peak flowmeters tested is absolutely acceptable for use clinically as long as correct technique is used by the patient and each instrument may be susceptible to significant elevation with use of the incorrect technique. The reason that PEF is inappropriately high as a result of the "spitting" maneuver is apparently as Connolly described it; he believes that the incorrect maneuver accelerates the airflow through the mouth, with the subject using the tongue and buccal musculature.

Other investigators have shown that patients will lie to clinicians about their PEF values. Some patients want to please their clinicians by reporting values obtained either by manipulating the peak flowmeter via incorrect technique (one of our patients admitted to doing this prior to the study) or by recording untrue values. Thus, regardless of which peak flowmeter is used, clinicians need to instruct patients carefully regarding correct use, proper recording of PEF, and avoidance of acceleration in the mouth (ie, the "spitting" maneuver). Emphasizing maximum expiratory effort with the mouthpiece well into the mouth, overlaying the anterior part of the tongue, prevents acceleration of airflow in the mouth.

REFERENCES

ARDS and Adrenal Insufficiency Associated With the Antiphospholipid Antibody Syndrome*

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The antiphospholipid antibody syndrome (APS) is typically characterized by recurrent arterial and/or venous thromboses, miscarriages, and thrombocytopenia. There have been five reported cases of ARDS associated with primary APS. Adre-

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A syndrome of recurrent thrombosis associated with a biological false-positive test for syphilis was reported by Johansson and colleagues in 1976; but only over the past decade has the association of recurrent vascular thromboses, fetal loss, and thrombocytopenia, combined with persistently elevated antiphospholipid antibodies, been designated as the antiphospholipid antibody syndrome (APS). APS is considered to be primary in the absence of connective tissue diseases and secondary in their presence; the most common connective tissue disease is systemic lupus erythematosus. The rising awareness of APS, along with its potential for multiple organ system involvement mediated by vascular thromboembolic events, has made its recognition increasingly important. Pulmonary manifestations associated with APS are well documented, but only five cases have been reported of patients with both APS and ARDS. Adrenal insufficiency (AI) has also been reported as a rare complication of APS. We report a case of both ARDS and AI associated with primary APS.

Case Report
A 33-year-old white woman with known lupus anticoagulant and Hashimoto's hypothyroidism presented to the hospital's emergency department with a 5-day history of nausea, vomiting, diffuse abdominal pain, and generalized joint pain. Her symptoms started while she was vacationing in Europe. Her medication consisted of levohyroxine and an aspirin a day. Her past medical history included testing positive for antithyroid microsomal and antithyroglobulin antibodies. Physical examination revealed the presence of tachycardia (131 beats/min), a macular violaceous discoloration of her distal phalanges, and diffuse abdominal tenderness. A leukocyte count of 11,400/µL with 80% neutrophils, 7% lymphocytes, and 9% monocytes, a hemoglobin of 10.9 g/dL, a hematocrit of 32.3%, and a platelet count of 69,000/µL were noted. The activated partial thromboplastin time (APTT) was 92.8 s with a prothrombin time (PT) of 13.3 s. Chest radiographs and abdominal series on admission were negative. Lupus anticoagulant and antiphospholipid antibodies in the form of antiphosphatidyl serine IgG antibodies were detected. Along with the thrombocytopenia, the criteria for APS were established.

Key words: adrenal insufficiency; antiphospholipid antibodies; antiphospholipid antibody syndrome; ARDS

Abbreviations: ACTH=adrenocorticotropic hormone; AI=adrenal insufficiency; APS=antiphospholipid antibody syndrome; APTT=activated partial thromboplastin time; PT=prothrombin time

