Series Editor: T.S. Dharmarajan, MD, FACP, AGSF

Functional Decline in an Elderly Nursing Home Resident

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CASE PRESENTATION

History

A 92-year-old female nursing home resident was transferred to a local university hospital for lethargy and inadequate oral intake. The nursing home staff reported that the patient had eaten poorly for a few weeks and had consumed almost nothing in the 2 to 3 days prior to admission. There was no history of vomiting, fever, chills, or productive cough.

The patient had been bed bound for several months due to advancing Alzheimer's disease. She needed assistance with all activities of daily living (ADL) and was bowel and bladder incontinent. Her ability to swallow solids and liquids without choking or coughing remained intact. Although able to feed herself meals prepared by the nursing home staff, the patient's intake had gradually decreased, leading to a 20-lb weight loss over 6 months. The patient's daughter had brought foods her mother liked and tried to encourage her mother to eat during visits to the nursing home. However, the patient's continued poor eating had necessitated the recent addition of nutritional supplements.

Past medical history was significant for diabetes diagnosed 10 years earlier; a cerebrovascular accident (CVA) 5 years prior, with residual left hemiparesis; Alzheimer's dementia diagnosed 3 years earlier; peripheral vascular disease resulting in left above-knee amputation 2 years prior; long-standing anemia (apparently evaluated in the past, etiology unclear); and multiple pressure ulcers. Medications administered in the nursing home included premixed insulin injection (70 neutral protamine Hagedorn/30 regular) 10 units daily in the morning, metoclopramide 10 mg thrice daily (TID), zinc sulfate 220 mg daily, vitamin C 500 mg daily, generic multivitamin tablet once daily, ferrous sulfate 325 mg daily, cascara 30 mg daily as needed, and milk of magnesia 15 mL daily as needed. Nutritional supplements included Pro-Mod® 2 scoops TID and Resource® Diabetic 1 can TID.

Physical Examination

Physical examination at admission revealed a lethargic but awake patient who was verbally responsive and able to follow commands. Her weight was 102 lb. Left-sided hemiparesis was apparent, consistent with a prior CVA. A feeble dorsalis pedis pulse was noted on the right side. A stage IV, 10 × 12 cm midline sacral ulcer with dull granulation tissue and undermining edges was apparent. Minimal serosanguinous discharge was present, with no evidence of surrounding cellulitis. A stage II ulcer on the left buttock and a right heel eschar also were apparent. The remainder of the examination was unremarkable.

Laboratory and Radiologic Studies

Results of laboratory studies ordered upon admission are summarized in **Table 1**. A recent total cholesterol assay in the nursing home was 153 mg/dL (normal, < 200 mg/dL). Chest radiography was negative for pulmonary infiltrate. Computed tomography of the head revealed mild atrophy with old small corona radiata and basal ganglia infarcts.

- **What factors may have contributed to this patient's recent poor intake and functional decline?**

APPROACH TO THE PATIENT WITH FUNCTIONAL DECLINE

A decline in functional status in an older individual always warrants evaluation. Such deterioration often

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Table 1. Initial Laboratory Values for Case Patient

Variable	Result	Normal Range
Hematologic values		
Leukocyte count (x 10^3/mm^3)	15.9	4.3–10.8
Hemoglobin (g/dL)	8.1	12–16
Hematocrit (%)	24.7	37–48
Mean corpuscular volume (fl)	81	86–98
Platelet count (x 10^3/mm^3)	74	150–450
Serum/plasma values		
Blood urea nitrogen (mg/dL)	14	10–20
Carbon dioxide (mEq/L)	23	21–30
Electrolytes		
Sodium (mEq/L)	140	136–145
Potassium (mEq/L)	3.3	3.5–5.0
Chloride (mEq/L)	112	98–106
Magnesium (mg/dL)	1.5	1.8–3.0
Calcium (mg/dL)	5.9 (corrected, 7.9)*	9–10.5
Inorganic phosphorus (mg/dL)	2.4	3–4.5
Creatinine (mg/dL)	1.2	< 1.5
Glucose (mg/dL)	173	< 126 (fasting)
Total protein (g/dL)	4.6	5.5–8.0
Albumin (g/dL)	1.3	3.5–5.5
Alkaline phosphatase (U/L)	537	30–120

*Calcium corrected for albumin.

