Chicken & Spinach Soup with Fresh Pesto

**Ingredients:**
- 2 tsp plus 1 Tbsp cooking oil
- 1/2 cup carrot or diced red bell pepper
- 1 large boneless, chicken breast (8 oz) cut into quarters
- 1 large clove garlic, minced
- 5 cups reduced-sodium chicken broth
- 1/12 tsp dried marjoram
- 6 oz baby spinach, coarsely chopped
- 1 15 oz can cannellini beans or great northern beans, rinsed
- 1/4 cup grated Parmesan cheese
- 1/3 cup lightly packed fresh basil leaves
- Freshly ground pepper to taste
- 3/4 cup croutons (optional)

**Directions:**
1. Heat 2 tsp cooking oil in a large saucepan over medium heat. Add carrot (or bell pepper) and chicken; cook, turning the chicken and stirring frequently, until the chicken begins to brown 3 to 4 minutes. Add garlic and cook, stirring, for 1 minute more. Stir in broth and marjoram; bring to a boil over high heat. Reduce the heat and simmer, stirring occasionally, until the chicken is cooked through, about 5 minutes.

2. With a slotted spoon, transfer the chicken pieces to a clean cutting board and cool. Add spinach and beans to the pot and bring to a gentle boil. Cook for 5 minutes to blend the flavors.

3. Combine the remaining 1 Tbsp oil, Parmesan and basil in a food processor. Process until a coarse paste forms, adding a little water.

4. Cut the chicken into small pieces. Stir the chicken and pesto into the pot. Season with pepper. Heat and serve.

This article is the first of a three-part series on how to work with the athletic patient. I was inspired by personal experience as well as many anecdotes of physicians who were poorly equipped to manage patients who are moderately to extremely active. Much of what is taught lays in between the pathologic state and the athletic state. In fact, with some extreme cases, the athletic state can develop into pathology. This series is meant to help differentiate what findings should be expected, which should be followed up with, what requires further diagnostics, and how to build a positive relationship with this population.

**What to expect to see in the athletic patient:**

Many texts and resources go into great detail about the changes that occur acutely when exercising. We learn how venous return increases in the heart causing preload to increase and maximize cardiac output. We understand how arteriole shunting allows muscles to obtain more oxygen and that the metabolites from exercise decrease hemoglobin’s affinity for oxygen to increase unloading at the muscles. But what about chronic activity? How does the body adapt a chronic state of exercise?

Let’s take our patient Jane Doe. She is a 32-year-old triathlete who lifts weights in the off season. She competes in national triathlons and has placed many times. She has no significant past medical history. Jane has a Primary Care Physician who is not aware of the physiologic changes that can occur with Jane and sends her to many unwarranted specialist consultations. Don’t worry, Jane has great insurance.

**Cardiovascular:**

Jane comes to the office for routine blood work and a physical. She states she has had bad acid reflux recently and the PCP gets an EKG as a part of his workup. The PCP notices a massive QRS peak, left axis deviation, and a 2nd degree AV block rhythm with marked bradycardia at 28bpm. Upon auscultation, he hears abnormal sounds. Panicked, the PCP sends Jane to the ER for myocardial infarction. In the ER, cardiac enzymes show elevated CK-MB and elevated lactate dehydrogenase. As Jane is about to be sent for further workup, an astute ER physician asks about her training revealing she had completed a 25-mile run the night prior.

Elite athletes, especially in high O2 demand sports like running, demonstrate several cardiovascular changes not seen in other individuals. First, the left ventricle hypertrophies while maintaining or increasing EDV. This is an important point that differentiates athletic heart from hypertrophic cardiomyopathy. The heart muscle gets thicker but the amount of blood that can enter prior to contraction stays constant or may be greater. This can cause an EKG to show a left ventricular hypertrophy pattern and even axis deviation from the larger size. Because of the larger size and greater strength of her heart, there will likely be two extra heart sounds; S3 and potentially S4. S3 occurs from an enlarged ventricle from dilated cardiomyopathy or an athletic heart. S4 can occur from excessively strong atrial contraction into the ventricle from a restrictive cardiomyopathy, or a strong athletic left atrium.

