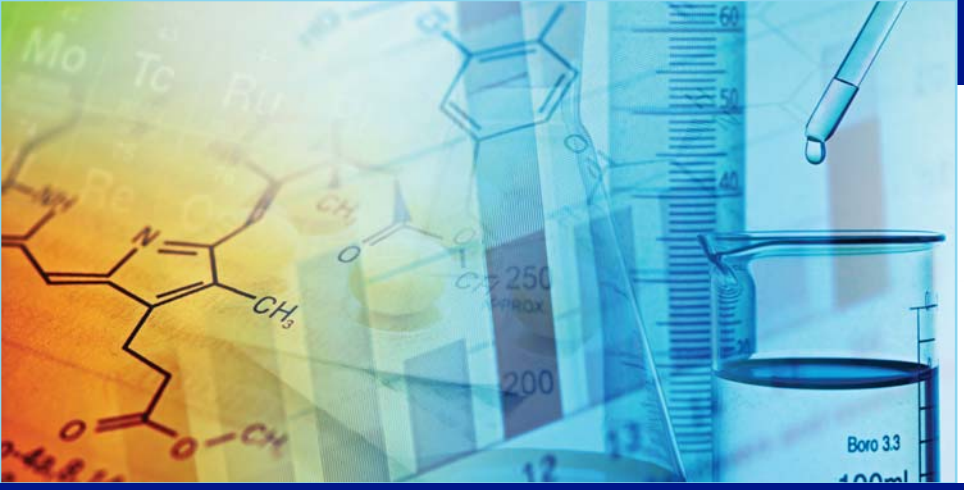

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Managing Iron Deficiency Anemia in Patients on Home Parenteral Nutrition

SAMANTHA SULLIVAN, BS, DIETETIC INTERN AND SANDRA I. AUSTHOF, MS, RD, LD, CNSC

Iron deficiency anemia (IDA) is estimated to occur in 1-2% of all adults in the U.S.[1] In home parenteral nutrition (HPN) patients, who have no enteral source of iron, the rate of iron deficiency anemia has been reported to occur in 40-55% of all adults[2,3] due to blood loss from the gastrointestinal (GI) tract, multiple surgeries, reduction of gastric acid (from medications), iron malabsorption, and lack of supplementation in parenteral nutrition (PN). IDA can develop within 2 to 149 months (mean 27.2 months) after HPN begins and occurs faster in patients with fistulae and bowel obstruction compared to short bowel syndrome and dysmotility.[2] The addition of iron to PN is rare due to risk of anaphylaxis and concern for incompatibilities with lipids.[3] Iron is not found in standard multiple trace element preparations except Addamel™N (Fresenius Kabi USA, LLC). Another concern is that parenteral iron may impair immune function and stimulate bacterial growth during acute infections.[3] This review will discuss iron metabolism, etiology of IDA, how to identify IDA, treatment, and monitoring.

Iron Metabolism

Iron is an essential nutrient required for oxygen transport, DNA synthesis, electron transport, and cell proliferation.[4,5] It is found in every living cell with 60% in the form of hemoglobin in circulating erythrocytes, 20% stored as ferritin,15% found in myoglobin, and the remaining 5% as enzymes and other proteins.[4,6] Iron balance, production and clearance of red blood cells (RBC), is tightly regulated by macrophages since there is no excretory pathway and excessive amounts can lead to tissue damage.[4,7] The absorption (1-2 mg/day) of iron takes place mainly in the duodenum and proximal jejunum as heme and non-heme due to the acidic medium. Heme iron, from animal sources, is absorbed more readily (15-25%) from the GI tract than plant sources or non-heme iron (2-20%) and both are absorbed in the ferrous form (Fe^{2+}) by different carrier proteins. Absorption is enhanced by ascorbic acid, meat, poultry, and fish. Calcium, dairy products, fiber, tea, coffee, spinach, legumes, antacids, proton pump inhibitors, and H2 antagonists all decrease iron absorption.

Once absorbed, iron is either stored in the ferric form (Fe^{3+}) or transported to the blood stream as Fe^{2+} via ferroportin, an iron transporter present on cells of the proximal small bowel, allowing iron entry into the plasma. During inflammation or infection, hepcidin, a peptide hormone synthesized primarily by the liver, binds and degrades ferroportin, which inhibits iron from entering the plasma.[5]

Iron must be oxidized to Fe^{3+} by hephaestin, a copper-containing enzyme, which releases iron into circulation, to attach to transferrin in the plasma. Once in the plasma, iron is transported to cells or bone marrow to produce red blood cells. Erythropoiesis is stimulated by the hormone, erythropoietin, which is secreted by the kidneys. Many nutrients, in addition to iron, are required in the development of RBC synthesis such as biotin, folate, zinc and the vitamins A, B_6, and B_{12}. A deficiency in any of these nutrients can cause inadequate synthesis of RBCs leading to anemia.

