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Kawasaki Disease—A Disease with Anesthetic Implications

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Kawasaki disease, also known as mucocutaneous lymph node syndrome, is an acute exanthematous disease affecting primarily infants and young children. In 17–30% of the patients, the myocardium, coronary arteries, and/or great vessels are involved significantly resulting in a mortality rate of about 2%. We describe one patient scheduled for cardiac catheterization and discuss the pathophysiology and usual clinical course of patients with Kawasaki disease.

REPORT OF A CASE

An 18-month-old boy was scheduled for elective cardiac catheterization. Nine months previous, he was admitted with a three-week history of vomiting, diarrhea, fever, conjunctivitis, cough, and skin rash. His therapy included antibiotics and aspirin. With this initial admission he was alert but febrile, irritable, and photophobic with conjunctival erythema. His gingiva were hyperemic. Palpable cervical, axillary, and femoral lymph nodes were present. He had a heart rate of 160 beats/min, an arterial blood pressure of 100/40 mmHg, and a grade I/VI systolic ejection murmur with an S₃ gallop. The extremities showed a puffy non-pitting edema of the feet with no abnormalities of the joints. The skin showed a diffuse, well-marginated, elevated, erythematous eruption which was apparently not pruritic. Anemia and a leukocytosis with a left shift were found on complete blood count.

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Key words: Complications: Kawasaki disease; mucocutaneous lymph node syndrome. Surgery: cardiac. Anesthesia: cardiac. A chest roentgenogram demonstrated moderate cardiomegaly. Both twelve-lead electrocardiographic and M-mode echocardiographic studies were interpreted as normal. However, a two-dimensional echocardiogram demonstrated dilation of the proximal parts of both left and right coronary arteries. Kawasaki disease was diagnosed and 100 mg·kg⁻¹·day⁻¹ aspirin administered as treatment during his 13-day hospitalization. During this period the infant was continuously febrile until the last day. Gradual improvement in irritability and appetite was noted. Approximately six weeks following discharge, pulsatile masses in the axillary, brachial, and femoral arteries were first noted.

With this admission, the child was in no distress with no history of exercise limitations. His physical examination was normal except as related to the cardiovascular system. A I/VI systolic ejection murmur was again heard with no gallop or other evidence of failure. The electrocardiogram and chest roentgenograms were within normal limits. Two-dimensional echocardiographic examination again documented dilation of the proximal segments of the coronary arteries. Aneurysms of both axillary arteries, the right bracheal artery, and the right femoral artery were palpable.

For cardiac catheterization, the 11.8-kg child was premedicated with 12.5 mg meperidine, 0.12 mg atropine, 3 mg promethazine, and 3 mg chlorpromazine, im. Upon arrival in the cardiac catheterization suite, an intravenous line was established as well as monitoring which included a continuous electrocardiogram, a blood pressure cuff, a Doppler flow probe, and a temperature probe. Immediately prior to the infiltration of the cannulation site with 1% lidocaine, 1 mg/kg ketamine and 1 mg/kg thiopental were administered iv. The course of cardiac catheterization was uneventful and no further medications were required. Vascular pressures and oxygen saturation values obtained during the catheterization were within normal limits. Cineangiograms confirmed the presence of a right coronary artery aneurysm measuring approximately 1 by 2.5 cm. Blood flow through this aneurysm was very sluggish. Smaller aneurysms of the left main and left anterior descending arteries also were found.

The post-catheterization course was uneventful and the child was discharged the following day on 40 mg·kg⁻¹·day⁻¹ aspirin and 2 mg·kg⁻¹·day⁻¹ dipyridamole. The possibility of myocardial revascularization was entertained, but in view of the child's size and current clinical status, deferred for further consideration for several years.

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DISCUSSION

Kawasaki disease is an entity first described in 1967^{2,3} and in English language reports in 1974.^{4,5} Japan, with over 25,000 reported cases, appears to have the highest prevalence of the disease.^{1,3} In the United States, more than 650 cases of Kawasaki disease have been reported.¹ Epidemiologic studies of three recent outbreaks of the disease in the United States have failed to identify an etiologic factor.^{1,6} The disease is reviewed in several sources.^{3,5,7–10} There is a higher incidence of the disease in males, children of middle to upper middle socioeconomic classes, children of Japanese ancestory, and perhaps in children with an antecedent upper respiratory tract infection.