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The erythrocyte sedimentation rate was 109 mm/h. Rapid plasma reagin was reactive, fluorescent treponemal antibody absorption was negative, and antinuclear antibodies were negative. HIV enzyme-linked immunosorbsent assay was nonreactive. Electrolytes were normal except for a sodium level of 123 mEq/L. Liver function tests were also normal except for serum glutamate oxalate transaminase (174 mg/dL) and lactate dehydrogenase (417 mg/dL). Approximately 36 hours after admission, the patient developed dyspnea, bradycardia (35 beats/min), and hypotension (73/62 mm Hg) that led to cardiopulmonary arrest requiring mechanical ventilatory support. A pulmonary angiogram revealed extensive pulmonary infiltrates without evidence of pulmonary embolization. A chest radiograph disclosed bilateral interstitial and alveolar infiltrates involving the entire lungs and consistent with ARDS (Fig 1). A Swan-Ganz catheter was placed, revealing a cardiac index of 2.9 L/min/m², pulmonary artery pressure of 44 mm Hg systolic and 30 mm Hg diastolic, and a pulmonary capillary wedge pressure of 16 mm Hg.

In the intensive care unit, the patient required intravenous dopamine at 3 μg/kg/min for 7 days, dobutamine at 7.5 μg/kg/min for 6 days, and norepinephrine at variable doses ranging from 4 μg/min to 16 μg/min for 2 days. Because of the difficulty in maintaining her cardiovascular status with cardiotoxic and vasopressor agents, a test was performed to measure the random serum cortisol level, yielding a value of 1.1 μg/dL. Subsequently, a CT scan of the abdomen was performed and revealed bilateral adrenal gland enlargement with areas of increased density in both adrenal glands consistent with bilateral adrenal hemorrhagic infarction (Fig 2). A rapid adrenocorticotropic stimulation test supported the existence of primary adrenal insufficiency with the serum cortisol levels being 1.1 μg/dL, 1.0 μg/dL, and 1.1 μg/dL at ½ hour, 1 hour, and 2 hours, respectively, after the administration of 0.25 mg of cosyntropin (adrenocorticotropic hormone, or ACTH). The patient was mechanically ventilated for 5 days, during which time she responded to a course of high-dose pulsed corticosteroids. Chest radiographs revealed gradual clearing over a 6-day period. Additional serologic tests revealed the absence of antibodies to double-stranded DNA, Smith antigen, Sjögren’s syndrome antigen A and B, and ribonuclease protein. She was discharged on steroid replacement therapy after a clear chest radiograph. The patient was readmitted 5 months later complaining of nausea, vomiting, and generalized weakness/fatigue. She sustained cardiac arrest with intractable heart failure and renal failure, which culminated in her death.

**DISCUSSION**

Individuals with APS are at an increased risk of widespread arterial and/or venous thromboses due to the presence of circulating antiphospholipid antibodies. The precise mechanism of action of antiphospholipid antibodies is unknown, but the present theory is that they interfere with the coagulation-anticoagulation pathways and endothelial cell function through the binding of antigens composed of negatively charged protein/phospholipid complexes, leading to a loss of the normal hemostatic balance.3,5

The most common pulmonary manifestations of APS are pulmonary thromboemboli and pulmonary hypertension secondary to multiple thromboemboli.3,5 There have been five reported cases of ARDS associated with APS.4,5 Of these cases, two of the four survivors responded dramatically to intravenous methylprednisolone; the other two patients responded to plasma exchange transfusion, cyclophosphamide, and anticoagulation, but did not receive steroids. The patient who did not survive was not treated with any of these modalities. The autopsy revealed extensive small vessel

**FIGURE 1.** Chest radiograph revealing bilateral interstitial and alveolar infiltrates involving the entire lungs. This radiograph was taken on the second hospital day. The right-neck density is a misplaced central venous catheter that was later repositioned. The Swan-Ganz catheter was inserted via the right femoral vein with wave forms indicating good positioning. The endotracheal tube at the level of the mid trachea was repositioned later at the level of the carina. The perceived widened mediastinum on this portable anteroposterior film with the patient somewhat rotated to the right was not present on repeat chest radiograph.