During a heart attack, the dying heart muscle releases certain enzymes that can be picked up in the blood. If you think about normal muscle growth, fibers are damaged and rebuilt. Studies show that skeletal muscle can release creatine kinase that is not able to be differentiated from cardiac CK-MB and is the source of her alarming enzymes. Some sources also report increase in cardiac Troponin and BNP after strenuous activity, but this is less understood. Low heart rates are also seen in the elite athlete, often reaching below 30 bpm.
Finally, a meta-analysis by Chillag showed that abnormal cardiac rhythms that occur with greater than 10% frequency in athletes include: sinus pause, Mobitz type 2 AV block, pre-ventricular contractions, pre-atrial contractions, wandering atrial pacemakers, and junctional escape rhythms. When examined, most of these arrhythmias were transient and disappeared during stress tests. Also, some discuss sudden cardiac deaths with athletics. Technically the most sudden deaths while running are from automobile accidents. The cardiac sudden deaths are mostly from structural or congenital abnormalities that are picked up early in life (long QT, WPW, congenital hypertrophic cardiomyopathy).

Renal:

Jane’s PCP receives her blood work and notes an elevated BUN and Creatinine with a significantly elevated BUN/Cr ratio. A urinalysis was also performed and showed proteinuria, hematuria, hyaline casts, and oliguria for the past day. Again, the PCP is alarmed and refers Jane to a nephrologist for further workup of Nephritic syndrome.

In 1971, Kenneth Gardner discussed the phenomenon called athletic pseudo-nephritis. Exercise causes an increase in overall blood pressure as well as a decrease in kidney blood flow. Blood is shunted to the muscles and away from organs such as the kidney and bowel. There has been an observed increase in the constriction of glomerular efferent arteriole as compared to the glomerular afferent arteriole. The result is an increased glomerular filtration pressure causing leak of proteins, RBC, and WBC into the nephron. Also, elite athletes can easily dehydrate create hyaline casts in the urine. These change can last from hours to days after strenuous exercise and can mimic Acute Renal Failure.

Hematologic:

Jane the reassures her PCP that she is not in renal failure and the physician wants to repeat the blood work due to “suspicious” previous results. On the first CBC, Jane had marked neutrophilia and anemia with normal-high hemoglobin. On blood smear, hematopoietic progenitor cells were identified. On the repeated CBC, Jane now had significant neutropenia and overall leukopenia. Hematocrit was still low and hemoglobin was elevated. She is sent to Hematology for consultation.

The immunology of sport has been extensively studied and there are still many questions yet to be answered. However, some patterns of change have been observed. Directly after extreme endurance training events (marathons, triathlons, etc.) there is an observed transient inflammatory response. This includes release of band cells and even more immature hematopoietic cells. Also, increase in skeletal muscle infiltration with CD8+ (cytotoxic T) cells as well as overall inflammatory markers were noted to be elevated 28hrs post exercise. Interestingly, at rest (days after intense exercise), elite athletes exhibit neutropenia, theorized to be from a decrease in systemic inflammation at rest. Gene expression of inflammatory cells were also investigated. In an article by Buttner et al., a team investigated gene expression showing an increase in expression of metalloproteases and heat shock proteins involved in mobilization of hematopoietic stem cells for greater white blood cell growth and development. In addition, oncogene expression known to play roles in cancer pathology was decreased 3-fold. The observed anemia is from a phenomenon known as “sport anemia.” It is due to an increase in RBC size with an even greater increase in plasma volume. This dilutes the blood causing an observed anemia.
Conclusion:

There are many other physiologic adaptations that occur due to intense, chronic exercise such as: hematochezia, melena, amenorrhea, constipation, diarrhea, myoglobinuria, etc. When evaluating a patient, inquiring about their physical activity is as important as any other aspect of the interview. Many people may not “appear” to be an elite athlete and can throw you off. There are amazingly profound changes that can occur throughout all organ systems as adaptation to the extreme stress these individuals endure. And many of these changes can appear quite dangerous and can be unexpected. It can be hard to keep all of this in mind when you see 100 patients who have the disease their symptoms dictate. Keep in mind how the patient feels physically, what their activity level is, and like Jane’s PCP, reach out for assistance if there is confusion or a potential risk to the patient.

References:


Article By: Adam Heilmann, M2
May Wellness Events Calendar

Do you know we have a Calendar of Events? Do you know it lists a variety of things to do on days, nights and/or weekends when you’re able to take a break from studying?! We do!

And...

May is now posted!

Here’s the link:

WELLNESS CALENDAR OF EVENTS!

The Student Gym is Shaping Up!

Get the most out of your workout in the Student Gym! We now have free weights and benches. Please continue to keep your suggestions coming and remember, don’t drop the weights!