

JAAPA letters to the editor, August 2009

Artist's rendition of hemangioma was not realistic

To the Editor:

I am writing to express my disappointment with an article in the May 2009 issue of *JAAPA*. I am referring to the CME article on infantile hemangiomas. As a parent of a child with three hemangiomas, I have to tell you that the artist's rendition on the cover does not even come close to depicting what a hemangioma looks like. Instead, the picture looks like a red Bandaid or an abrasion.

Based on what I know about hemangiomas, this does not represent what the typical lesion looks like. Then, I could not believe that this CME article did not contain a single photograph of a hemangioma. The only two pictures provided were drawings. I would think that a CME article on a dermatologic condition should contain at least a picture or two of what is being described.

I discussed this article with a number of my PA colleagues who know my daughter. They agree that this was a poor attempt at helping others diagnose this condition.

Ginger Spitzer, PA-C

Why was antiphospholipid antibody syndrome not included?

To the Editor:

I would like to point out a potentially dangerous error in the article, "Genetic susceptibility to VTE: A primary care approach" (published in the July 2009 issue), by Herbert D. Ridings, MA, PA-C; Lynn Holt; Rebecca Cook, MD; and Marisa B. Marques, MD. In this article, there is a blatant misuse of statistics to calculate the 20 most common causes of VTE. Table 1 lists seven disorders that are, in the authors' opinions, the most common causes of VTE; the conditions can affect 0.02% and 0.3% of the population, and 2.0% of whites. There is no mention in this list of a disease that effects between 2% and 5%—depending on which study you read—of the population: antiphospholipid antibody syndrome (APS or APLS, lupus anticoagulant syndrome, Hughes syndrome, and a number of other misnomers). This disease causes venous thrombosis, arterial thrombosis and is a truly systemic disease that, in the opinion of some of the world's top rheumatologists, is the predominant cause of clotting problems in accident and emergency admissions.

I find it completely unprofessional for a journal to include a poorly documented opinion as a potential CME educational tool. I would like to hear your comments on the subject matter and if *JAAPA* will be publishing a correction notice so that those who have been misinformed can be made aware that another disease should be included among the top five of any list of causes of VTE, and for which a series of simple studies should be undertaken in many, if not all, cases of VTE.

Sir Edward Bulfin, PhD, KCHS

Author's response: The objective of the article “Genetic susceptibility to venous thromboembolism: A primary care approach” was to provide primary care clinicians with information about the common genetic causes of venous thromboembolism. This was considered important because genetic tests are now available to identify these disorders and because the presence of these genetic abnormalities significantly increases a person's risk of thromboembolism, particularly when the disorders are coinherited or present in the setting of one or more of the acquired risk factors, such as pregnancy or prolonged bed rest.

Antiphospholipid antibody syndrome (APS) is not a genetic disorder. It is an acquired risk factor with no genetic abnormality that can be identified through genetic testing. Antibodies are present with APS, but they are some of the same antibodies associated with diseases like systemic lupus erythematosus. APS has a wide range of clinical manifestations, and diagnosis depends on excluding the other autoimmune disorders associated with these APS antibodies.

A full discussion of the acquired risk factors for venous thromboembolism was beyond the scope of this article, and that point was made in the opening paragraphs of the article. If the audience is interested in a follow-up article that describes the 20 or 25 acquired risk factors that can cause venous thromboembolism, the authors are willing to provide this in the future.

As a final point, the autoimmune nature of APS and diseases like SLE suggest that a genetic component does exist with these diseases, and there is reason to hope that the Human Genome Project will identify the nature of these genetic abnormalities in the future.

Herb Ridings, MA, PA-C
Lynn Holt, MS
Marissa Marques, MD