato definitivamente acquisito che alcune citochine proflogogene, quali il *Tumor Necrosis Factor-α* (TNFα), l'interleuchina-1 (IL-1) e l'interleuchina-6 (IL-6), siano localmente presenti in concentrazioni elevate durante le prime fasi di malattia, così come alcuni studi ne dimostrano concentrazioni elevate anche a livello sistemico. Vi sono inoltre evidenze di come il rilascio locale di alcune citochine proinfiammatorie sia mediato anche dal coinvolgimento dei cheratinociti dei piani cutanei, evento questo che può giustificare il

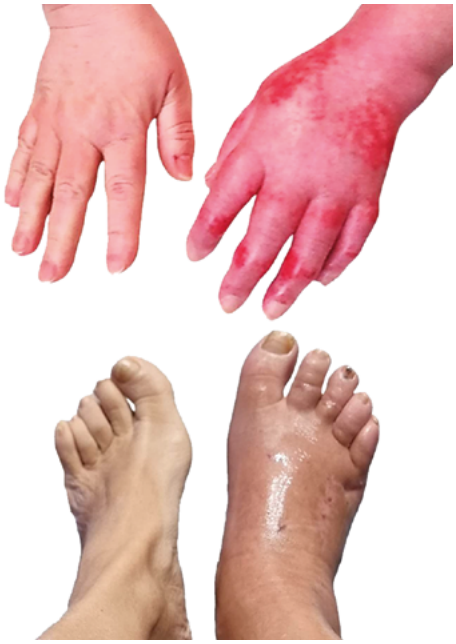

FIGURA 3.
Immagini di sindrome algodistrofica con interessamento della mano e del piede. È evidente l'intenso quadro flogistico apprezzabile nelle fasi iniziali di malattia.

quadro clinico intensamente flogistico tipico della malattia al suo esordio.
Con queste premesse e ad avvalorare i riscontri epidemiologici e clinici precedentemente riportati, vanno considerate altre possibili dinamiche patogenetiche che possono porre in relazione la sindrome algodistrofica con la vitamina D. Tutto ciò a prescindere dal ruolo principale di tale vitamina, ovvero quello connesso con il sistema regolatorio del metabolismo minerale e che collega stati carenziali con un aumento della fragilità scheletrica e del rischio di frattura. Il rischio di contrarre malattie neurologiche caratterizzate da un processo flogistico (neuroflogosi) sembra essere influenzato dai valori plasmatici di vitamina D, così come il loro decorso risulta essere maggiormente gravato da fasi di attivazione in caso di valori inadeguati di vitamina D [12].
A ulteriore riprova di un possibile ruolo patogenetico nei confronti dello sviluppo di algodistrofia, va considerata quella che può essere l'influenza di tale vitamina sulla produzione endogena di citochine proflogistiche. Nel 2014 è stato pubblicato uno studio osservazionale su 957 soggetti ultrasessantenni che, oltre al dosaggio della $25(OH)D_3$, sono stati indagati valutando le concentrazioni plasmatiche di alcune citochine [13]. I livelli plasmatici di IL-6 erano significativamente superiori nei soggetti con valori di vitamina D inferiori a 25 nmol/L rispetto a coloro che presentavano valori normali (> 75 nmol/L). Va qui ricordato che l'IL-6 è una delle citochine la cui concentrazione risulta aumentata sia localmente, in sede di malattia, che a livello sistemico in corso di algodistrofia. Analogo riscontro è stato osservato anche per un'altra citochina proflogogena coinvolta nella patogenesi dell'algodistrofia, ovvero il TNFα. In 69 donne sane di età compresa tra i 25 e gli 82 anni la concentrazione plasmatica della $25(OH)D_3$ è risultata inversamente correlata con i livelli di TNFα [14], citochina anch'essa coinvolta nella patogenesi della sindrome algodistrofica. A ulteriore riprova di un possibile nesso causale tra i livelli di vitamina D e i livelli di citochine proflogogene, sono stati pubblicati studi che dimostrano come la supplementazione vitaminica è in grado di ridurre la concentrazione plasmatica di TNFα IL-1 e IL-6 [15].
Alla luce di tutti i riscontri di letteratura fin qui presi in considerazione, è opportuno che alcune considerazioni finali vadano riportate circa le dinamiche che regolano i rapporti tra la vitamina D e la sindrome algodistrofica.
Non esiste a tutt'oggi alcun riscontro che identifica nella supplementazione con metaboliti della vitamina D una strategia da adottare nel trattamento della sindrome algodistrofica.
Esistono tuttavia dimostrazioni di come situazioni carenziali di vitamina D siano in grado di favorire un aumento di incidenza della malattia algodistrofica. Tale relazione trova il suo razionale nelle seguenti dinamiche patogenetiche:

