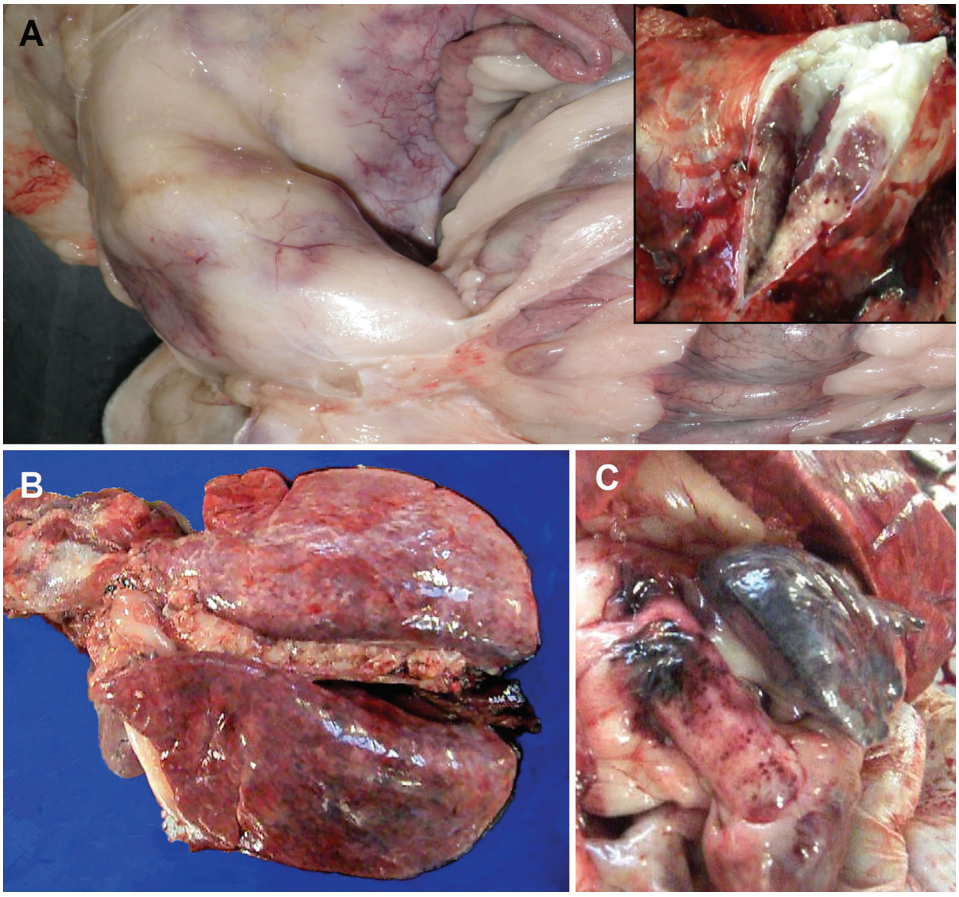


**Figure 1**—Photographs of the abdominal cavity (A), lungs (B), and heart (C) of an obese 10-year-old Vietnamese potbellied pig (*Sus scrofa*) that spontaneously died while ranging free with a herd mate. The potbellied pig had been fed a diet based on commercial dog food for several months. A—View of the parietal peritoneum. The abdominal fat is diffusely thickened and firm and has yellow-brown discoloration and petechiae. Inset—Detail view of cut abdominal fat illustrating the infiltrative lesions. B—View of the lungs. The lungs were diffusely pale gray to mottled red, with a rubbery consistency. They failed to collapse when the thoracic cavity was opened. C—View of the heart. Notice the multifocal hemorrhages at the aortic arch.

## History

A 10-year-old neutered female Vietnamese potbellied pig (*Sus scrofa*) was submitted to the Department of Veterinary Pathology, Clinical Veterinary Hospital, Alfonso X el Sabio, University of Madrid, for postmortem examination. The animal belonged to an animal welfare organization and was ranging free with another herd mate when it spontaneously died. Previously, the potbellied pig had spent several months in a kennel and been fed a commercial dog food–based diet.

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## Clinical and Gross Findings

At necropsy, the potbellied pig had a body condition score of 5/5,[1] and was considered obese; it weighed approximately 117 kg (257.4 lb). A gross examination revealed diffusely thickened and firm abdominal fat of the parietal peritoneum, with a yellow-brown coloring and petechiae **(Figure 1)**. When the thoracic cavity was opened, the lungs had a diffusely pale gray color with mottled red areas. The lungs failed to collapse and had a rubbery consistency. Other findings included the presence of pericardial petechiae and multifocal hemorrhages on the parietal surface of the aortic arch.