Etiology of IDA

There are four major causes of IDA.[4-6] The first is increased loss of blood from acute or chronic bleeding or multiple surgeries. Next is increased demand for oxygen during rapid periods of growth such as pregnancy, infancy, childhood, adolescence, and also during menstruation. The third cause is decreased intake or absorption due to lack of dietary iron. This would include patients on long-term PN without iron therapy or who may have damage to the intestinal lining of duodenum and proximal jejunum, as can be seen in Crohn's disease and Celiac disease. In addition, consuming foods that inhibit absorption or taking medications that reduce the production of gastric acid (antacids,

continued on page 2...

proton-pump inhibitors, and H2 blockers) can lead to IDA. The last cause of IDA is the decreased production of red cells as seen in chronic renal disease from decreased production of erythropoietin.

Identifying Iron Deficiency Anemia

IDA can be confirmed from the medical and surgical history, a physical exam, and procedures, but it is primarily diagnosed from serum lab tests. The complete blood count (CBC) can quickly evaluate the serum blood cells and is routinely used to identify IDA by assessing the hemoglobin (Hgb), hematocrit (Hct), mean corpuscular volume (MCV), and red cell distribution width (RDW) (see Table 1). Hgb and Hct will not show depletion until the patient is depleted of all iron stores, erythrocytes become deficient in iron, the cells start to become hypochromic, and blood levels cannot meet daily needs. Common characteristics of IDA are abnormally small blood cells (microcytosis) and pale blood cells (hypochromia).[9] Since RBCs decrease in size during IDA, the MCV, which measures the average RBC size, will be low (<80 fL). The RDW measures the variation in RBC size. In IDA, the RDW level will be elevated since RBCs will be unequal in size (a mix of both large cells and small cells) also known as anisocytosis. The next set of labs to order after the CBC, is iron studies (see Table 1). The iron studies consist of serum iron, ferritin, total iron binding capacity (TIBC), and transferrin saturation percent (%Sat). Serum iron is a measure of all iron in the body that is mostly bound to transferrin. Ferritin is stored cellular iron. Both are low in iron deficiency anemia. Ferritin will be elevated during the presence of inflammation since it is a positive acute-phase reactant protein.[4,9] TIBC measures the amount of circulating transferrin that is available to bind iron. It will be elevated in iron deficiency anemia since the capacity to bind iron is high, but iron is not available. The %Sat measures the amount of circulating transferrin that is available to bind iron and will be low in IDA.

IDA is clinically manifested in many ways. Frequently reported symptoms during a physical exam include fatigue, pallor (pale skin, gums, and nail beds), rapid or irregular heartbeats, and headaches or problems concentrating. In addition, brittle or spoon-shaped nails (koilonychia), the desire to eat ice, chalk, clay, glue, or other non-food items (pica), restless leg syndrome, shortness of breath, and glossitis may also be present.[5] An endoscopy or colonoscopy can determine the presence of gastric bleeding or colon and rectal bleeding, respectively.

Treatment

Once IDA is confirmed, treatment is required. The total iron deficit and the IV iron repletion dose can be calculated (see Table 2). There are two medically proven options to replace iron stores in patients: oral and intravenous (IV) supplementation.[10] Oral supplementation is the preferred method of treatment because it is safer and more cost effective (see Table 3). The recommended oral dose is 100-200 mg of elemental iron daily. Iron supplementation that is taken with ascorbic acid can increase absorption. The down side to oral supplementation is the GI-related side effects, such as diarrhea, constipation, nausea, and abdominal pain.[11] Oral iron supplementation may be taken with food to help reduce side effects, but absorption may be decreased. Reducing the oral dose or dividing in multiple doses throughout the day can decrease GI-related side effects yet still be effective and improve patient adherence.[12]