- situazioni carenziali di vitamina D sono in grado di determinare un aumento di incidenza di eventi fratturativi che rappresentano l'evento predisponente più tipico della malattia;
- situazioni di livelli inadeguati di vitamina D favoriscono il verificarsi di fratture intra-articolari che correlano con maggior frequenza con la comparsa di algodistrofia;
- situazioni carenziali di vitamina D correlano con un maggior rischio di caduta, favorendo in questo modo il verificarsi di fratture che, a loro volta, possono rappresentare l'innesco della malattia algodistrofica.

Circa il ruolo predisponente di situazioni carenziali nell'amplificare le probabilità che l'evento fratturativo si complichi con la comparsa di una sindrome algodistrofica, il tramite patogenetico più verosimile potrebbe essere rappresentato da un assetto immunologico più propenso a tale evento in ragione di aumentati livelli sierici di citochine proflogogene, ovvero i mediatori responsabili dell'innesco e della fase flogistica della malattia algodistrofica. Da questo punto di vista, la supplementazione con vitamina D, in soggetti carenti, potrebbe trovare un razionale nell'ipotesi che la normalizzazione dei livelli plasmatici di $25(OH)D_3$ determinerebbe una ridotta produzione di tali mediatori della flogosi algodistrofica.
Sulla base di tali considerazioni, un aspetto sicuramente meritevole di indagini cliniche potrebbe essere rappresentato da un possibile ruolo terapeutico della somministrazione di vitamina D nei soggetti affetti da algodistrofia. A tal riguardo, disponiamo oggi di strategie terapeutiche che hanno profondamente implementato i risultati ottenibili nel trattamento della malattia. L'efficacia di molecole appartenenti alla classe dei bisfosfonati è da considerarsi un riscontro definitivo, comprovato da studi randomizzati contro placebo e da meta-analisi, ovvero dagli strumenti della "Evidence Based Medicine" dotati dei più elevati livelli di attendibilità. Tra i diversi bisfosfonati, neridronato è la molecola per la quale è stato dimostrato il più elevato e convincente profilo di efficacia, in grado di indurre una remissione di malattia in tempi brevi e mantenuta permanentemente nel tempo [16,17]. A riprova di ciò, neridronato è l'unico bisfosfonato a disporre di una certificazione di efficacia deliberata dall'Agenzia del Farmaco e comprovata dalla specifica indicazione riportata in scheda tecnica.
Poiché il presupposto fondamentale è l'impiego di elevati dosaggi ottenibili solo per via parenterale, tali riscontri sono stati inizialmente ottenuti per il tramite della somministrazione per via venosa, il che implica il trattamento in ambiente ospedaliero con le annesse problematiche di tipo logistico. Da qui il tentativo, purtroppo mai supportato da riscontri di efficacia, di impiegare antiche molecole, disponibili anche per la somministrazione intramuscolare, con quindi la possibilità di un trattamento più rapido e gestibile in ambiente domiciliare. A fronte dell'assenza di riscontri di efficacia impiegando molecole quali il clodronato che, per caratteristiche farmacocinetiche e

farmacodinamiche, non è in grado di esercitare un'accettabile azione terapeutica quando somministrato per via intramuscolare, sono recentemente comparsi in letteratura studi che viceversa documentano per il neridronato una sostanziale analogia di efficacia tra la somministrazione endovenosa e quella intramuscolare [17,18]. A riprova di tale equivalenza, è la recente certificazione AIFA che ha conferito al neridronato l'indicazione per il trattamento dell'algodistrofia anche al farmaco somministrato per via intramuscolare [19].

Pur a fronte dell'elevato profilo di efficacia degli schemi terapeutici che prevedono l'impiego di neridronato parenterale, potrebbe essere un interessante spunto di ricerca l'indagine circa un beneficio aggiuntivo o più rapido quando insieme al bisfosfonato, venga contestualmente utilizzata una supplementazione con vitamina D. Ciò tuttavia in considerazione che sia per il neridronato che per un ipotetico ruolo terapeutico della vitamina D, la condizione essenziale è quella di somministrare il trattamento nelle fasi precoci di malattia, ovvero quando le dinamiche patogenetiche legate all'aumento di concentrazione locale di citochine proflogogene innesca e mantiene le manifestazioni cliniche di malattia.