Table 2. A Geriatric Functional Continuum

Stage for Potential Intervention	Clinical Correlate
Primary	Independence
	Coping
Intermediate	Independence with difficulty
	The "dwindles"
	Functional decline
Secondary	Frailty
	Failure to thrive
	Disability
	Failure to cope
Tertiary	Dependence
	Taking to bed
	Cachexia
	Pre-death

Adapted with permission from Hamerman D. Toward an understanding of frailty. Ann Intern Med 1999;130:946.

signifies the presence of a new underlying medical illness or recent event (eg, infection) or the decompensation of a chronic problem. In addition, adverse effects of drugs are a common cause of functional decline that should not be overlooked. It is important that health care practitioners recognize factors that contribute to functional decline and intervene to minimize their impact on the quality of life of elderly individuals.

Frailty is a term sometimes used to describe the state of declining health and function in later life. Like the term *failure to thrive* (borrowed from the pediatric vernacular), frailty does not have a precise scientific or clinical definition. Hamerman[1] recently proposed a useful "geriatric functional continuum" that places frailty halfway between functional independence and pre-death (**Table 2**). Although not exclusively a problem of the elderly, frailty typically is observed in the "oldest old" (ie, those beyond the age of 85 years). Conditions associated with frailty include functional decline with inability to perform ADL, chronic diseases, cognitive decline, polypharmacy, dependency on caregivers, hospitalization or institutionalization, and nutritional impairment (ie, reduced oral intake, unintended weight loss, cachexia [loss of muscle mass], serum albumin < 3.5 g/dL).[1]

In the case of this elderly nursing home resident, several conditions likely are interacting to produce a state of frailty. In addition to her advanced age and multiple chronic illnesses, worsening of her Alzheimer's dementia has led to dependence on others for ADL and a progressive decrease in food intake, the latter resulting in severe malnutrition that was evident upon her admission (ie, low serum albumin, total protein, total cholesterol, hemoglobin, calcium). Her 20-lb weight loss (ie, > 10% of body weight) over 6 months was further evidence of malnutrition. Other likely contributors to this patient's poor intake and functional decline include an infectious/inflammatory process related to pressure ulcers or another source of sepsis and adverse effects from medications. Metoclopramide, an antiemetic and gastrointestinal (GI) prokinetic agent, was used in this patient apparently for diabetic gastroparesis and gastroesophageal reflux; a recent report describes functional decline and failure to thrive in older adults perhaps attributable to this drug.[2] Oral iron administration

Table 3. Approach to Assessing Nutritional Status in Older Adults

History
History of alterations in weight, appetite, or taste; changes in food intake
History of GI or abdominal symptoms (anorexia, dysphagia, nausea, vomiting, dyspepsia, diarrhea, abdominal pain)
History suggestive of depression
Use of tobacco, alcohol, or illicit drugs
Current medications (eg, antihypertensives, anticholinergics, antihistaminics, iron supplements)
Socioeconomic status (poor)
Weight loss (> 10% in 6 months or > 5% in 1 month)
Alteration in functional status (ADL, IADL, AADL)
Physical examination
BMI adjusted for age (normal, 18.5–24.9/kg/m^2)
Muscle wasting, skin turgor
Oral/dental problems that interfere with mastication
Signs of vitamin or mineral deficiency (cheilitis, glossitis, cognition related, neuropathy, tetany, bone pain, fractures)
Functional status (ADL, IADL, AADL)
Swallowing evaluation (if indicated)
Diagnostic studies
Hemoglobin, hematocrit
Total lymphocyte count
Renal and hepatic function
Serum levels of total protein, albumin, prealbumin, total cholesterol
Thyroid function tests
Serum levels of iron, iron-binding capacity, ferritin, folic acid, vitamin B_{12}
Chest radiograph
Urinalysis
Fecal occult blood
Anergy panel
Tuberculin skin test
Dietary assessment
Daily energy (caloric) intake (dietary recall, caloric count)
Recent change in dietary habits
Use of vitamins and nutritional supplements
Additional studies
Specific assessment as needed based on evaluation (eg, screening for cancer, depression, infection)

AADL = advanced activities of daily living; ADL= activities of daily living; BMI = body mass index; GI = gastrointestinal; IADL = instrumental activities of daily living.

is known to cause taste disturbances and GI motility disorders.