Even though oral supplementation is safer and more cost effective, IV preparations are the most efficatious.[10, 12] IV therapy provides a faster response rate. For patients with inflammatory bowel disease, IV iron is commonly used in severe anemia (Hgb <10 g/dL), patients experiencing intolerance to oral iron, having a lack of response or in need of fast recovery.[12] Currently, there are five IV treatments on the market (see Table 4). Ferric gluconate, iron sucrose, ferumoxytol, and ferric carboxymaltose need to be administered multiple times before full replacement of iron can be achieved. Low molecular weight (LMW) iron dextran can be given in one administration (total-dose infusion) to replace iron. LMW iron dextran is the only IV iron that can be added to non-lipid PN.[2] Due to the instability of total nutrient admixtures, the positive charge (cationic) in ferric iron can neutralize the negative charge (anionic) surface in fats which results in the breakdown of the emulsion.[13]

All intravenous iron preparations can be associated with acute reactions such as abdominal pain, nausea, chest pain, dyspnea, flushing, pruritus, hypotension, and anaphylactic reactions. During the 1980's and 1990's high molecular weight (HMW) iron dextran formulas were being used in the United States. These formulas effectively replaced iron stores; however, the risk of adverse drug effects (ADEs) such as anaphylaxis was high and resulted in the production of non-dextran formulations. The non-dextran formulations were associated with minimal ADEs, which made them more appealing. In 2009, the Food and Drug Administration placed a black box warning on iron dextran requiring that a test dose be administered with resuscitation equipment and personnel trained in the detection and treatment of anaphylactic-type reactions readily available during the infusion.[14]

One retrospective review[15] described that the use of IV iron sucrose, sodium ferric gluconate, and LMW iron dextran infusions were associated with lower risks of adverse reactions compared to HMW iron dextran. In a study conducted by Koutroubakis,16 fifty patients with anemia were given LMW iron dextran. Four patients (8%) developed adverse reactions and one patient developed anaphylaxis. It was concluded that LMW iron dextran was safe.

Monitoring

Patients receiving HPN should be screened for anemia every 6-12 months when stable and every 3 months when iron deficiency exists to ensure repletion.[17] Iron deficiency anemia is usually resolved after 6-8 weeks of treatment. Inadequate response may be related to continued blood loss, inflammation, ineffective absorption, or poor adherence. Hemoglobin should

begin to improve by 1-2 g/dL the first 2 weeks, then 0.7 -1 g/dL every week thereafter.[6,18] Pica or restless leg syndrome should disappear once therapy has begun. The reticulocyte count should rise after one week of therapy[9] and if it increases within 4 weeks, treatment is probably effective. Ferritin levels may take up to 32 weeks to improve. Once hemoglobin normalizes, CBC and iron studies should be monitored every 3-4 months up to a year. Therapy should continue until iron stores are repleted.

Conclusion

Iron deficiency anemia is a common complication occurring in one-third to one-half of patients on long term PN because iron is not typically added to PN mixtures and many of them suffer from conditions/diseases that put them at high risk for developing IDA in the United States. Patients with IDA can exhibit symptoms of fatigue, shortness of breath, rapid or irregular heartbeats, and glossitis all which affect quality of life. Low molecular weight iron dextran is the only IV iron that is compatible in non-lipid containing PN formulas; however, other IV sources can be given outside PN. It is imperative that this patient population be monitored regularly and supplied with iron therapy as needed.

TABLE 1:
Labs Values in Normal and Iron Deficiency Anemia

Lab Value	Normal Values	Fe Deficiency Anemia
Hemoglobin	Women: ≥12.0 g/dL Pregnant Women: ≥11.0 g/dL Men: ≥13.0 g/dL	Women: <12 Men: <13 males
Hematocrit	Women: >36 % Pregnant Women: >33 % Men: >39 %	
Mean Corpuscular Volume (MCV)	80 – 100 fL	<80 fL
Red Cell Distribution Width (RDW)	11.5 – 15 %	
Iron	Women: 50 - 170 mcg/dL Men: 60 - 170 mcg/dL	<40 mcg/dL
Ferritin	Women: 12 - 150 ng/mL Men: 12 - 300 ng/mL	≤15 ng/mL
Total Iron Binding Capacity (TIBC)	240 - 450 mcg/dL	>450 mcg/dL
Transferrin Saturation Percent (%Sat)	20 - 50% (Serum iron ÷ TIBC x 100 = %Sat)	<15 %

TABLE 2.
Determining Total Iron Deficiency and Iron Repletion Dose

Formula to determine total iron deficit:
Total iron deficit (mg) = weight (kg) x (ideal Hgb – actual Hgb [g/dL]) + depot iron (500 mg)
Formula to determine IV iron repletion dose:
Iron (mg) = 0.3 x body weight (lbs) x (100 – [actual Hgb (g/dl) x 100/desired Hgb]