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VITAMIN D
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La carenza di vitamina D in età pediatrica: un problema che viene da lontano ma che riemerge

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LE PREMESSE, PERCHÉ C'È IL RISCHIO

La carenza di vitamina D in età pediatrica costituisce un problema sanitario con risvolti sociali che possiamo considerare non risolto. Infatti, la vitamina D rappresenta un fattore fondamentale per lo sviluppo del sistema muscolo-scheletrico ed è al centro del processo di crescita del bambino. L'azione classica della vitamina D durante l'età pediatrica è proprio quella di favorire una corretta formazione della massa ossea. La vitamina D viene prodotta per il 90-95% dall'esposizione solare e soltanto per il 5-10% assicurata dall'assunzione con alimenti [1,2]. Questa situazione diventa critica se l'esposizione solare non viene ad essere garantita nei modi e tempi adeguati; ciò può portare a situazioni rilevanti di difetto dei livelli della vitamina D. A questo proposito, uno studio inglese, valutando i dati di prevalenza del rachitismo da deficit di vitamina D, ha rilevato che i casi di rachitismo segnalati in quel paese sono aumentati particolarmente nell'ultimo decennio [3]. Questo è stato attribuito al fatto che probabilmente è cambiata profondamente la componente etnica della popolazione che vive in Inghilterra. La popolazione con colorito della pelle più scuro è aumentata notevolmente, determinando un rischio maggiore in quel Paese, dove la supplementazione, anche nel primo anno di vita, non è consigliata fortemente. Il fototipo scuro infatti non permette un assorbimento completo mediante l'esposizione solare e necessita di una supplementazione [3].

La carenza di vitamina D peraltro è possibile anche in altri Paesi come il nostro dove, anche se l'esposizione al sole è molto più presente, l'aumento del numero di bambini di pelle scura è stato marcato nell'ultimo decennio. Mentre in alcuni paesi del Nord Europa viene applicata una politica di supplementazione negli alimenti della vitamina D (fortificazione degli alimenti) con una diminuzione significativa e diffusa del rischio, nel nostro Paese la mancata supplementazione può aumentare l'incidenza di ipovitaminosi. Anche in Italia e negli altri paesi dell'area mediterranea quindi, questo può costituire un fattore di rischio importante e socialmente rilevante per ipovitaminosi D [4]. A tal proposito diverse società scientifiche hanno stabilito i dosaggi raccomandati ed i tempi di somministrazione della vitamina D per un accrescimento osseo adeguato [2]. Questo vale anche nel corso del primo anno di vita quando la somministrazione è fortemente consigliata ma che in alcune situazioni può trovare una compliance alla somministrazione non adeguata con un aumento sensibile del rischio. Tale rischio si può concretizzare anche in altre fasi della vita pediatrica [2].

IPOVITAMINOSI D: I FATTORI DI RISCHIO

Il rischio di carenza di vitamina D in età pediatrica è spesso dovuto ad alcuni fattori determinanti:

1. alla latitudine: più ci allontaniamo dall'equatore, minore è la quantità di irraggiamento solare disponibile ed utile per aumentare i livelli di vitamina D;
2. all'etnia: il colore della pelle scuro determina un ostacolo alla formazione della vitamina D mediante irraggiamento;
3. a fattori culturali, per esempio, la copertura estrema per motivi religiosi della madre durante la gravidanza determina un rischio di grave ipovitaminosi;
4. alla dieta che può giocare un ruolo determinante, se viene limitata l'assunzione o l'assorbimento di prodotti alimentari che contengono la vitamina.

Nel corso del primo anno di vita la profilassi con 400 UI/die viene eseguita in tutti i lattanti perché tale supplementazione è riconosciuta necessaria per la prevenzione del rachitismo. Le scorte di vitamina D del neonato sono direttamente proporzionali allo stato vitaminico materno, spesso scarse; il neonato e il lattante

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vengono scarsamente esposti al sole e la velocità di crescita staturo-ponderale certamente particolarmente elevata. Inoltre, il latte materno e l'eventuale latte di formula contengono delle quantità di vitamina D spesso non sufficienti. Sebbene la supplementazione nel corso del primo anno sia molto consigliata dai pediatri, per una serie di situazioni le mamme possono ad un certo punto interromperla o essere poco costanti nella somministrazione. Uno studio americano ha evidenziato che spesso il motivo della interruzione era dovuto al fatto di considerare che la vitamina D fosse presente anche nel latte formulato, si trovasse anche in altri alimenti introdotti con lo svezzamento, il bambino venisse considerato già sufficientemente grande [4].

