Despite custodial care he contrived to sniff glue on two further occasions. Both of these episodes were associated with a recurrence of loin pain, haematuria, and proteinuria. Although urea and creatinine concentrations remained normal, microscopic haematuria persisted after six weeks' abstinence from glue.

Comment

We believe this case to be an example of renal damage caused by the inhalation of toluene in glue. Renal failure has been recorded after the abuse of other solvents such as carbon tetrachloride[2] and trichloroethylene.[3] O'Brien *et al*[4] reported a case of hepatorenal damage after inhalation of toluene from a liquid cleaner, but similar damage from the toluene in glue has not been recorded. Since this adhesive is now the most widely abused agent in the west of Scotland,[1] and probably in Britain, it is important to realise that renal damage may result from such abuse.

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[1] Watson JM. Clinical and laboratory investigation in 132 cases of solvent abuse. *Medicine, Science and the Law* 1978;**18**:40-3.
[2] New PS, Lubash GD, Scherr L, Rubin AL. Acute renal failure associated with carbontetrachloride intoxication. *JAMA* 1962;**181**:903-6.
[3] Baerg RD, Kinberg DV. Hepatic necrosis and acute renal failure in solvent sniffers. *Ann Intern Med* 1970;**73**:713-20.
[4] O'Brien ET, Yeoman WB, Hobby JAE. Hepato-renal damage from toluene in a glue sniffer. *Br Med J* 1971;ii:29-30.

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Reactivation of vasculitis after influenza vaccination

In the past few years use of viral influenza vaccines has increased. Despite criteria regulating their administration, particularly in immunologically depressed patients, they are often overprescribed without consideration of possible adverse reactions.

Case report

A 55-year-old woman was admitted to hospital because of an ataxic gait and progressive weakness. She had been well until six months previously, when after an influenza-like syndrome she had become febrile and fatigued, with arthralgia and unsteady gait. During the next months these symptoms worsened and she lost weight. There was no history of allergic reactions. On admission she was alert but pale, weak, and febrile. She had brisk reflexes on her right side and an ataxic gait, tending to fall to the right.

Packed cell volume was 30%, white cell count $7{\cdot}9 \times 10^9$/l with neutrophils without eosinophils, platelet count 355×10^9/l, and erythrocyte sedimentation rate (Westergren) 85 mm in the first hour. Lupus erythematosus cells were not seen. Concentrations of C3, C4, immunoglobulins, cryoglobulins, blood urea, bilirubin, glucose, and lipids and results of liver function tests were normal. Serum creatinine concentration was 61·98 μmol/l (0·7 mg/100 ml), and protein electrophoresis showed 70 g total protein/l with an increase in α_2-globulin. A lumbar puncture showed no abnormalities of cerebrospinal fluid. Urinary protein excretion was 1·5 g/24 hours, the sediment containing 40-50 red cells, 30-40 white cells, and many casts per high-power field. Urine cultures were negative. *X*-ray films of the chest, an intravenous urogram, an abdominal aortogram, and a carotid angiogram were normal. A renal biopsy specimen showed necrotising vasculitis of small- and medium-sized arteries and a focal glomerulonephritis with granulomatous infiltration around the glomerular tufts.

Prednisone 1 mg/kg/day was started. In a few weeks she showed clinical and biochemical improvement, the erythrocyte sedimentation rate being 15 mm in the first hour. The prednisone dose was decreased gradually to a maintenance dose of 12·5 mg/48 hours, and she remained well.

Eighteen months later 900 IU of a killed mixovirus influenza vaccine preparation was administered without our knowledge (315 IU of A/Bangkok/1/79(H_3N_2), 315 IU of A/USSR/90/77(H_1N_1), and 270 IU of B/Singapore/222/79). Ten days later she was readmitted to hospital with a recurrence of her original symptoms (fever, weakness, arthralgia, ataxic gait) and an erythrocyte sedimentation rate of 84 mm in the first hour. Prednisone was increased to 1 mg/kg/day and cyclophosphamide added to treatment. In two weeks she had improved; prednisone was again gradually decreased and she was discharged. Twelve months later she remained well taking low-dose prednisone on alternate days and cyclophosphamide.

Comment

Vasculitis may be triggered by several types of antigenic substances,[1] [2] but many unknown mechanisms exist to which it might be related. Often it appears after a short episode of an influenza-like syndrome, suggesting that viral infections may precede it, but this is difficult to prove. Traditionally the vasculitis syndrome may start with the same unspecific, non-infectious symptoms as influenza, making a distinction between both diseases almost impossible and therefore blurring the true incidence of their association.