RECOGNIZING MALNUTRITION IN THE ELDERLY

Adequate nutrition is required for normal homeostasis. The body cannot function effectively or recover well from illness in the absence of essential macro- and micronutrients. Unfortunately, nutritional deficits and malnutrition are common and often underrecognized problems within the elderly population. Among older persons, it is estimated that up to 15% of ambulatory outpatients, 35% to 65% of hospitalized patients, and 25% to 60% of long-term care (nursing home) residents have malnutrition.[3] Even in the hospital setting, many older adults are maintained on less than adequate nutrient intake for energy requirements, contributing to nutritional deficits and a high mortality risk.[4] Routine oral or enteral nutritional supplementation seems to improve nutritional status, with benefits not restricted to patient groups.[5] Thus, increased efforts to recognize the signs and symptoms of malnutrition among the elderly are warranted.

Assessing Nutritional Status in the Elderly

Table 3 lists steps in assessing the elderly patient for malnutrition. Malnutrition results from imbalance between energy intake and expenditure and is suggested by the presence of unintentional weight loss. Unintentional weight loss of 1% to 2% in 1 week, 5% over 1 month, or 10% in 6 months warrants evaluation.[6] Among the elderly, weight loss usually is the result of decreased intake rather than increased level of activity and, thus, energy utilization.[7] Increased energy requirements may occur, however, in chronic infections (eg, pressure ulcers, sepsis), with dementia as a compounding factor.[7,8] In most situations, loss of appetite associated with weight loss is caused by medications or disease and should be investigated. Other causes include oral or dental problems; difficulty swallowing from any cause; needless dietary restrictions; comorbid conditions causing anorexia or malabsorption; and polypharmacy leading to nausea, interference with taste, or absorption. In addition, decreased intake may be due to unavailability of food (relating to financial status or restricted mobility) or the inability to plan and prepare meals. Speech and swallow evaluation may be indicated if dysphagia is suspected.

Several clinical and biochemical parameters are useful for assessment of nutritional status in older adults. Serial weight measurements are inexpensive and easily obtained in the office, hospital, or nursing home setting. Anthropometric measures include body mass index,

skinfold thickness, and mid-arm circumference. Useful laboratory tests include serum albumin or prealbumin, total protein, cholesterol, transferrin, hemoglobin, total lymphocyte count, and anergy panel (correlates with immune defense mechanisms). Low serum albumin, which reflects nutritional status in the previous 21 to 28 days based on the half-life of albumin, is an independent risk factor for poor outcome and increased mortality. Serum albumin has been well studied as a prognostic marker and falls only when malnutrition is moderately severe and prolonged or accompanied by trauma or catabolic states such as infection (acute phase reactant).[3,9] Albumin levels also can be low in the presence of renal losses or hepatic disease. In acute hospital settings, prealbumin level is helpful in that it has a shorter half-life than albumin (2 to 3 days).[3,9]

Key Point

Malnutrition is common in the frail elderly and often multifactorial in basis. Several clinical and laboratory parameters are used for an objective diagnosis. Anorexia is not solely a result of the aging process; illness and medication side effects often contribute to poor appetite and intake.

- **What measures are appropriate for improving the nutritional status of an older adult?**

ADDRESSING NUTRITIONAL DEFICIENCIES

The first step in addressing nutritional deficiencies in older adults is to identify underlying causes leading to malnutrition and to correct or modify those that are reversible. Subsequent efforts should be aimed at trying to enhance the patient's oral intake.

Several strategies may help improve oral intake in an elderly patient who is malnourished. These measures include removing needless dietary restrictions, providing preferred food choices and additional portions, offering between-meal snacks, and adding calorie-rich foods (eg, ice cream, desserts) or nutritional supplements. Strategies for improving motivation include seating the patient with preferred company, having family members present during meals, and improving the environment, if at all possible. Kindness, humor, and verbal persuasion can be helpful.[6] Individuals with restricted mobility, who have difficulty buying or cooking meals, can be helped by caregivers or individuals in their social network or through local programs such as "meals on wheels." Cognitively intact individuals benefit from repetitive reminders to improve feeding and nutrition.

