To the Editor: Myxoma is well known as a cause of cardiac embolization (1, 2). However, the reason that embolic events frequently occur in patients with cardiac myxoma remains unknown. We report on myxoma cells that were strongly stained by the terminal deoxyribonucleotidyl transferase–mediated dUTP (deoxy uridine 5′-triphosphate)-biotin nick-end labeling (TUNEL) method. A. Myxoma cells in the outer zone (right lower section) were specifically stained by TUNEL, whereas those in the middle and inner zones showed slight TUNEL-positive staining. (Magnification, ×100) B. High-power photograph (×400) of myxoma cells in the inner zone showing TUNEL-negative staining. C. High-power photograph (×400) of myxoma cells in the outer zone showing strong TUNEL-positive staining.

Apoptosis in Cardiac Myxoma

To the Editor: Myxoma is well known as a cause of cardiac embolization (1, 2). However, the reason that embolic events frequently occur in patients with cardiac myxoma remains unknown. We report on myxoma cells that were strongly stained by the terminal deoxyribonucleotidyl transferase–mediated dUTP (deoxy uridine 5′-triphosphate)-biotin nick-end labeling (TUNEL) method.

A 78-year-old woman was admitted to our hospital because of a right cardiac tumor. She had recently experienced pulmonary embolism and full-body edema. After a 2.8-cm solid mass with lobulated surface was resected from the right atrial septum and was histopathologically diagnosed as cardiac myxoma, TUNEL was performed in paraformaldehyde-fixed, deparaffinized 5-μm-thick myxoma tissue using the Apop Tag kit (Oncor, Gaithersburg, Maryland). Myxoma cells were positively stained by TUNEL, indicating apoptosis (Figure).

To evaluate the distribution of apoptotic myxoma cells, the apoptotic index (3), expressed as the percentage of TUNEL-positive myxoma cells among total myxoma cells, was used in every five high-power fields of the one-third outer, middle, and inner zones, respectively. Eighty-four percent of myxoma cells in the outer zone were positively stained by TUNEL. In contrast, only 6% and 1% of TUNEL-positive myxoma cells were found in the middle and inner zones, respectively.

The incidence of embolization in patients with cardiac myxoma is 30% to 40% (2), and emboli are usually derived from the superficial portions of cardiac myxoma (4). Our findings indicate that cardiac myxoma tissue seems to be fragile because apoptosis leads to the disappearance of superficial myxoma cells. Therefore, we suggest that apoptotic myxoma cells may be related to embolic events in patients with cardiac myxoma.

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References

Propanolol Administration in a Patient with Thyroid Storm

To the Editor: Four weeks after stopping oral methimazole therapy, a 50-year-old woman with Graves disease presented with restlessness, palpitations, diaphoresis, and vomiting, without signs of heart failure. The thyroid-stimulating hormone level was undetectable, and free thyroxine and triiodothyronine levels were markedly elevated. Propranolol, saturated solution of potassium iodine, propylthiouracil, and hydrocortisone were administered. Hypotension developed 3 to 4 hours after each propranolol dose; this therapy was then discontinued, and the blood pressure was supported with norepinephrine (21 μg/min). Jugular venous distention, bibasilar crackles, an S3 gallop, and severe lactic acidosis (lactic acid level, 10.9 mmol/L [normal range, 0.5 to 2.2 mmol/L]) developed. The creatinine kinase level was 3060 U/L with a normal MB fraction. An echocardiogram showed four-chamber dilatation with hypokinesis and markedly reduced left ventricular function. The patient had a cardiac index of 1.1 L/min per m2 (normal, 2.4 to 4.0 L/min per m2) and a mixed venous oxygen saturation (SvO2) of 31.3% (normal, 70% to 75%). After a switch to dobutamine (12 g/kg of body weight per minute), the cardiac index improved to 3.1 L/min per m2, the SvO2 improved to 57%, and blood pressure and lactic acid level normalized. At hospital discharge 3 weeks later, the patient’s echocardiogram was normal.

Thyroid hormone increases the density of β-receptors and cyclic adenosine monophosphate and decreases density of α-receptors (1, 2). Plasma levels and urinary excretion rates of norepinephrine and epinephrine are normal in thyrotoxic patients (3). The hyperadrenergic state may be caused by hypersensitivity to catecholamines with upregulation of β-receptors (4). Thyrotoxicosis is associated with reversible and irreversible dilated cardiomyopathy, but the cause is uncertain (5). Increased oxygen demand may cause severe myocardial ischemia, but this was unlikely given our patient’s normal creatine kinase–MB measurement. The improvement with a β1-agonist and resolution of echocardiographic findings suggest that propranolol exacerbated subclinical thyroid cardiomyopathy and shock. Although propranolol may result in dramatic improvement of the signs and symptoms of thyroid storm, it should be used with caution and in a monitored setting.