**FIGURE 2.** CT scan of the abdomen at the level of the adrenal glands demonstrating bilateral adrenal gland enlargement (thick arrows) with areas of increased density (thin arrows) in both adrenal glands consistent with bilateral adrenal hemorrhagic infarction.
thromboses, including extensive interstitial and intra-alveolar hemorrhage of the lungs without evidence of vasculitis, consistent with ARDS. Our patient responded to a course of high-dose pulsed corticosteroids. The mechanism of ARDS in APS is unclear, but the postulated explanation is that an acute increase in hydrostatic pressure by an occluding embolus may cause exudation of fluid from blood vessels into the lung parenchyma and result in ARDS, or pulmonary microemboli can cause vascular injury in the pulmonary circulation, leading to increased transport of protein through the pulmonary microvascular barrier into the parenchyma.

AI has also been reported as a rare but potentially fatal complication of APS. Of the 28 reported cases of AI associated with APS, 23 had depressed cortisol levels, 22 had positive ACTH stimulation tests, and 17 had CT or MRI findings revealing bilateral adrenal hemorrhage and adrenal enlargement or atrophy. The remainder of the cases were not documented with CT or with biochemical adrenal testing. One patient did not survive. The most common symptoms of AI are abdominal pain, nausea, vomiting, fatigue, hypotension, and fever; our patient manifested all of these except fever. The exact cause of adrenal hemorrhage in APS is unknown. Venous drainage of the adrenal gland, which occurs via a small number of venules that empty into a central vein, makes this gland particularly prone to hemorrhage after venous thrombosis. Hemorrhagic infarction of the adrenal glands secondary to adrenal vein thrombosis appears to be the mechanism of AI in APS. The CT findings in our patient are consistent with bilateral adrenal hemorrhagic infarction.

Although there is a remote possibility that ARDS was a result of the AI, the review of the literature did not reveal any reported cases of ARDS associated with AI. In addition, the findings of a normal pulmonary capillary wedge pressure and cardiac index further support the unlikelihood of the ARDS being a result of the AI. We believe that the lungs and adrenal glands were the focus of small vessel thrombosis in our patient, leading to ARDS and AI. This case emphasizes the importance of recognizing APS, with its potential for multiple organ system involvement. This is the sixth reported case of ARDS associated with APS and the only reported case, to our knowledge, of both ARDS and AI associated with APS.

**REFERENCES**


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**Circumflex Coronary Artery Dissection Following Waterskiing**

**Jeffrey Greenberg, MD; Michael Salinger, MD; Frank Weschler, MD; Brian Edelman, MD; and Randall Williams, MD**

A previously healthy 35-year-old woman experienced chest discomfort following mild blunt chest trauma while waterskiing. During the following 48 h, she underwent evaluation in two separate emergency departments (EDs) where she was found to have normal ECGs and cardiac enzyme values. She was subsequently discharged from both EDs. Twelve hours after the second ED visit, she was discovered unconscious at home and was resuscitated from ventricular fibrillation. Emergency cardiac catheterization demonstrated moderate circumflex disease without angiographic evidence of a false lumen or intimal flap. Left ventriculography demonstrated posterolateral wall hypokinesis. Eight days after remaining ventilator-dependent and unconscious, the patient was declared brain-dead. At autopsy, the patient was found to have a dissection of the circumflex artery. Waterskiing is an unusual source of cardiac trauma; however, we believe this to be the first reported case of dissection in the circumflex artery following blunt chest trauma.

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**Key words:** coronary disease; nonpenetrating wounds; coronary aneurysm; sudden cardiac death; heart injuries

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*A previously healthy young woman suffered a coronary artery dissection following blunt chest trauma while waterskiing. Coronary artery dissection is a relatively rare phenomenon, and it is even rarer for it to be caused by blunt chest trauma. Seven previous reports of cases due to blunt trauma were found in the English-language medical literature. Five of the cases reported a tear in the left anterior descending coronary artery, one in the right coronary artery, and one in the left main coronary artery. The patient in our case was found to have a dissection of*