clussions in the submucosa of the ulcerated ileum were also positive. Occasional epithelial cells were positive in the lungs and bile ducts.

Virology
Adenovirus was cultured from routine clinical samples and also from postmortem samples of lung and small intestine. The virus was identified as type 32 by neutralisation of infectivity using hyperimmune guinea-pig antiserum.

Discussion
Previous reports have confirmed the serious pathogenic potential of adenovirus in immunocompromised transplant recipients. Diarrhoea caused by adenovirus infection in bone marrow transplant recipients is well recognised and needs to be distinguished clinically from graft versus host disease. Many adenovirus types have been recognised in these infections including types 2, 5, 11, 12, 31, and 35. This is the first report that we are aware of adenovirus type 32 causing similar disease in a bone marrow recipient, although isolation of this serotype has been documented in cases of secondary immunodeficiencies such as lymphoma and AIDS, causing hepatosplenomegaly, encephalitis and gastroenteritis, respectively.1 Diarrhoea caused by adenovirus types 40 and 41 is well recognised in the immunologically competent host.

In this case the virus gave rise to bowel symptoms including diarrhoea, a severe hepatitis and probably directly, as well as indirectly through the hepatitis, contributed to the adult type respiratory distress syndrome.

The probe used is reported to be specific for adenovirus types 5, 7, 11, 20, 40, and 41, but there is considerable homology in the DNA sequences and so other adenovirus types may be detected. Adenovirus should be considered in the differential diagnosis of serious sepsis in the immunocompromised patient, and is not an uncommon infection in these patients. In situ hybridisation offers a quick and sensitive method of viral detection, detecting the virus in cells without obvious nuclear inclusions on histology.

The authors thank Professor J S Lilleyman for referring this patient.


Sudden death from coronary artery dissection

A C Bateman, P J Gallagher, A C Vincenti

Abstract
Spontaneous dissection of the coronary arteries is an uncommon condition that may lead to sudden, fatal coronary artery occlusion. Three cases of sudden death attributable to coronary artery occlusion are presented. Dissection was associated with Marfan's syndrome in the first case, and occurred three weeks postpartum in the second case. In case 1, dissection occurred within the intima, and was not associated with an inflammatory cell infiltrate. In cases 2 and 3, dissection occurred between the tunica media and the external elastic lamina, and was associated with a mixed inflammatory infiltrate, rich in eosinophils, T lymphocytes, and histiocytes. The spatial limitation of the inflammatory infiltrate to the adventitial compartment, together with the absence of inflammation in case 1, suggests a reactive origin rather than a causative role for the inflammatory cells. Detailed examination of serial blocks of any coronary artery occlusion is essential in young patients.

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Keywords: Coronary arteries, dissection, sudden death, inflammation.

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She suffered further chest pain on rising the following morning. This pain became more severe and persisted until about 1050 hours, when she collapsed and suffered a cardio-respiratory arrest. Attempts at resuscitation were unsuccessful. Her second child had been delivered three weeks previously. The pregnancy was uneventful, but 27 hours after the onset of labour, she underwent Caesarean section for cephalopelvic disproportion. For the past three years, she had suffered from indigestion, relieved by cimetidine. There were no other features in the past medical history. At necropsy, there were no external features of Marfan's syndrome. The heart was of normal size (280 g), and sectioning the left ventricle revealed pallor of the anterior wall. The left anterior descending coronary artery was completely occluded by recent thrombus, 1-5 cm from its origin. The remaining coronary arteries were fully patent, and the cardiac valves were normal. Both lungs showed severe pulmonary oedema.

**Case reports**

**CLINICAL DETAILS**

### Case 1

A 25 year old man collapsed and died whilst walking to a bus stop. He suffered from mental handicap and probable Marfan's syndrome. His arm span (197 cm) exceeded his height (192 cm), echocardiography had revealed aortic root dilatation (diameter 4-2 cm; normal range 2-0–3-7 cm), and liver biopsy had revealed cystic medial degeneration within small arteries. His past medical history included hypertension, and thromboses within the splenic and mesenteric arteries. Serum antithrombin III estimation was normal, and homocystinuria had been excluded by repeatedly normal urine amino acid chromatography. At necropsy the heart weight was increased (746 g) mainly due to left ventricular hypertrophy (maximum thickness of left ventricle 3-5 cm). Sectioning the left ventricle revealed scarring in the posterior wall, suggestive of previous myocardial infarction. The left main coronary artery was distended at its origin, and filled with thrombus. The right coronary artery and the cardiac valves appeared normal. Both lungs showed severe vascular congestion and pulmonary oedema.