TABLE 3.
Oral Iron Replacement Treatments

Oral Iron Treatment	Brand Name	Tablet dose	Elemental Iron
Ferrous sulfate	Feosol Feosol elixir	325 mg 5 mL	65 mg 44 mg
Ferrous gluconate	Fergon Fergon elixir	325 mg 5 mL	36 mg 34 mg
Ferrous fumarate	Feostat oral suspension	325 mg 5 mL	106 mg 100 mg
Iron polysaccharide	Niferex Niferex Elixir	150 mg 5 mL	100 mg 100 mg

TABLE 4:
Intravenous Iron Replacement Treatments

Generic Name	Brand Name	Dose
Low weight-iron dextran	INFed (50 mg/mL elemental Fe)	500-1000 mg over 1 hr
Ferric gluconate	Ferrlecit Nulecit (12.5 mg/mL elemental Fe)	max 125 mg over 20-30 mins
Iron sucrose	Venofer (20 mg/mL elemental Fe)	200 mg over 60 mins
Ferumoxytol	Feraheme (30 mg/mL elemental Fe)	510 mg over 15 mins
Ferric carboxymaltose	Injectafer (50 mg/mL elemental Fe)	up to 750 mg over 15 mins

continued on page 4...

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Have you heard about the 17th Martha Nelson Lewis Symposium?

A long-standing tradition at The Ohio State University, this symposium aims to "update knowledge and clinical acumen in contemporary areas of medical nutrition therapy, wellness and education." This year's program: *Growing the Future of Health: Nutrition Innovations across the Spectrum* is featuring some of our own O.S.P.E.N. members as speakers!

David Evans, MD, will be speaking on *"Enhanced Recovery after Surgery (ERAS) and Implications for the Dietitian,"* Kristen Roberts PhD, RDN, LD, CNSC will talk about *"Emerging Therapies for the Patient with Short Bowel Syndrome"* and Ainsley Malone, MS, RDN, LD, CNSC, FAND, FASPEN, will present a session on *"Critical Care Nutrition Guidelines."*

So, if you find yourself in the Columbus area on Friday, September 23, 2016 between 8:00 a.m. – 4:00 p.m., sign up and stop by The Ohio State University Medical Center's Ross Heart Hospital Auditorium.

For more information, visit: **http://medicine.osu.edu/hrs/md pages/marthanelsonlewis.aspx.**

News from the Scholarship Committee!

Do you want to attend Clinical Nutrition Week in Orlando, FL but don't have the funds to do so??

The O.S.P.E.N. Scholarship Committee is pleased to announce the availability of four $1,200 scholarships to attend Clinical Nutrition Week (CNW) in Orlando, FL, February 18-21, 2017. Two scholarships will be awarded to those who will be presenters at CNW 2017 and two will be awarded to those that have never attended CNW.

Eligibility for the scholarship is as follows:

- Member of O.S.P.E.N. at the time of application.
- Employed in a position with responsibilities in the area of nutrition support at the time of application.
- Members of the O.S.P.E.N. Board of Directors or Standing Committees are not eligible for the award.
- Members who won the O.S.P.E.N. scholarship the previous year are not eligible.

More information, including deadline to apply, coming soon!

Spring 2016 O.S.P.E.N. Conference was a Success!

Thank you to those that attended the Annual Spring O.S.P.E.N. Nutrition Symposium *"Breaking News…Nutrition Care of the Critically Ill and Wound Care Patients"* at Cleveland Clinic Main Campus in Cleveland, Ohio. Also, special thanks to the members of the O.S.P.E.N. Planning Committee – your dedication and hard work with the planning and hosting definitely paid off!

Ron Rock, RN, and Sue Meyer, RD: the nurse-dietitian team from the Cleveland Clinic giving their presentation on wound healing and the importance of nutrition.

Recent ASPEN President, Gordon Sacks, presenting the new SCCM/ ASPEN Guidelines for nutrition support management of critically ill patients.

Clinical Nutrition Week 2017 Registration Opens August 1st!

