usual in patients with Down's syndrome.4

Our patient had in neonatal age a perimembranous inlet VSD without any evidence of aneurysm of the membranous septum.

At three months of age, a large aneurysm of the membranous septum was observed on the two-dimensional echocardiography and confirmed at cardiac catheterization and magnetic resonance imaging that showed, at eight months of age, the evidence of complete closure of the VSD (Fig 2).

To our knowledge, spontaneous closure of perimembranous inlet VSD has not been described before in a patient with Down syndrome and associated aortic coarctation.

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REFERENCES

Atrial Fibrillation Following Pulse Methyiprednisolone Therapy in an Adult*

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The authors believe this to be the first case report of atrial fibrillation following pulse methylprednisolone therapy in an adult. The literature regarding the complications of pulse methylprednisolone therapy is reviewed, with particular emphasis on the incidence and pathogenesis of arrhythmias. (Chest 1993; 104:622-23)

High doses of corticosteroids are standard treatment for a vast array of medical disorders. While the chronic effects of steroids are well described, there is less information on the physiologic effects of acute high-dose steroid administration. This report describes the first documented case of atrial fibrillation following high-dose methylprednisolone therapy in an adult.

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The statements contained herein are those of the authors and do not necessarily reflect those of the Navy Department or Naval Service at large.

Case Report

A 22-year-old Filipino man presented to our emergency room in September 1990 with the chief complaint of a rapid heart rate. The past medical history was remarkable for multiple sclerosis since early 1988. During a 3-day admission to the neurology service earlier in the month the patient had received 3 doses of methylprednisolone, 1 g IVPB, each dose administered over a period of 2 h on 3 consecutive days. Approximately 4 h following his final dose of methylprednisolone, the patient noted the acute onset of hiccoughs, followed 2 h later by a rapid, irregular heart rate.

This first episode spontaneously resolved, but both the hiccoughs and the rapid heart rate recurred, resulting in the patient's presentation to our emergency room. He denied the use of alcohol and caffeinated beverages, and was taking no medications or drugs. The medical history was significant only for multiple sclerosis. The ECG showed atrial fibrillation with a rate of 145 beats per minute. The chest x-ray film, serum electrolyte values, thyroid function test results, and echocardiogram were normal. The patient was admitted to the cardiology ward. Digoxin was administered intravenously for rate control, and chlorpromazine was given for control of the hiccoughs. Approximately 3 h after admission both the hiccoughs and the atrial fibrillation resolved. They have not recurred after 1 year of follow-up.

Discussion

This case illustrates the onset of atrial fibrillation following high-dose intravenous corticosteroid therapy. Only two such cases have been previously described in the literature, both in children with the nephrotic syndrome.1 Our patient represents the first documented case of atrial fibrillation following pulse methylprednisolone therapy in an adult. In their review of 50 adult patients receiving methylprednisolone pulse therapy, Garrett and Paulus2 noted that 2 patients experienced palpitations, but there was no ECG documentation of an arrhythmia. Sudden death has been reported following the rapid (30 min to 2 h) intravenous infusion of methylprednisolone, with all patients described having severe multisystem illnesses and no clear evidence of a precipitating arrhythmia.3

Interestingly, our patient's atrial fibrillation was preceded temporally by paroxysms of hiccupping. Most patients who receive pulse methylprednisolone do not experience atrial fibrillation. We propose that this patient's hiccupping lowered the stimulus threshold for the induction of atrial fibrillation. Experiments in dogs suggest that enhanced local release of acetylcholine within the myocardium lowers the atrial fibrillation threshold.4 A similar mechanism may be at least partially operative here—an interplay of autonomic influences in the setting of a myocardium exposed to high doses of steroids tips the balance toward production of an arrhythmia. One possible scenario is that repetitive phrenic stimulation induced by hiccupping activates vagal efferents to the heart, which, in the presence of a myocardium exposed to high levels of steroids, induces atrial fibrillation.