Sono state riportate delle condizioni di rischio di deficit che riguardano anche il bambino di età maggiore ai 12 mesi di vita [1], dovute spesso a regimi dietetici inadeguati: per esempio, una dieta ricca in fitati (questo anti-nutriente, che si trova principalmente in semi, cereali, fagioli e legumi, riduce l'assorbimento di calcio, magnesio e zinco durante la digestione, diminuendone in questo modo l'assimilazione da parte dell'organismo umano), ostacola l'assorbimento della vitamina D [2]. Situazioni di rischio sono dovute a patologie croniche come l'insufficienza epatica cronica e l'insufficienza renale cronica, ma anche l'obesità che costituisce certamente un altro problema sociale e sanitario. La vitamina D è sequestrata dal tessuto grasso e resa non disponibile, abbassando sensibilmente i livelli nel soggetto sovrappeso. Questa situazione che si stima sia presente nel 30% della nostra popolazione pediatrica può costituire un fattore di rischio tra i più importanti per ipovitaminosi D. Vi sono inoltre delle patologie da malassorbimento, come la fibrosi cistica, le malattie infiammatorie intestinali croniche, la celiachia non diagnosticata, che vanno ad ostacolare l'assorbimento della vitamina D. Anche alcuni farmaci, assunti in maniera continuativa per patologie croniche come antiepilettici, fenobarbital, fenitoina, corticosteroidi per via sistemica, farmaci antiretrovirali, antimicotici per via sistemica rappresentano fattori di rischio per ipovitaminosi. Infine, costituiscono un fattore di rischio anche una serie di situazioni come l'immobilizzazione prolungata dovuta, per esempio, a paralisi cerebrale o a malattie neuromuscolari o neurodegenerative [2].

LA VITAMINA D E LE AZIONI EXTRA SCHELETRICHE

Alla vitamina D, specie in età pediatrica, vengono attribuite anche una serie di azioni extra scheletriche, oggetto di continuo dibattito. Molti autori considerano la vitamina D come un ormone pleiotropico: la presenza della vitamina D e l'attivazione dei recettori della vitamina D in cellule diverse, ha degli effetti di omeostasi su diversi organi ed apparati. I recettori della vitamina D sono presenti nelle diverse cellule che costituiscono il sistema immunitario, influenzando sia la componente innata che adattativa [5]. La vitamina D è in grado di interagire sia con i monociti, macrofagi, cellule dendritiche (sistema immunitario innato), ma anche con i linfociti T (sistema immunitario adattativo), modulando la risposta immunologica del bambino. Momenti diversi e successivi di ipovitaminosi D espongono il bambino, che può avere anche altri fattori di rischio, a sviluppare una sensibilizzazione allergica e poi asma bronchiale. Inoltre, altri dati della letteratura confermano che esiste una relazione tra funzionalità respiratoria e livelli di vitamina D. Ciò è evidente già durante la gravidanza: mamme con bassi livelli sierici di vitamina D durante la gestazione partoriscono bambini che presentano una funzionalità polmonare ridotta. Questo rappresenta un fattore di rischio per sviluppare *wheezing* ed ostruzione bronchiale con particolare facilità nei primi mesi di vita [6]. La vitamina D è in grado di modificare in maniera significativa anche l'effetto di alcuni fattori prognosticamente negativi per la funzionalità polmonare del neonato, come l'esposizione al fumo di sigaretta. Se la mamma fuma durante la gravidanza, il fatto che abbia dei livelli di vitamina D corretti, rappresenta un fattore protettivo che sembra neutralizzare l'effetto negativo dell'esposizione al fumo sullo sviluppo polmonare [7].