### Case 2

A 34 year old woman developed severe pain in her chest and arms at 2230 hours, which initially appeared to be relieved with oral antacids.

### Case 3

A 40 year old woman felt acutely unwell and developed pain in her chest and arms. No obstetric history was available. About one hour later, she had a cardiorespiratory arrest. Attempts at resuscitation were unsuccessful. She had complained of chest pain one year previously, thought by her general practitioner to be of muscular origin. Since that time, she had suffered occasional shortlived episodes of minor chest pain. There were no other features in the past medical history. At necropsy, there were no features of Marfan's syndrome. The heart was of normal size, and sectioning of the left ventricle revealed an area of fibrosis in the posterior wall, consistent with previous myocardial infarction. One of the diagonal branches of the left anterior descending coronary artery appeared to be occluded by recent thrombus. The right coronary artery contained an old occlusion. The cardiac valves were normal.

**HISTOLOGICAL EXAMINATION**

In case 1, microscopic examination of the left main coronary artery revealed moderate intimal thickening, associated with a dissection within the intima. There was no evidence of mucoid degeneration within the artery, and there was no surrounding inflammatory cell infiltrate. There was no evidence of a systemic vasculitis.

Microscopy of the occluded segment of the left anterior descending coronary artery in case 2, and the diagonal branch of the left anterior descending coronary artery in case 3, revealed similar appearances. Each artery contained a large dissection situated between the outer part of the tunica media, and the external elastic lamina (figure). Alcian blue staining revealed no evidence of cystic medial degeneration in either case. In both cases the adventitia and surrounding adipose tissue contained a polymorph inflammatory cell infiltrate, consisting of moderate numbers of eosinophils and mono-

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**Factors associated with coronary artery dissection**

<table>
<thead>
<tr>
<th>Associated factor</th>
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<tbody>
<tr>
<td>Pregnancy and recent childbirth</td>
<td>Corrado et al²</td>
</tr>
<tr>
<td>Connective tissue diseases (for example, Marfan's syndrome)</td>
<td>Corrado et al²</td>
</tr>
<tr>
<td>Chest trauma (for example, external cardiac massage)</td>
<td>Corrado et al²</td>
</tr>
<tr>
<td>Coronary artery angiography and angioplasty</td>
<td>Cripps et al²</td>
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<tr>
<td>Coronary artery bypass grafting</td>
<td>Cripps et al²</td>
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nuclear cells, and occasional neutrophils. This infiltrate did not extend into the tunica media. The mononuclear cell component was further characterised by immunohistochemistry, using the antibodies L26 (CD20), MB2, polyclonal CD3, UCHL-1 (CD45R0), and PGM-1 (CD68). This revealed a high proportion of macrophages and moderate numbers of T, but few B, lymphocytes. No evidence of systemic vasculitis was detected in either case.

**Discussion**

Dissection of the coronary arteries is an uncommon condition. It was first described in 1931, and occurs most frequently in young women. Corrado et al. found coronary artery dissection in three of 150 cases of sudden death in subjects aged 35 years or less. The left anterior descending coronary artery is most commonly involved. The factors predisposing to coronary artery dissection are summarised in the table. Two of the three cases in the present report possessed such associations. Case 1 was a diagnosed example of Marfan’s syndrome, and case 2 was a young woman in the postpartum period. The hormonal and other physiological changes associated with pregnancy, and the physical strain of labour have been proposed as contributory factors in this association, but the exact mechanism remains unclear. Case 3 differed in that no predisposing factor could be identified.

Most cases of spontaneous coronary artery dissection, such as the three detailed in this report, present with sudden death. Dissections occurring during invasive coronary artery procedures may also result in angina, bradycardia and other cardiac arrhythmias, and myocardial infarction.