For the case patient, it would be appropriate to take steps to identify and treat sources of infection or inflammation, which most likely are her pressure ulcers. In addition, the patient's drug regimen should be addressed; iron, metoclopramide, and vitamin C all can be temporarily withheld. Finally, it would be appropriate to redouble efforts to enhance her oral intake by providing foods of her preference (even if the food choices are not in the best interest of managing her diabetes) and giving importance to the quality of life. If attempts to improve oral intake fail, the remaining options are parenteral nutrition and enteral nutrition via a nasogastric tube or percutaneous endoscopic gastrostomy (PEG) tube. However, it is important to exhaust all strategies for improving oral intake before initiating tube feeding.[10,11]

Table 4. Results of Anemia Testing in Case Patient

Variable	Result	Normal Range
Serum iron (μg/dL)	18	50–170
TIBC (μg/dL)	194	250–450
Serum ferritin (ng/mL)	75	10–120
Serum folate (ng/mL)	> 20	3–16
Serum vitamin B_{12} (pg/mL)	933	200–900

TIBC = total iron-binding capacity.

INITIAL MANAGEMENT OF CASE PATIENT

The patient's anemic status on admission prompted a packed red blood cell transfusion and an evaluation for anemia, with results noted in **Table 4**. Post-transfusion, the patient's hemoglobin and hematocrit values were corrected to 11.9 g/dL and 35%, respectively. The patient was treated with empiric antibiotics for underlying sepsis while awaiting culture results. Her pressure ulcers were treated with occlusive dressings, and a surgical consultation was requested for possible débridement.

- **Was this patient's anemia a result of her aging?**

ANEMIA IN THE ELDERLY

Anemia is a sign of underlying illness, which warrants evaluation for a cause and appropriate treatment. Anemia is not a part of normal aging, but its prevalence increases with age and is highest among those older than 85 years.[12]

The most common cause of anemia in older adults in the community is iron deficiency, whereas chronic disease is the most likely cause in the hospitalized elderly (**Table 5**). It is not uncommon for iron-deficiency anemia and anemia of chronic disease to coexist, and at

Table 5. Comparative Features of the Common Causes of Anemia in Older Adults

Feature	Iron Deficiency	Chronic Disease
Pathogenesis	Inadequate food intake, malabsorption, GI or other blood loss	Cytokine-mediated blunted response to erythropoietin Abnormal mobilization of iron stores
Serum iron	Low	Normal or low
TIBC	Increased	Low to normal
Serum ferritin	Low	Normal to high
Bone marrow iron	Depleted	Normal
Red cell morphology	Hypochromic, microcytic	Normochromic, normocytic Can be hypochromic, microcytic
Treatment	Iron supplements (oral or parenteral)	Treat underlying condition

GI = gastrointestinal; TIBC = total iron-binding capacity.

times the diagnosis remains unclear even after extensive evaluation.[13] Medications that commonly cause bleeding in the elderly include aspirin, nonsteroidal anti-inflammatory agents, steroids, and anticoagulants. With iron deficiency, common causes of GI blood loss in the elderly (ie, peptic ulcer disease, vascular malformations, neoplasms, diverticular disease) should be excluded. Other causes of anemia in the geriatric population include vitamin B_{12} deficiency, folic acid deficiency, sideroblastic anemia, myelodysplastic syndrome, multiple myeloma, lymphoma, and leukemia.

Symptoms of anemia may be nonspecific and may include fatigue, generalized weakness, and poor oral intake. However, the presentation also may be misleading in that anemia may be the precipitating factor for cardiac failure, myocardial infarction, falls, or stroke.

Diagnostic tests for anemia include complete blood count, mean corpuscular volume, serum iron and total iron-binding capacity (TIBC), serum ferritin, and evaluation for GI and other sources of blood loss. Serum ferritin level is the most useful noninvasive test for diagnosing iron-deficiency anemia[12]; levels less than 18 ng/mL are specific for iron-deficiency anemia, whereas levels above 100 ng/mL exclude iron deficiency. As serum ferritin is an acute phase reactant, it is not a reliable indicator in the presence of infection or inflammation.