SUPPLEMENTAZIONE ED EFFETTI EXTRA SCHELETRICI

Nella controversia sul ruolo extra scheletrico della vitamina D, si inseriscono dati discordanti sugli effetti di salute della supplementazione. Un'ampia recente revisione della letteratura, effettuata da endocrinologi, ha evidenziato effetti molto scarsi sulla salute della supplementazione con vitamina D. Non emergono dati a favore della supplementazione per una serie di patologie come il diabete, il cancro, le malattie autoimmuni, la sclerosi multipla, l'asma [8]. Allo stesso tempo però, sono state pubblicate anche altre metanalisi sugli effetti della supplementazione di vitamina D sull'asma durante l'età pediatrica con risultati più favorevoli. Se la supplementazione con vitamina D non è in grado di ridurre il numero delle riesacerbazioni di asma in tutti i bambini asmatici, il rischio di avere asma può essere ridotto nei bambini che hanno abitualmente (vedi fattori di rischio) dei livelli di vitamina D particolarmente bassi, cioè al di sotto dei 10 ng/ml [9]. In un'altra revisione molto recente, dove è stato valutato l'utilizzo della vitamina D per il management dell'asma, gli autori non trovano grosse evidenze per effetto della supplementazione della vitamina D o dei suoi metaboliti nel ridurre il rischio di riesacerbazioni dell'asma. Peraltro, proprio i pazienti più a rischio, che sono quelli che hanno un'asma severa e che presentano dei livelli di vitamina D particolarmente bassi, sono poco rappresentati negli studi oggetto della revisione. Se ne ricava che dal punto di vista pratico il bambino asmatico che dobbiamo considerare più a rischio (asma moderata severa e che presenta dei fattori di rischio per ipovitaminosi D), può giovarsi della supplementazione con vitamina D [10].

La supplementazione durante la gravidanza non sembra essere in grado di prevenire l'asma nel bambino in età scolare, anche se, dati di un trend di efficacia si sono avuti per quel che riguarda la prevenzione del *wheezing* o broncospasmo in età prescolare. Ai tre anni, infatti, vi era una tendenza, peraltro al limite della significatività, nella coorte con supplementazione, per una maggiore protezione dagli episodi di *wheezing* e di broncospasmo. Questo dato è stato recentemente confermato in un'analisi sulla stessa coorte, attribuendolo all'effetto della vitamina già dalla gravidanza sulla funzionalità polmonare e sul sistema immunitario [11].

VITAMINA D E OBESITÀ

Esiste chiaramente anche una relazione tra vitamina D e sindrome metabolica: il 30% dei bambini nel nostro Paese è in sovrappeso e di questi, molti sono obesi. La deficienza di vitamina D sembra influenzare ed essere influenzata dalla sindrome metabolica e dall'obesità. I livelli acquisiti con la supplementazione di vitamina D nel bambino sono condizionati proprio dal peso o meglio dal *body mass index* (BMI) del bambino. In altri termini, supplementare un bambino di peso normale oppure uno con sovrappeso o obesità può dare dei risultati diversi in termini di

TABELLA II.
Fabbisogni giornalieri di vitamina D raccomandati tra 1-18 anni (da Peroni, 2022) [14].

	IOM 2011 e AAP 2012			LARN 2012			Endocrine Society 2011	
Età	EAR, UI/die	RDA, UI/die	UL, UI/die	EAR, UI/die	RDA, UI/die	UL, UI/die	Fabbisogno giornaliero, UI/die	UL, UI/die
1-3 anni	400	600	2.500	400	600	2.000	600-1.000	4.000
4-8 ani	400	600	3.000	400	600	2.000 (4-10 anni)	600-1.000	4.000
9-18 anni	400	600	4.000	400	600	4.000 (11-18 anni)	600-1.000	4.000

EAR: *Estimate Average Requirement* (fabbisogno medio: apporto stimato in grado di coprire i fabbisogni del 50% della popolazione); RDA: *Recommended Dietary Allowances* (assunzione raccomandata per la popolazione: apporto stimato in grado di coprire i fabbisogni di oltre il 97,5% della popolazione); UL: *Tolerable Upper Intake levels* (livello massimo tollerabile di assunzione: apporto al di sopra del quale è possibile l'insorgenza di eventi avversi). * Fabbisogni consigliati per i soggetti a rischio di deficit di vitamina D.

raggiungimento di livelli sierici di vitamina D soddisfacenti. Uno studio ha evidenziato come la supplementazione con dosi convenzionali di vitamina D non sia efficace per modificare i livelli di vitamina D sierici nel paziente obeso [12]. Questo studio ha dimostrato che i bambini con obesità hanno una maggiore resistenza, in termini di mancata risposta, alla supplementazione con vitamina D. Inoltre, un lavoro ha dimostrato come vi sia un effetto condizionante determinato dall'associazione tra BMI e livelli di vitamina D sulla meccanica respiratoria nel soggetto con asma lieve. Infatti, nel soggetto di peso normale livelli corretti di vitamina D erano associati ad una funzionalità respiratoria decisamente più adeguata, cosa che non era evidente in caso di sovrappeso [13].