In addition to autonomic influences on arrhythmogenesis, the role of electrolyte shifts deserves consideration. Fujimoto et al5 measured serum sodium and potassium, as well as the fractional excretion of these electrolytes, before and during pulse methylprednisolone therapy in 20 patients. They observed a small but significant increase in serum potassium together with increased urinary potassium excretion over the 3-day course of therapy. Increased urinary potassium loss was independent of the effects of diuretics. Although

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these changes did not directly correlate with arrhythmic events in their patients, they postulated that methylprednisolone mediates potassium efflux via a direct effect on the cell membrane. Local potassium efflux may in turn influence arrhythmogenesis.3

These data underscore the need for monitoring serum electrolytes. Because high-dose steroid use promotes potassium loss at the renal level, serum potassium should be measured before treatment and deficits should be corrected. This may be especially important in patients receiving diuretics, those with prior courses of methylprednisolone, and those in whom the half-life of the drug is prolonged due to hepatic dysfunction. Furthermore, the effect of intramyocardial potassium fluxes on arrhythmogenesis is impossible to predict. Given this uncertainty, telemetry monitoring should be considered for those patients with known or suspected myocardial disease or severe multisystem illnesses characterized by a changing electrolyte or acid-base status. Our patient did not fall into any of these risk groups and had normal serum electrolyte levels.

In summary, this represents the first documented case of atrial fibrillation following pulse steroid therapy in an adult. Given the varied and sometimes nonspecific indications for the use of steroids, vigilance is indicated when administering high-dose pulses of these medications. Pulse doses should be administered with close attention to concomitant medical problems and the avoidance of a physiologic state that might favor arrhythmogenesis. Potassium should be monitored before treatment, deficits corrected, and telemetry considered for high-risk patients. In our patient, early control of hiccupping might have prevented the onset of atrial fibrillation in the setting of high-dose steroid therapy.

REFERENCES

A guidewire, inserted into a 65-year-old patient with multisystem organ failure through the left subclavian vein, intertwined and knotted with an existing central venous catheter previously inserted via the right subclavian vein. Both the guidewire and catheter were removed without any harm to the patient.

Chest 1993; 104:623-24

Guidewires simplify the catheterization of central veins.1,4 Rare complications have been reported with their use.3,7 We present an unreported complication of a guidewire knotting with an existing central venous catheter.

CASE REPORT

A 65-year-old man was treated in the ICU after surgery for a ruptured aortic aneurysm complicated with multisystem organ failure. Ventilation was achieved with the use of a nasotracheal tube, and hemodialysis was performed with a catheter in the left femoral vein. Total parenteral nutrition, fluids, and drugs were administered through a double-lumen central venous catheter, which easily had been inserted using a right infraclavicular approach, a guidewire, and a vessel dilator. Four days later, due to a suspected line sepsis, it was decided to first insert a new double-lumen central venous catheter via a left infraclavicular approach and thereafter remove the right-sided catheter. Using the Seldinger technique, the left subclavian vein was located, and a guidewire easily was inserted. Then over the guidewire, after the forceful and difficult insertion and removal of the vessel dilator, the double-lumen catheter was threaded into the superior vena cava. However, the double-lumen catheter had to be removed because the guidewire could not be pulled out through it. It then became apparent that the guidewire was stuck.

A chest x-ray film (Fig 1) showed the existing right-sided double-lumen catheter in the right atrium intertwined and hooked by the left-sided guidewire. Careful traction of the catheter failed to dislodge it from the guidewire. A repeat chest x-ray film showed that the knot had moved into the superior vena cava. With gentle simultaneous traction of the double-lumen catheter and insertion of the guidewire, they were freed from each other and both easily pulled out with no harm to the patient.

Afterward, examination of the catheter and guidewire revealed a kink of the guidewire about 10 cm from the distal J-end which probably caught and knotted with the catheter about 7 cm from its base.

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Intertwining and Knotting of a Guidewire With a Central Venous Catheter*

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FIGURE 1. Initial chest radiograph showing the existing right subclavian double-lumen catheter intertwined and knotted with the newly inserted guidewire at the entrance to the right atrium.