Treatment of iron deficiency typically is oral iron supplementation in the form of ferrous sulfate, the most economical effective form. Elixir treatment is available for patients on tube feeding. Although anemia is corrected in weeks, continuing therapy for about 3 to 6 months helps replenish iron stores. Acute blood loss may be corrected with packed red blood cell transfusion—the quickest method, particularly in the frail elderly. In anemia of chronic disease, the body has adequate iron stores but mobilization of stored iron is poor and unavailable to synthesize hemoglobin. Cytokines such as tumor necrosis factor, interleukin-1, and interferon are key factors responsible for shortened red blood cell survival and blunted erythropoietin response in this setting. Common causes of anemia of chronic disease are infection, inflammatory conditions (eg, rheumatoid arthritis), pressure ulcers, renal failure, hematologic malignancy, and cancer.[12,13] Treatment of anemia of chronic disease is directed at the underlying etiology (eg, erythropoietin for anemia of chronic renal failure; control of infection for anemia in osteomyelitis).

Returning to the case patient, the anemia workup revealed normal vitamin B_{12} and folate levels, ruling out deficiency of either vitamin as a cause of her anemia. Vitamin B_{12} status would be particularly relevant in this case because deficiency is a well-recognized cause of both anemia and neuropsychiatric disturbances, such as dementia.[14] Although the patient's transferrin saturation (a calculation based on serum iron and TIBC) was 9% and thus suggested iron deficiency (values below 20% are diagnostic), her ferritin level was not low but high. The elevated ferritin value reflects an acute phase reactant effect, most likely the result of the patient's pressure ulcers. With evidence of infection, inflammation, and malnutrition, it is conceivable that this nursing home resident had both iron-deficiency anemia and anemia of chronic disease. Correcting the patient's anemia, therefore, would entail iron administration and treatment of her pressure ulcers. Management of pressure ulcers is particularly challenging in the setting of advanced dementia.

Key Point

Anemia is not a part of normal aging and warrants evaluation for an underlying cause, the most common being iron deficiency and chronic disease. Iron-deficiency anemia usually is due to blood loss from the GI tract or another source. Low serum ferritin is an indicator of iron deficiency, although ferritin values may be normal or elevated in the presence of infection or inflammation.

FURTHER DETERIORATION OF CASE PATIENT

While hospitalized the patient remained alert but had no improvement in her oral intake, despite her daughter's efforts to bring and feed her foods that she preferred. A 3-day caloric count by the hospital nutritionist confirmed less than 30% of the daily requirement. Speech and swallow evaluation did not reveal any abnormality. A nasogastric tube was inserted on hospital day 5 as a temporary method to feed, while a family meeting was scheduled to discuss options for further management. In anticipation of this discussion, the daughter confirmed that her mother had no living will or any prior expressed wishes, written or verbal, regarding future modalities of treatment.

- **Would PEG tube placement be indicated for this patient?**

CONSIDERATIONS IN THE DECISION TO INSERT A PEG TUBE

The initial approach to a patient with poor oral intake always should be to provide help with feeding, to offer a variety of foods (to suit preferences), and to avoid restrictive diets (taking into account comorbidities). Failure of such measures often leads to consideration of enteral feeding as the next step—either temporarily via a nasogastric tube or more permanently via PEG tube placement. Such was the case with this elderly patient. After several attempts to improve her oral intake failed to improve her nutritional status and to halt the progression of her illness, a nasogastric tube was inserted. At this point, it would be appropriate to discuss the relative risks and benefits of further options, including PEG tube placement.

Indications for PEG Tube Placement

PEG is a long-term means of providing fluids, nutrition, and medications to an individual via a tube inserted through the abdominal wall into the stomach, guided by an endoscopic approach. A functional GI tract is essential for PEG tube placement. Common indications for PEG include chronically poor oral intake due to dysphagia from neurologic causes, such as cerebrovascular disease or advanced neurodegenerative disease (eg, Alzheimer's dementia, Parkinson's disease), or as the result of a neoplasm (typically esophageal cancer). The inability to eat for short periods of time (ie, less than 2 or 3 weeks) secondary to a correctable or reversible condition is not an indication for PEG and may be best treated by a nasogastric tube. Recurrent aspiration pneumonia also is not an indication for PEG.[10,15]