**Formulate differential diagnoses from the history, clinical findings, and Figure 1—then turn the page→**

## Histopathologic Findings

Samples of lungs, heart, parietal peritoneum, kidneys, liver, stomach, pancreas, small and large intestines, and spleen were fixed in neutral-buffered 10% formalin, embedded in paraffin, sectioned at 4-µm intervals, and stained with H&E stain for histologic examination. Microscopic evaluation determined the presence of moderate congestion and fibrosis of the mesentery and associated adipose tissue as well as a severe multifocal granulomatous steatitis. Cholesterol deposits and multiple foci of degeneration and necrosis were also visible. These areas appeared to be surrounded by numerous epithelioid macrophages, giant multinucleated cells, and a few scattered lymphocytes, plasma cells, and neutrophils **(Figure 2)**. Randomly distributed multifocal lesions were observed in all pulmonary lobes; microscopically, they were identified as expanded alveolar septa. The expansion was caused by the presence of multiple, intravascular, round, well-delimited clear globules (likely lipid droplets), which varied in size (20 to 120 µm). Similar vacuoles were observed within the glomerular capillaries of both kidneys.

To detect the presence of fat in tissues, samples are frequently frozen and tested with Sudan stain. In the case described in the present report, tissue sections from the lungs and mesentery were prepared with osmium tetroxide. This method has been previously reported to detect fat emboli in formalin-fixed tissues.[2] Samples were sectioned (2 to 3 µm) to allow adequate penetration of the reagent, then washed in distilled water for 30 minutes. After immersion with continuous agitation in an aqueous solution of osmium tetroxide for 2 hours, samples were washed under running tap water for 30 minutes. Subsequently, tissue sections were immersed in 0.5% periodic acid until the dark osmium-infiltrated tissues were uniformly cleared (30 minutes); sections were rinsed again with tap water. Finally, sections were placed

