the existence of adipose and fibrous tissue.

Barium enema showed abnormally high location of transverse colon situated immediately beneath the diaphragm. One year later, the patient represented for a follow-up visit and the chest roentgenogram showed an obvious large herniation of bowel loops through Morgagni’s foramen.

**DISCUSSION**

Large accumulation of adipose tissue in the peridiaphragmatic area usually represents herniation of abdominal fat either through a foramen or a defect in the diaphragm. A plain chest roentgenogram in the frontal projection shows paracardiac masses, and the identification of their nature is difficult. However, they are easily diagnosed by CT.2

The omentum contains adipose tissue which in the obese1 or after prolonged use of glycocorticoids4 may be massive. This is the most common abdominal compound that herniates through Morgagni’s foramen and is almost always located at the right side in the cardiophrenic angle.

Fat is also accumulated in the cardiophrenic angle and cardiac apex in mediastinal lipomatosis.5 Cushing’s syndrome, and after prolonged corticosteroid therapy.6

When the amount of it in the right cardiophrenic angle is large, it is necessary to make the distinction between herniated fat and epicardial fat pad. The identification of fine linear densities within the fat, which undoubtedly represent omental vessels, can be useful for this distinction.7

Barium studies do not have practical value since the herniated fat does not contain gastrointestinal loop. The small hernia of Morgagni’s foramen is asymptomatic and the differentiation is of no clinical importance. We consider that our case represents herniation of omental fat through Morgagni’s foramen caused by fat accumulation from prolonged corticosteroid therapy.

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**Continuous Six-Month Infusion of Intravenous Nitroglycerin in a Patient Awaiting Cardiac Transplantation**

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We report a patient with ischemic cardiomyopathy who was treated with a continuous high dose infusion of intravenous nitroglycerin for six months while awaiting cardiac transplantation. Surprisingly, methemoglobinemia did not develop and nitroglycerin continued to be clinically effective in relieving angina during the six-month period. We believe this to be the first reported instance of the safe use of such prolonged infusion of intravenous nitroglycerin.

(Chest 1991; 100:1470-71)

Due to an increasing number of cardiac transplant candidates and a relatively fixed donor pool size, the waiting period for transplantation has increased considerably in recent years.1 Medical management of transplant candidates with end stage congestive heart failure due to either ischemic or nonischemic cardiomyopathy often involves continuous intravenous inotropic and vasodilator therapy for long periods of time. Intravenous nitroglycerin (NTG) has been utilized in this setting for the treatment of both unstable angina pectoris and severe CHF. Prolonged use of intravenous NTG may have limitations since its hemodynamic effects attenuate within 6 to 8 h of continuous administration.2 Additionally, sustained infusions of large doses of intravenous NTG can cause methemoglobinemia.3

In this report, we describe a patient with ischemic cardiomyopathy who received a continuous six-month intravenous infusion of high dose NTG while awaiting transplantation.

**Case Report**

A 54-year-old white man was transferred to our institution for transplant evaluation. He had previous inferior and anteroseptal wall myocardial infarctions and two days prior to transfer, suffered an anterior wall MI. Because of persistent postinfarction angina, he was treated with intravenous NTG (40 μg/min). Electrocardiogram showed sinus rhythm and complete right bundle branch block.

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The hemoglobin-methemoglobin levels were measured at frequent intervals during the six-month infusion of intravenous NTG (Fig 1). He did not have any clinical evidence of methemoglobinemia, but on one occasion, the serum methemoglobin level was elevated (normal 0 to 2 percent of total hemoglobin) and returned to normal with reduction in the dose of NTG.

**DISCUSSION**

This case report illustrates the safety of a high dose sustained infusion of intravenous NTG. Our patient had no side effects attributable directly to the NTG infusion. High plasma concentrations of NTG can induce methemoglobinemia by oxidizing hemoglobin to methemoglobin faster than the methemoglobin reductive enzyme system can reconvert it. Because of the inability of methemoglobin to bind reversibly with molecular oxygen, the oxygen-hemoglobin dissociation curve is displaced to the left. This can potentially exacerbate angina in patients with coronary artery disease by disrupting the balance between oxygen supply and demand. The development of methemoglobinemia is thought to relate to the cumulative dose of NTG, but in our patient, no correlation between plasma methemoglobin level and cumulative NTG dosage was seen.

Our patient was relieved of chest pain with increments in intravenous NTG dose, although he was repeatedly unresponsive to sublingual NTG. In view of his coronary anatomy, it was presumed that the chest pains represented myocardial ischemia. The need for increasing doses of intravenous NTG suggested the development of nitrate tolerance. Tolerance to nitrates is a complex phenomenon, and it is believed to be due to exhaustion of intracellular sulphydryl groups that are required for generation of S-nitrosohiol and cyclic guanosine monophosphate. It is thought to be unrelated to the pharmacologic formulation of the particular nitrate. Cross tolerance to sublingual NTG may occur in patients receiving long-term oral and intravenous nitrate preparations. Animal studies indicate an inverse relationship between plasma nitrate levels and onset of hemodynamic tolerance during prolonged nitrate therapy, but the relationship between nitrate blood levels and development of clinical tolerance in humans is unknown. Despite the occurrence of hemodynamic tolerance with long-term use of nitrates, it appears that in many patients, clinical responsiveness such as relief of angina may persist, as was observed in our patient.

We conclude from this case report that high doses of intravenous NTG can be administered safely for sustained periods of time and may be clinically effective in patients with refractory angina. This form of therapy may provide an alternative to mechanical assistance (such as intraaortic balloon pump) in the pretransplant management of selected patients with ischemic cardiomyopathy and unstable angina.

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