Risks and Benefits of PEG Tube Placement

Prior to PEG tube placement, it is essential to discuss the risks and benefits of long-term tube feeding with the patient (if capacity is intact) or surrogate and caregiver. It should be made clear that the final decision must be guided by the patient's own wishes, especially if expressed previously.[11,15,16] A nutritionist, nurse, and psychosocial worker ideally are included in these discussions, as nutritional, nursing, social, and ethical issues are involved. The discussion should address potential complications of PEG tube insertion, expected outcomes and impact on the patient's quality of life (**Table 6**), care of the feeding tube, and responsibilities of the caregiver.[10,11,15,16] It is important to avoid undue expectations on the part of the patient or caregiver. Also, it should be borne in mind that PEG does not preclude oral intake; the tube can be removed if the primary condition for which the tube was placed has been ameliorated and adequate oral intake is once again achieved. In the elderly, oral intake provides satisfaction and contributes to quality of life. Nevertheless, many health care providers do not find it easy to limit or stop tube feeding once it has been initiated.[11]

Safety studies suggest a low rate of mortality and major complications when PEG tube placement is performed by an experienced practitioner.[17] However, data suggest that the 30-day perioperative mortality is 6% to 28%.[18,19] Survival after PEG tube placement for more than 50% of patients is between 3 and 12 months.[10,18–20] In addition, aspiration pneumonia, diarrhea, dislodgment or clogging of the tube, and insertion site cellulitis are commonly associated complications.[15,21] In terms of benefits, data from most studies suggest that PEG has not improved the quality of life or prolonged survival in the majority of older adults. However, prospective placebo-controlled double-blind studies are difficult to perform because of ethical concerns.

In summary, the decision for PEG tube placement involves consideration of the expressed wishes of the patient in addition to the physician's clinical judgment. Feeding is a sentimental issue, and the decision to

Table 6. Impact of PEG Tube Placement on Outcomes in Patients with Dementia

Outcome	Impact of PEG Tube Placement	Reference
Survival	Prolongation of survival not meaningful in dementia; most individuals survive < 1 year.	10,18–20
Nutritional parameters	No consistent long-term improvement seen. Weight loss (lean and fat body mass) persists.	18,20
Aspiration pneumonia	Enteral feeding is an independent risk factor for aspiration; aspiration on tube feeding is a cause of death in the cognitively impaired.	27,28
Pressure ulcers	No beneficial effect on new ulcer development or consistent effect on healing of existing ulcers.	27,29
Infection	No clear decrease in incidence of infection.	27
Functional status	No improvement in performance of ADL or IADL	18,27
Quality of life	Social isolation and depression common; loss of the joy of eating.	10,18,27,30,31
Comfort	Tube may cause discomfort; efforts to pull out the tube may result in injury. Behavioral changes may require restraints.	18,27,30,31

ADL = activities of daily living; IADL = instrumental activities of daily living; PEG = percutaneous endoscopic gastrostomy.

withdraw or withhold feeding involves moral, ethical, and legal concerns. Tube feeding is considered a medical intervention, not obligatory care.[11,16,22] Artificial nutrition and hydration are considered medical treatment and may be withheld with clear-cut evidence that the patient does not want the therapy (if capacity is intact) or would not have preferred it (through an expressed wish in a living will or an advanced directive).[22]

Key Point

Long-term feeding via a PEG tube should be a consideration only after all other means of oral feeding have been exhausted. A detailed discussion of the risks and benefits of PEG and alternative feeding approaches should occur between the health care provider and patient (based on capacity) or surrogate prior to the decision to proceed with PEG. Most data do not support long-term benefits to health, survival, or quality of life following PEG tube placement.

LATE HOSPITAL COURSE OF CASE PATIENT

On rounds later that day, the patient appeared to be in discomfort and complained of pain in her extremities. Examination demonstrated positive Chvostek's sign (twitching of facial muscles) and Trousseau's sign (carpopedal spasm), suggesting tetany. The patient's serum calcium at this time was 4.2 mg/dL (normal, 9–10.5 mg/dL). A 25-hydroxyvitamin D [25(OH)D] measurement was 9 ng/mL (normal, 10–55 ng/mL), confirming the presence of vitamin D deficiency.