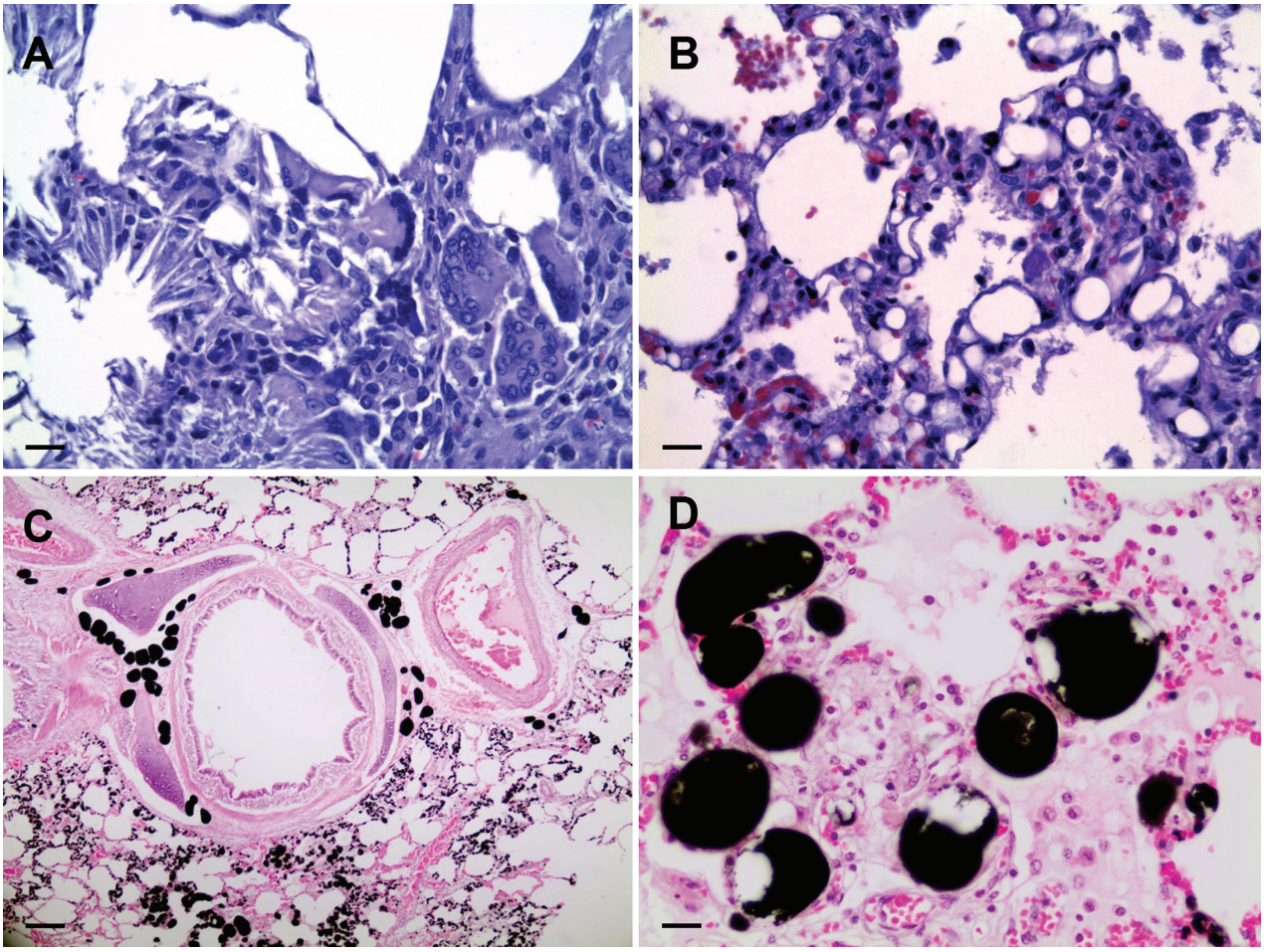


**Figure 2**—Photomicrographs to illustrate the microscopic lesions identified in the Vietnamese potbellied pig in Figure 1. A—Section of the parietal peritoneum. Severe, granulomatous steatitis is evident. Numerous macrophages, epithelioid macrophages, and giant multinucleated cells are present around cholesterol deposits with fewer scattered lymphocytes, plasma cells, and neutrophils. H&E stain; bar = 100 µm. B—Section of lung tissue. Randomly distributed, multiple, intravascular, well-delimited clear globules that varied in size (20 to 120 µm) are visible. H&E stain; bar = 100 µm. C—Another section of lung tissue. Multiple, well-defined black globules have displaced blood cells within arterioles, capillaries, and venules. Osmium tetroxide staining method; bar = 400 µm. D—Higher-magnification view of the section in panel C. Alveolar septa are expanded by black globules of different sizes. These globules correspond to fat emboli that collapse the alveolar capillaries. Osmium tetroxide staining method; bar = 100 µm.

in distilled water for 5 minutes, routinely processed, and stained with hematoxylin. Positive reactions for fat appeared as multiple, well-defined black globules that displaced blood cells within arterioles, capillaries, and venules. The liver and pancreas and the adipose tissue around them did not have any lesions.

## Morphologic Diagnosis and Case Summary

Morphologic diagnosis: severe, acute, diffuse pulmonary fat embolism and severe, chronic, multifocal granulomatous steatitis and steatosis of the mesenteric adipose tissue.

Case summary: pulmonary fat embolism secondary to steatitis (yellow fat disease) and steatosis of the mesenteric adipose tissue in a potbellied pig.

## Comments

Fat embolism is the blockage of blood vessels by fat globules, which mainly occurs in lung capillaries. Adipose tissue from bone marrow or lipid-containing tissues (eg, mesentery or subcutaneous tissue) can be released into the circulation as a result of major fractures, hyperlipemia, development of fatty liver, or adipose tissue damage owing to trauma, pancreatic necrosis, or yellow fat disease. Fat embolism is associated with clinical signs of organ dysfunction (fat embolism syndrome). The main clinical signs in affected cats, dogs, and horses are petechiae and respiratory distress.[2–9]