The underlying pathologic changes appear to be related to a systemic vasculitis involving both arteries and veins.11 Cardiovascular manifestations of the disease include carditis, ^{12,13} myocardial fibrosis ¹² endocardial fibroelastosis, ¹² conduction disturbances, ¹³ valvular dysfunction, ^{14,15} coronary artery aneurysms, ¹⁶ coronary artery stenosis, 12 coronary artery thrombosis, 12 and aneurysmal changes, thrombus formation, and rupture of large vessels.¹⁷ It is this involvement which is the cause of the 0.5-2.8% mortality rate associated with the disease. 1,12 Coronary artery lesions were found in 17% of unselected patients examined by arteriography one to six months following the onset of the disease. 18 Some of these patients with significant coronary lesions were totally asymptomatic. Sudden deaths two and four years following the acute episode have been reported. 16,19 Although serial study has demonstrated regression of about half of the aneurysms, the long-term effect on the arteries is not clear.20

Aspirin is generally used in addition to symptomatic treatment. The use of aspirin may reduce the incidence of coronary artery aneurysm formation. Dipyridamole is a vasodilator which in combination with aspirin prolongs platelet survival. As a result, it has been used with aspirin as a prophylactic agent. Surgical procedures which have been performed on children with Kawasaki disease include aortocoronary bypass grafting, 22-24 coronary aneurysmectomy, mitral valve replacement, and excision of large artery aneurysms. Infants also have been presented for general surgical procedures involving the biliary tract and bowel.

In the case presented, the child's initial clinical course was typical for Kawasaki disease. Despite the absence of clinical evidence for cardiac failure or ischemic changes, catheterization revealed aneurysmal involvement of both right and left coronary arteries.

The anesthetic considerations in these patients relate primarily to the associated cardiac manifestations. The preanesthetic evaluation should focus on signs and symptoms of cardiac failure, cardiac dysrhythmias, myocardial ischemia, and valvular dysfunction. Twelve-lead electrocardiography and two-dimensional echocardiography are indicated for the identification of involvement of coronary arteries. The anesthetic care of children with Kawasaki disease and coronary artery damage must insure a proper balance between myocardial oxygen demand and myocardial oxygen supply. This can be accomplished by the utilization of anesthetics and other drugs which allow manipulation of heart rate, myocardial contractility, filling pressure, and peripheral vascular resistance. In adult patients with coronary artery disease, numerous anesthetic agents and techniques have been used successfully. It is essential that vasoactive and antiarrhythmic drugs are readily available. Monitoring should allow early detection of myocardial ischemia and would include an electrocardiogram and blood pressure. In addition, direct blood pressure monitoring, central venous pressure, and pulmonary artery pressure monitoring may be indicated. Because Kawasaki disease may affect any of the coronary arteries, both leads II and V₅ of the electrocardiogram should be monitored in an effort to detect myocardial ischemia.

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Deep Body Thermometry during General Anesthesia

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Conventional techniques for measuring central or "core" patient temperatures during general anesthesia require passage of thermistor or thermocouple probes through body orifices into the nasopharynx, esophagus, rectum, or auditory canal. These techniques can cause tissue trauma and hemorrhage and are subject to sampling error because of imprecise placement of the probe. In this study of adults undergoing elective surgery, we measured the *in vivo* equilibrium and response characteristics of a commercially available non-invasive deep body thermometry (DBT) system using a heated skin surface probe designed to provide atraumatic approximations of core body temperatures. We then compared those characteristics to those of a conventional, passive, wedged nasal thermistor device.

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METHODS

The apparatus evaluated was the Thermo-Finer Coretemp® Model DC-1 DBT system† using a 40-g, 4.5cm diameter surface probe with a cast aluminum shell containing an electrical heating element and two thermistors separated by an insulator. The probe is connected by a flexible electrical cable to amplifier, comparator, and heater circuitry within an AC line-powered control box with digital display to the nearest 0.1° C. For each patient, one of two available probes was connected interchangeably to one of two control boxes used for this study. To obtain control values, one of four Yellow Springs Instruments® series 701 vinyl-covered thermistor probes was connected to the amplifier and readout circuits provided in a Tektronix® 414 electrocardiogram and pressure monitor with function controls set for digital display of temperature to the nearest 0.1° C. Prior to patient use, the Yellow Springs thermistor probes and the Coretemp probes were applied simultaneously to a temperature-controlled water bath; they were used for this study only if found to indicate the same temperature to within 0.1° C at 34.0° and 36.0° C. Both the DBT and Yellow Springs probe systems met

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