Are you thinking about attending CNW17 in beautiful Orlando, Florida? If not, you should reconsider! This annual event keeps you up-to-date on research and practice developments in the field of clinical nutrition and metabolism. Learn from other leaders in the field, contribute your expertise, and network with a large audience of nutrition support professionals with a variety of backgrounds (physicians, nurses, dietitians, pharmacists, researchers, students). Register here: **http://www.nutritioncare.org/cnw.**

Or, if you want to do more than just attend – consider submitting an abstract! Find out more information here: **http://www.nutritioncare.org/abstracts/.**

ORIGINAL, INTERNATIONAL, AND ENCORE ABSTRACTS	LATE-BREAKING ABSTRACTS
Opens – July 1, 2016	Opens – September 26, 2016
Closes– September 7, 2016	Closes – October 17, 2016
Submission Fee: $50	Submission Fee: $100

O.S.P.E.N. Member of the Year Award

This year's award was extra special, as O.S.P.E.N. recognized not one, but TWO incredible members!

Vince Vanek and Petrea Cober

O.S.P.E.N. President Kim Orben, RD, giving Dr. Vince Vanek his much deserved award at the Spring Symposium.

To highlight their contributions, we'd like to share an excerpt from each of their nominations:

Petrea Cober has been instrumental in every aspect of our organization. She is dependable, informed of all the rules and regulations we need to work with and so willing to investigate and submit any papers we may need. We truly can NOT function without her. She is so willing to be helpful, she truly goes beyond the expectations of a treasurer…

Vince Vanek, MD, continues to be active in ASPEN and OSPEN. His knowledge and experience within the organization is invaluable. And whenever he is asked for assistance — his response is always "yes." He is the leader of the program planning committee and ensures O.S.P.E.N. offers quality conferences. His continued leadership and dedication to O.S.P.E.N. deserves to be recognized and celebrated.

Congratulations and THANK YOU to Petrea and Dr. Vanek. It's people like you who make O.S.P.E.N. great!

Have You Considered Becoming More Active as an O.S.P.E.N. Member?

Members are always welcomed and encouraged to volunteer for committees and/or serve as an officer of the organization. For more information on getting involved, contact a member of the membership committee (located in the roster at the end of this newsletter).

Mark Your Calendars for the Upcoming Fall O.S.P.E.N. Symposium!

The Ohio Society for Parenteral and Enteral Nutrition Presents:

Malnutrition:
Bringing the Skeleton Back
Out of the Hospital Closet

Thursday, November 3
8:15 a.m. - 12:15 p.m.
Finnegan Auditorium
St. Elizabeth Youngstown Hospital

It's been over 40 years since the benchmark article *"The Skeleton in the Hospital Closet"* first called attention to the problem of malnutrition in the hospital setting…and we've come a long way since then!

Our distinguished panel of speakers will update us on this important topic, including:

- Criteria for identifying adults and children with malnutrition
- The impact of malnutrition on clinical and financial outcomes
- An "up close" look at the adult nutrition focused physical exam
- Future directions, including a call for malnutrition as a national goal

We hope you can join us for this timely and important program! Watch your e-mail or visit **www.mercy.com/youngstown** for more information.

The *O.S.P.E.N. Access* is produced by the
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Continuing Education through ASPEN's Webinar Programs

Did you know that ASPEN's webinars are not just for dietitians?

ASPEN designs their webinars for a multitude of different healthcare professionals who practice (or are interested in) the sciences of clinical nutrition and metabolism — including nurses, pharmacists, and physicians. For more information on registering and reviewing previous webinars visit **http://www.nutritioncare.org/webinars/.**

The upcoming webinar is as follows:

Microbiome: Research Update and Cutting Edge Applications: October 19, 2016 at 4:00 - 5:30pm ET

Did you miss one?

Don't worry about it! Each webinar is recorded and readily available on demand, so when you have time, it's there waiting for you. These sessions can be purchased for a nominal fee and CE credit is available for programs from the 2015 series and later. For all the details, browse the eLearning Center's website at: **http://www.prolibraries.com/aspen/?select=sessionlist&conferenceID=11.**

O.S.P.E.N. is on *Facebook*!

If you have not done so yet, please make sure to check us out at **www.facebook.com** and send us a friend request! Also, feel free to contact us directly via our O.S.P.E.N. email address at O.S.P.E.N.website@gmail.com if you do not have a *Facebook* account. We are looking forward to hearing from you!