- **Did this patient likely have coexisting osteomalacia and osteoporosis?**

OSTEOMALACIA IN THE ELDERLY

This patient's very low 25(OH)D value was suggestive of vitamin D deficiency–induced osteomalacia. The presence of vitamin D deficiency would not be surprising in a malnourished patient who was eating poorly and was bed bound, with no exposure to sunlight (no dermal synthesis of vitamin D). Combined with the clinical features of twitching from hypocalcemia and muscle pain, vitamin D deficiency in the presence of malnutrition essentially confirms the diagnosis of osteomalacia.

Osteomalacia refers to a qualitative defect in bone mineralization. Osteomalacia from vitamin D deficiency is a common but underrecognized cause of hypocalcemia in the geriatric population. Vitamin D deficiency and resultant hypocalcemia can be severe enough to produce tetany. Although vitamin D deficiency can be multifactorial in older adults, the most common basis for osteomalacia is inadequate oral intake. Dietary vitamin D is available in the United States in fortified food (eg, dairy products, orange juice) or as nutritional supplements. The other important source of vitamin D is synthesis in the skin through the action of ultraviolet B light. Thus, homebound or bed bound individuals with restricted mobility are at risk for vitamin D deficiency. The incidence of hypovitaminosis D in hospitalized adults may be as high as 57%.[23] An additional cause of osteomalacia is hypophosphatemia resulting from poor

oral intake, diminished GI absorption, antacid use, or excessive urinary excretion of phosphorus.

Osteoporosis, another common bone disorder in older adults, often coexists with osteomalacia. In contrast to osteomalacia, osteoporosis is characterized by a decrease in bone mass (quantitative defect), with intact mineralization. It is quite likely that with vitamin D deficiency and hypocalcemia, the case patient also had osteoporosis. Among the many secondary causes of osteoporosis, the most obvious cause in older adults is age-associated decreases in hormone levels (estrogen or testosterone).

Dietary supplementation with calcium and vitamin D reduces bone loss at the femoral neck and spine and reduces the risk of nonvertebral fractures.[23,24] Calcium and vitamin D administration also can help reduce the risk of falls in the elderly.[25] Muscle and bone pain and muscle weakness are typical features of osteomalacia.[26] Falls are a consequence of musculoskeletal involvement.

Key Point

Osteomalacia from vitamin D deficiency is common but underrecognized in the geriatric population. Presentation can be asymptomatic or relate to muscle and/or bone involvement. Low 25(OH)D levels are diagnostic of osteomalacia from vitamin D deficiency.

DEATH OF CASE PATIENT

The following afternoon, the daughter was scheduled to meet with the physician, nutritionist, nurse, and psychosocial worker to discuss feeding options for her mother, including PEG tube placement. However, her mother died prior to the discussion.

CONCLUSION

In the end, the difficult decision regarding placement of a PEG tube for long-term feeding in this patient was avoided, and the reader is left to contemplate the complex issues that would have been considered in the discussion. This was a classic story of an elderly nursing home resident who had progressed to a point of functional decline where it was too late to reverse the tide. At this point, it would be very difficult to justify extreme measures to improve this woman's nutritional status. It is unlikely that placement of a PEG tube at this late stage would have improved the quality of her life or substantially prolonged her survival.

In this elderly woman, functional decline was most likely the result of worsening dementia and a progressive decrease in food intake resulting in severe malnutrition, evident at hospitalization. Besides dementia, factors contributing to her poor intake included infection/inflammation related to pressure ulcers or additional sources of sepsis and adverse effects on appetite from medications. Superimposed delirium from infection cannot be excluded. Furthermore, this nursing home resident's poor functional status led her to become bed bound. Poor food intake and lack of sunlight exposure led to severe vitamin D deficiency and hypocalcemia over time, manifesting as tetany prior to death. Timely screening and attention to her nutritional status, with administration of vitamin D and calcium, would have prevented tetany; osteomalacia is underdiagnosed in older adults and is a preventable disease.

Decline in functional status in older individuals always warrants evaluation. Deterioration in function may be a result of progressive cognitive decline, an underlying new illness, decompensation of a chronic problem, or an adverse drug event. Early intervention, particularly for those individuals who are still relatively independent, is key to maximizing function and quality of life in the elderly. Finally, malnutrition is common and underdiagnosed in the geriatric population. Timely nutritional assessment and institution of corrective measures may help prevent the functional decline and poor outcome seen in this case patient. **HP**

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