COME SUPPLEMENTARE LA VITAMINA D IN ETÀ PEDIATRICA?

I dosaggi proposti variano molto ed è importante rifarsi alle dosi che sono consigliate dalle linee guida nazionali e internazionali [2,14]. Solo la supplementazione o la fortificazione degli alimenti fanno la differenza nel mantenimento di livelli adeguati specie nelle categorie dei bambini a rischio. La sola esposizione solare non fa spesso la differenza a tutte le età pediatriche. Infatti, uno studio del 2018 ha dimostrato che soltanto la supplementazione aumenta in maniera significativa ed efficace i livelli di vitamina D nella popolazione pediatrica e nelle gravide [15].

Un punto rilevante ormai chiarito è che le dosi debbono essere assunte quotidianamente e non in bolo (mensile o settimanale). Vi è una spiegazione biologicamente plausibile a questo, che alte dosi in bolo unico possono indurre l'espressione a lungo termine di enzimi del catabolismo della vitamina D che hanno la capacità di inattivare la vitamina somministrata in grosse quantità [16]. Quindi è importante supplementare in maniera giornaliera, costante, precisa, per evitare meccanismi di questo genere. Questo era stato già segnalato nel 2013 in una revisione della letteratura, dove la valutazione del rischio per lo sviluppo di patologie del tratto respiratorio in età pediatrica era ridotto in maniera più significativa nei casi in cui la quantità era somministrata in dose quotidiana e non in bolo [17,18].

CHE DOSAGGI DI SUPPLEMENTAZIONE?

Dobbiamo considerare che la supplementazione è importante per raggiungere dei livelli sierici adeguati (Tab. I). È rilevante per quel che riguarda lo sviluppo di una corretta massa ossea; l'osteoporosi è una patologia che secondo molti inizia in età pediatrica. Quindi assicurare un apporto di vitamina D e di calcio appropriati fin dalle prime fasi della vita è essenziale. Si crea una massa ossea che costituisce un vero e proprio tesoro per le età successive della vita. Peraltro, per raggiungere livelli di vitamina D efficaci per le funzioni extra ossee è probabilmente necessario raggiungere livelli sierici maggiori di quelli che consideriamo utili alla salute dell'osso. Nella prima infanzia tutti i bambini dovrebbero ricevere nei primi 12 mesi di vita, ma probabilmente anche nel corso dei primi 24 mesi di vita, una supplementazione adeguata: 400 e poi 600 unità di vitamina D. Nella seconda e terza infanzia, è importante sottoporre a profilassi con vitamina D i bambini e adolescenti che presentano fattori di rischio di deficit di vitamina D. Lo stile di vita fa la differenza: se il soggetto, bambino o adolescente vive e gioca molto spesso all'aria aperta e mangia in maniera varia, l'esposizione alla luce solare e la dieta assicurano anche un buon assorbimento di vitamina D. Durante i mesi invernali, vale la pena supplementare la vitamina D, considerando ancora una volta la presenza di fattori di rischio come la mancata esposizione solare, il sovrappeso e l'obesità. L'apporto giornaliero sarà di 400 unità giornaliere nel corso del primo anno di vita, per poi variare dalle 600 alle 1000 unità/die. Il soggetto che presenta dei livelli molto bassi di vitamina D, e con una situazione carenziale clinicamente evidente, ha bisogno certamente di maggiori livelli di supplementazione di vitamina D [2].

CONCLUSIONI

I livelli di vitamina D possono essere particolarmente bassi a tutte le età pediatriche, specie in presenza di fattori di rischio. La fortificazione degli alimenti è una situazione che, a livello di popolazione, nei Paesi dove la vitamina D è sempre stata particolarmente bassa, per esempio per scarso irraggiamento, ha contribuito a risolvere problematiche di salute come il rachitismo [17,18]. Ma il problema non è risolto: questo approccio insieme con la supplementazione quotidiana va considerato perché, cambiati i fattori di rischio, con più bambini di pelle scura, cambiati gli stili di vita, il rischio di ipovitaminosi D è aumentato. Anche nei bambini del nostro Paese sono aumentati i casi a rischio e più vulnerabili e per questo motivo gli effetti della vitamina D e l'eventuale supplemen-

tazione dovrebbero essere particolarmente attenzionati e consigliati.

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