Yellow fat disease is a disorder of fat depots characterized by steatosis, steatitis, and necrosis of adipose tissue. It has been described in many species, specifically cats, pigs, horses, and minks. It is commonly related to ingestion of feed rations with low concentrations of antioxidants (selenium or vitamin E) or rations containing a high percentage of polyunsaturated fatty acids.[9–13] Similar to the case described in the present report, fat embolism attributable to yellow fat disease caused by low dietary levels of antioxidants in an Appaloosa horse has been reported. When feeding rations low in vitamin E, progressive peroxidation of unsaturated fatty acids can occur, causing liver necrosis and myocardial and skeletal muscle degeneration.[2] Peroxidation of unsaturated fatty acids can also take place in animals fed diets with adequate concentrations of antioxidants but excess fat content.[9–13] In the potbellied pig of the present report, lesions associated with antioxidant deficiency were not found. In addition, commercial balanced adult dog diets typically have approximately 50 U of vitamin E/kg (22.7 U/lb) and 10% to 15% total fat.[14] These values are higher than the general recommendations for vitamin E and fat contents of diets for potbellied pigs. The most appropriate rations to use for Vietnamese potbellied pigs are maintenance rations specifically formulated for pigs containing approximately 2% to 3% of fat and a daily administration of 40 U of vitamin E/kg (18.2 U/lb).[15] The obese condition of the pig of the present report and the administration of a diet specifically formulated for dogs probably contributed to consumption of a higher than recommended proportion of polyunsaturated fatty acids rather than insufficient vitamin E intake.

To our knowledge, there is just 1 other report[16] of lipid emboli in a Vietnamese potbellied pig. In that pig, intravascular fat droplets were detected by means of an indirect method based on anti-fibrinogen immunohistochemical analysis, but the cause of the fat emboli remained unclear. However, 3 possible explanations were given for the release of adipose tissue into the blood vessels of that pig: the presence of abdominal fat necrosis, hepatic lipidosis, and the surgical procedures performed (exploratory celiotomy, CT-guided collection of a bone marrow aspirate sample, and percutaneous hepatic biopsy).[16] In the potbellied pig of the present report, signs of trauma were not identified during necropsy; it had not undergone any surgical procedures during the preceding months. Hepatic lipidosis and pancreatic necrosis were ruled out on the basis of the absence of pathological lesions in the liver and pancreas and the adipose tissue surrounding both of them.

## Acknowledgments

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## References

1. The pig site. Body condition scoring. Available at: www.thepigsite.com/stockstds/23/body-condition-scoring. Accessed Dec 2, 2014.
2. Suárez-Bonnet A, Espinosa de los Monteros A, Herráez P, et al. Fat embolism secondary to yellow fat disease in an Appaloosa horse. *J Vet Diagn Invest* 2008;20:684–687.
3. Batra P. The fat embolism syndrome. *J Thorac Imaging* 1987;2:12–17.
4. Akhtar S. Fat embolism. *Anesthesiol Clin* 2009;27:533–550.
5. Peltier LF. Fat embolism: a perspective. *Clin Orthop Relat Res* 1988;232:263–270.
6. Sevitt S. The significance and pathology of fat embolism. *Ann Clin Res* 1977;9:173–180.
7. ten Duis HJ. The fat embolism syndrome. *Injury* 1997;28:77–85.
8. Sierra E, Rodríguez F, Herraez P, et al. Post-traumatic fat embolism causing haemothorax in a cat. *Vet Rec* 2007;161:170–172.
9. Caswell JL, Williams KJ. Respiratory system. In: Maxie MG, ed. *Jubb, Kennedy, and Palmer's pathology of domestic animals*. 5th ed. New York: Saunders Elsevier; 2007;523–653.
10. Bruijn CM, Cróese Veldhuis EJB, Van Oldruitenborgh-Oosterbaan Sloet MM. Yellow fat disease in equids. *Equine Vet Educ* 2006;18:38–44.
11. Glyn M. Steatitis in foals. *Vet Rec* 1972;90:82.
12. Mason KE, Hartsough GR. Steatitis or yellow fat disease in mink and its relation to dietary fats and inadequacy of vitamin E. *J Am Vet Med Assoc* 1951;119:72.
13. Van de Kerk P, Danse LHJC. Yellow fat disease in piglets and fattening pigs. *Tijdschr Diergeneeskd* 1973;98:1118–1132.
14. Association of American Feed Control Officials. AAFCO methods for substantiating nutritional adequacy of dog and cat foods. Available at: www.aafco.org/Portals/0/SiteContent/Regulatory/Committees/Pet-Food/Reports/Pet_Food_Report_2013_Midyear-Proposed_Revisions_to_AAFCO_Nutrient_Profiles.pdf. Accessed Nov 9, 2015.
15. Tynes VV. Potbellied husbandry and nutrition. *Vet Clin North Am Exot Anim Pract* 1999;2:193–208.
16. Newkirk KM, Fineschi V, Kiefer VR, et al. Lipid emboli in a Vietnamese potbellied pig (*Sus scrofa*). *J Vet Diagn Invest* 2012;24:625–629.