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Ischemic Colitis Associated with Decongestant Use

To the Editor: Pseudoephedrine is a widely used over-the-counter nasal decongestant that has been associated with transient ischemic colitis in a woman who used oral contraceptives (1) and four perimenopausal women (2). It is unclear in those reports whether the changing levels of estrogen and progestrone and the associated thromboembolic effect contributed to the colitis (3). We report a case of ischemic colitis in a man who had used pseudoephedrine and did not have underlying thrombophilia or vasculitis.

A 33-year-old man presented with abrupt onset of lower abdominal cramps, diarrhea, and hematochezia of 2 days’ duration. He had chronic asthma and had been using albuterol and corticosteroid inhalers. He had no history of cigarette smoking or use of cocaine and amphetamine. For 5 days before presentation, the patient had taken Sudafed 12 Hour (Warner-Lambert, Morris Plains, New Jersey; pseudoephedrine hydrochloride extended-release tablets, 120 mg) twice daily for nasal congestion. The last dose was taken the morning of presentation. Colonoscopy revealed patchy erythema and mucosal edema in a segment of the descending colon, and colonic biopsy showed mucosal necrosis and edema with hemorrhage consistent with ischemic colitis. The mesenteric arteries were normal according to magnetic resonance angiography, and stools were negative for enteric pathogens. The presentation the symptoms have not recurred.

The patient discontinued using the decongestant. His abdominal cramps and bloody diarrhea resolved, and 10 months after presentation the symptoms have not recurred.

Thrombophilic evaluation for factor V Leiden mutation, prothrombin G20210A mutation, protein C, protein S, antithrombin III, plasminogen, homocysteine, and anticardiolipin antibody was unrevealing. Serologic studies for vasculitis, including antinuclear antibody and complements, were unremarkable. Echocardiogram with contrast and Doppler ultrasonography of bilateral lower extremities were normal.

This report further suggests that pseudoephedrine is an independent risk factor for ischemic colitis. We cannot prove that pseudoephedrine causes ischemic colitis or exclude the presence of an unknown thrombophilic condition. A case–control study is indicated to assess this hypothesis.

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References

Parvovirus B19 and Glomerulonephritis in a Healthy Adult

To the Editor: Human parvovirus B19 causes an exanthematous self-limiting childhood febrile illness and is associated with acute polyarthritis, myocarditis, vasculitis (1), hepatitis (2), and the nephrotic syndrome in sickle-cell disease (3). We describe glomerulonephritis in an otherwise healthy adult.

A 32-year-old woman presented with dyspnea and renal failure. Two weeks before presentation, a transient erythematous rash and swollen feet and fingers had developed. Her 8-year-old son developed a similar self-limiting rash at the same time.

The patient was pyrexial (temperature, 38.5°C), tachycardic, and tachypneic, with pulmonary and ankle edema. By presentation, arthritis and rash had resolved. Chest radiography confirmed pulmonary edema and cardiomegaly. The hemoglobin level was 8.8 g/dL (after recovery, it increased to 12.1 g/dL). The leukocyte count was 7.5 × 10^9/L, and transient lymphopenia was noted (lymphocyte count, 1.01 × 10^9/L). The C-reactive protein level was 120 mg/L (normal, <10 mg/L). Urea and creatinine levels were elevated (20.5 mmol/L and 167 µmol/L, respectively). Hematuria and proteinuria (2.16 g/24 h) were present. Renal ultrasonography, electrocardiography, and echocardiography were normal or insignificant. Blood cultures and results of serologic testing for rubella, Epstein–Barr virus, and respiratory viruses (including coxsackie B) were negative. Antibodies to antinuclear antibody, antineutrophil cytoplasmic antibody, rheumatoid factor, anti–double-stranded DNA, antineutrophil cytoplasmic antibody, and C3 and C4 were negative or normal. In the patient and her son, parvovirus B19 IgM was detected in serum samples obtained on admission. Renal biopsy showed acute proliferative glomerulonephritis (diffuse IgG, IgA, and C3 staining), and parvovirus B19 antigen was not detected in the specimen. The patient gradually recovered with careful fluid management. Within 3 months, parvovirus B19 IgM was not detectable and proteinuria and hematuria had resolved.

The temporal association suggests that parvovirus B19 infection precipitated glomerulonephritis. Lack of antigen detection in the biopsy specimen supports a probable immune-mediated cause (1). Parvovirus B19 infections in adults are rarely diagnosed, but they may be included in the differential diagnosis of patients with a recent febrile illness presenting with acute renal failure or the nephrotic syndrome. Treatment usually focuses on the symptoms, although immunocompromised patients may require intravenous immunoglobulins.

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