Coronary artery dissection may be compatible with survival, especially when it occurs during procedures such as coronary angiography. Cripps et al. found “extensive” dissection complicating 3-6% of 880 patients undergoing coronary angioplasty. In these circumstances, symptoms may settle following conservative management. In the study by Cripps et al., 20 of 32 patients were successfully managed medically. However, 12 patients required immediate coronary artery bypass grafting. One case of spontaneous postpartum coronary artery dissection has been successfully treated by cardiac transplantation.

Histological examination of involved coronary arteries reveals a plane of dissection usually within the outer third of the tunica media. The three cases in the current report demonstrate unusual planes of dissection. The dissection occurred within the intima in case 1, and between the outer tunica media and the external elastic lamina in cases 2 and 3. In many previously reported cases the connective tissue surrounding the abnormal coronary artery contained an inflammatory cell infiltrate rich in eosinophils. The significance of this inflammatory infiltrate is unclear. Robinowicz et al. noted the prominence of eosinophils, have suggested a causative role for the inflammatory infiltrate, possibly through damage to the arterial wall via the release of lytic substances from eosinophils, including major basic protein. One case report documents coronary artery dissection in a 42 year old woman with a long history of asthma, and suggests that the predominance of eosinophils provides evidence of a hypersensitivity vasculitis. However, apart from the history of asthma, the features of this case do not differ significantly from the other reported cases of coronary artery dissection. Dowling and Buja found that the inflammatory infiltrate was limited to the adventitia and concluded that the inflammation is more likely to be reactive in origin.

In this report, we have demonstrated a mixed inflammatory infiltrate surrounding the abnormal coronary arteries in two cases. The infiltrate in both cases contained a moderate proportion of eosinophils, but significant numbers of macrophages were also present within the adventitia and surrounding adipose tissue, encroaching on the coronary artery lumen. However, the tunica media did not contain any evidence of inflammation. The high proportion of macrophages and lymphocytes present indicates an element of chronicity, and may suggest a causal relationship. However, it is difficult to envisage why an inflammatory process should remain so sharply confined to one anatomical compartment of the arterial wall. In coronary vasculitis due to Kawasaki’s disease, inflammation involves the entire thickness of the arterial wall. Similarly, arteries involved by giant cell arteritis or Takayasu’s disease may show inflammatory involvement of all layers of the vessel wall. An alternative explanation for the appearance of the inflammatory infiltrate is that it represents a reaction to arterial dissection that has occurred gradually, over a period of at least many hours. Thus, the tunica media, once detached from the external elastic lamina, may not become involved by an inflammatory infiltrate that originates from venules within the adventitia. At a later stage, acute occlusion of the coronary artery could be triggered by a small intimal tear, allowing blood to fill the “potential” space previously created by detachment of the media from the external elastic lamina. Interestingly, no inflammatory cell infiltrate was associated with the coronary artery dissection in case 1. Although the plane of dissection was different in this case, this observation provides further evidence that inflammation is not a prerequisite for dissection.

A proportion of cases of coronary artery dissection, particularly those associated with Marfan’s syndrome, show mucoid degeneration within segments of coronary artery sampled from distant sites, and it has been suggested that this medial degeneration could predispose to dissection. Cystic medial degeneration of the aorta is not uncommonly detected in patients with Marfan’s syndrome, and may be associated with aortic dissection. In these cases direct extension of the dissection into the coronary arteries may occur. Similar changes have been documented within the coronary arteries of these patients. No medial degeneration was detected within the coronary
arteries of the three cases presented in this report.

Coronary artery dissection remains a rare phenomenon, which nevertheless may present to histopathologists as unexplained sudden death. Coronary artery dissection should be considered in cases where an apparent coronary artery thrombosis is present, particularly in young patients with no evidence of coronary atherosclerosis, who may possess one of the documented associated factors. The left main stem and left anterior descending coronary arteries should be examined in particular detail, as most dissections occur within these vessels. All coronary arteries containing apparent thrombosis should be carefully examined histologically in this group of patients. A detailed examination of this nature is especially warranted in cases such as these, because the affected patients are usually young and previously apparently healthy, and may die suddenly for no immediately apparent reason.

We thank Professor C F George for his permission to include the first case in this report.

Sudden death from coronary artery dissection.

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