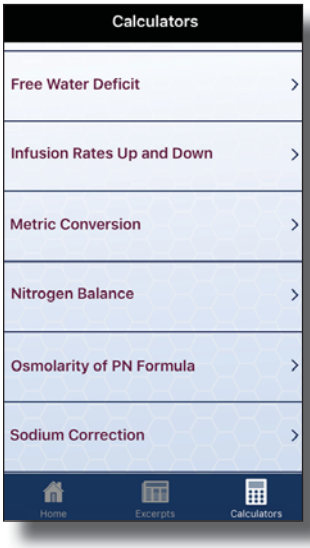
Have You Joined *ASPENconnect* Yet?

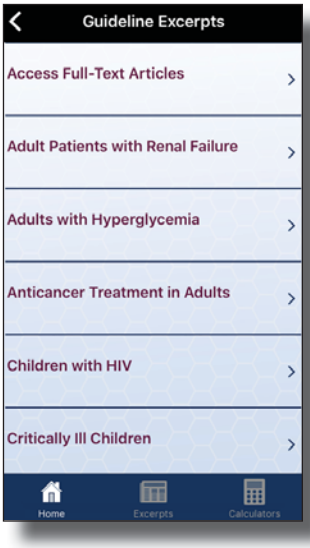
It's like LinkedIn®, but specifically for A.S.P.E.N. members, so it's a great place to connect with professionals in your field. In fact, you can pull your information from LinkedIn® right to your *ASPENconnect* profile! Connect with colleagues ad even create your own personal list of contacts. Additionally, on the *ASPENet* forum pages, you can post comments, view discussions, and get advice from your colleagues. Just use the A.S.P.E.N. website login. Learn more at: **www.community.nutritioncare.org/home.**

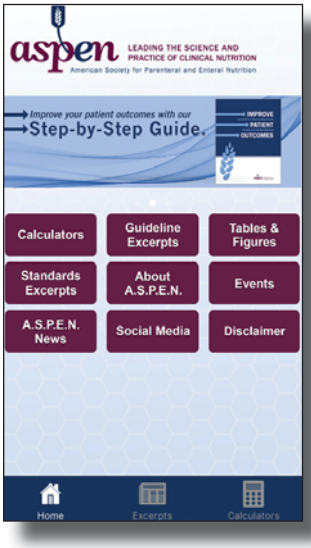
Download the ASPEN Clinical App!

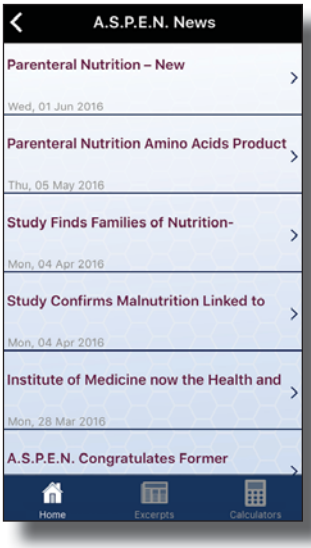
If you haven't already done so, check out the ASPEN Clinical App. The app puts practical information right at your fingertips. Need to calculate free water deficit? Go to the app! Need to reference a guideline? Go to the app! Want to know what's new with ASPEN? Go to the app! Want a quick reference table for treating hyperkalemia? Go to the app!

This app works on Androids and iPhones and is free for any ASPEN member. Just visit the *iTunes* or *Google Play* Store and search "ASPEN Clinical App." Even if you're not a member, you can email info@nutritioncare.org for a free 3-day trial.









Malnutrition Awareness Week™

The 5th annual Malnutrition Awareness Week is coming fast! Scheduled for September 26 - 30, 2016, this event intends to raise awareness of malnutrition while encouraging all healthcare professionals to look for and take action before it becomes a much worse problem for our patients. The week is specifically focused on providing educational programs and disseminating resources to help all clinicians, as well as the general public, about this often overlooked condition.

We encourage you and your colleagues to visit the official **Malnutrition Awareness Week** website! Register for more information and find out how you and your institution can participate. Join your colleagues or make connections with other clinicians who share your passion to continue the fight against this recurring condition.

In conjunction with Malnutrition Awareness Week, ASPEN has announced the following lineup of webinars, complete with continuing education credits, and is free to all ASPEN members. The webinars will also include a chat with the experts on the following topics:

- **September 26:** *Improving Malnutrition From the Physician Perspective*
- **September 27:** *Combating Malnutrition in Spanish Speaking Populations: Available Programs and Resources*
- **September 28:** *Aging Does Not Matter: Malnutrition in the Aging Population*
- **September 29:** *Malnutrition Interventions and Programs for Older Adults*

Webinars are scheduled daily at 1:00 - 2:30 p.m. EST.

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