and has been attributed to chronic infection in a moist wound as well as to mechanical irritation from tube motion. Initially, granulation tissue is soft and vascular but as it matures it becomes fibrous and covered with epithelium.

Laser bronchoscopy has been used to treat obstructing lesions in the central tracheobronchial tree. It is particularly suitable for obliterating tumors which may be life-threatening. In experienced hands, laser treatment using a rigid bronchoscope under general anesthesia has the advantages of being safe and effective immediately. Cavaliere and colleagues utilized it in 37 cases of tracheal granulation tissue resulting from metallic tracheostomy tubes and suture threads after sleeve resection. Their results were almost always curative. Similarly, our patient demonstrated no recurrence of the lesion two years after Nd:Yag laser treatment, suggesting that the procedure was curative.

An obstructing central airway lesion may be associated with few symptoms, as in our patient who noted only minor “tracheal discomfort.” This may be particularly difficult to detect in a patient with underlying lung disease which produces similar signs and symptoms. Clues may include findings atypical for the patient’s disease (eg, positional dyspnea, localized wheezing) or unusual difficulty in removing or inserting the catheter. The true incidence of granulation tissue in the airway from a transtracheal catheter is unknown. However, it is important to consider this possibility since it is potentially life-threatening and amenable to treatment.

REFERENCES

Acute Myocardial Infarction Complicating the Clinical Course of Dilated Cardiomyopathy in Childhood*

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An acute myocardial infarction occurred in a 6-year-old child with dilated cardiomyopathy. This caused severe hemodynamic deterioration that led to a fatal outcome. Autopsy revealed diffuse myocardial atrophy without cell infiltration, normal epicardial coronary arteries, and a massive healed anteroapical infarction. Coronary embolism or spasm could not be ruled out as the cause of the infarction.

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Acutely myocardial infarction is extremely rare in children. Even though acute myocardial infarction may be detected in association with dilated cardiomyopathy in adults, this association has never been previously proved in childhood.

CASE REPORT

A 6-year-old white boy was referred because of progressive congestive heart failure lasting for 10 months. There was neither previously detected fever nor evidence of infectious disease. No clinical problems during pregnancy and delivery and no relatives with cardiac disease were reported.

The physical examination revealed a dyspneic child with blood pressure of 90/60 mm Hg and a regular pulse rate of 120 beats per minute. Edema of the lower limbs and face (+1/+4) and a third heart sound were noted. The liver was palpable 5 cm under the costal margin. The chest x-ray film showed moderate cardiac silhouette enlargement and pulmonary vascular congestion. The electrocardiogram (ECC) showed right and left atrial and left ventricular overload (Fig 1). Blood biochemical studies disclosed the following values: creatinine, 0.5 mg/ml; urea, 12 mg/ml; phosphorus, 4.5 mg/ml; and calcium, 9.7 mg/ml. On the basis of these findings, a diagnosis of idiopathic dilated cardiomyopathy was made. The patient was put on a regimen of digitalis and diuretics, with little improvement, and outpatient follow-up was recommended for further diagnostic evaluation.

One month later, the patient was admitted with severe substernal burning pain of recent onset accompanied by pallor, diaphoresis, vomiting, and intense dyspnea. The ECC (Fig 1) was compatible

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With acute transmural myocardial infarction in the anterior wall, subsequent cardiac enzyme assessment confirmed the presence of myocardial necrosis. The echocardiogram disclosed cardiac enlargement and diffuse left ventricular hypokinesis (fractional shortening = 13 percent). Cardiac catheterization showed marked elevation of both left and right ventricular filling pressures (36 and 26 mm Hg, respectively) and a cardiac index of 1.9 L/min/m². Left ventricular angiography revealed striking diffuse dilatation and hypokinesis (ejection fraction = 0.15). No intracardiac shunts, mural thrombosis, or organic valvular dysfunction could be detected. No obstructive lesions or abnormalities in the origins of both coronary arteries were found.

Progressive clinical deterioration ensued despite intensive care treatment, and cardiac arrest supervened two months thereafter. At autopsy, the massively enlarged heart weighed 350 g, and an anteroapical transmural infarction in the healing stage was seen. The epicardial coronary arteries were free of obstructive atherosclerotic lesions. All cardiac valves were normal, and no thrombus could be detected in the cardiac chambers. The left ventricular endocardium showed some areas of mild thickening. Microscopic study showed a diffuse pattern of myocyte hypertrophy intermingled with myocytolysis and atrophied cardiac fibers. Neither scattered necrotic areas nor mononuclear cell infiltrate foci were seen except near the myocardial infarction region. There was no evidence of myocardial fibrosis, and no arteritis could be seen. The anteroapical region showed scarring associated with interstitial edema and a focal perivascular mononuclear cell infiltrate (Fig 2).

**COMMENT**

Dilated cardiomyopathy in childhood often carries an ominous prognosis. Infections, arrhythmias, and thromboembolic phenomena are frequent complications that may lead to death. In the present case, a documented myocardial infarction was the clinical complication that ultimately led to irreversible pump failure.

There have been previous reports of pediatric patients with dilated cardiomyopathy showing ECG changes compatible with myocardial infarction. However, these changes were usually transient, or there was no angiographic and pathologic documentation of myocardial infarction. Therefore, our case is unique in that the clinical picture of acute myocardial infarction was morphologically confirmed.

Coronary spasm with subsequent thrombosis might be a cause of myocardial infarction in pediatric patients with dilated cardiomyopathy, even though coronary artery spasm has never been documented in this clinical group. Reduced coronary perfusion due to low-output cardiac failure or arrhythmia could also have been responsible for the myocardial ischemic event. However, no clinical data are available to support the arrhythmia hypothesis, because ambulatory ECG monitoring was not performed. Although no intracavitary thrombus has been detected, the possibility of coronary artery embolism cannot be ruled out either.

**REFERENCES**