Influenza vaccines are useful in certain subjects, particularly the immunologically depressed,[5] but are often used more widely than is specifically indicated. Doctors should bear in mind that influenza vaccines, especially the broad-spectrum ones (trivalent), contain several types of attenuated viruses, all with their own antigenic power; thus the broad-spectrum vaccines are more likely to induce a wide antibody response and activate some immunological disturbance.

As knowledge of agents that cause systemic disease is still insufficient, doctors should be cautious in prescribing influenza vaccines as they may trigger some immunological illness that may itself be worse than most influenza syndromes.

[1] Mordes JA, Johnson MK, Soter NA. Possible naproxen-associated vasculitis. *Arch Intern Med* 1980;**140**:985.
[2] Phanuphak P, Kohler PF. Onset of polyarteritis nodosa during allergic hyposensitization treatment. *Am J Med* 1980;**68**:479-85.
[3] Wharton CFP, Pietroni R. Polyarteritis after influenza vaccination. *Br Med J* 1974;ii:331-2.
[4] Blumberg S, Bienfang D, Kantrowitz FG. A possible association between influenza vaccination and small vessel vasculitis. *Arch Intern Med* 1980;**140**:847-8.
[5] Center for Disease Control. Influenza 1980-1981. Recommendation of the Public Health Service immunization practices advisory committee. *Ann Int Med* 1980;**93**:466-8.

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Recurrent pericarditis: a rare complication of influenza vaccination

Viral infection of the respiratory tract caused by influenza is common and widespread during the winter. The usual benign course of the disease is sometimes complicated by bacterial pneumonia, myocarditis, and pericarditis.[1] These complications are especially dangerous in the elderly and compromised hosts. A trivalent vaccine preparation of inactivated influenza virus antigen is available for this group of patients, and its composition is adjusted according to the new virus serotypes appearing each year. Adverse reactions to the vaccine that have been reported are local skin reactions, systemic symptoms of toxicity, allergic reactions, and the Guillain-Barré syndrome[2-4]; we report on a patient who developed pericarditis.

Case report

A 61-year-old man was admitted because of a fever of 38°C and a continuous chest pain aggravated by breathing and changing his position. A week before admission he had received an intramuscular injection of influenza vaccine containing A/Bangkok/1/79/(H_3N_2), A/Brazil/11/78/(H_1N_1), and B/Singapore/222/79 (Vaccigrip-Institut Marieux, Paris). Physical examination disclosed a fever of 37·5°C and a pericardial friction rub. Laboratory examination showed erythrocyte sedimentation rate 70 mm in

first hour, haemoglobin 13·1 g/dl, and white cell count 11·5 × 10^9/l. Antinuclear and anti-heart-muscle antibodies were absent and complement concentration normal. Chest x-ray films showed slight cardiac enlargement, and on echocardiography a small amount of pericardial effusion was detected. Treatment with aspirin 2 g daily was started with rapid clinical improvement. Two weeks later the echocardiogram was normal.

He had first suffered from fever and chest pain two years previously; the symptoms had begun 10 days after an influenza vaccination with A/Texas/1/77/(H_3N_2), A/USSR/9/77/(H_1N_1), and B/Hong Kong/5/72 (Vaccigrip-Institut Marieux). A clinical diagnosis of acute benign pericarditis had been confirmed by echocardiography, and the symptoms had completely resolved after a month of steroid treatment.

Comment

The distinct time interval between the appearance of pericarditis and administration of the vaccine on two consecutive occasions suggests that the recurrent pericarditis was a direct consequence of the vaccinations. As the two vaccines were of different antigenic composition and no signs of autoimmune disease could be detected the exact mechanism of the pericarditis remains obscure. To the best of our knowledge pericarditis has not previously been reported as a complication of influenza vaccine.

[1] Hildebrandt HM, Maassab HF, Willis PW. Influenza virus pericarditis. *Am J Dis Child* 1962;**104**:179.

[2] White CS III, Adles WH, McGann VG. Repeated immunizations: possible adverse effects. *Ann Intern Med* 1974;**81**:594.

[3] Center for Disease Control. Influenza vaccine 1980-1981. *Ann Intern Med* 1980;**93**:466-8.

[4] Clinical studies of influenza vaccines 1976: a conference held at the National Institutes of Health, Bethesda, Maryland, 20-21 January 1977. *J Infect Dis* 1977;**136**, suppl:S341-742.

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Periampullary adenoma causing pancreatitis

Benign duodenal tumours are rarely of clinical importance, but those in the region of the ampulla may present with obstructive jaundice,[1] the preoperative diagnosis usually being pancreatic carcinoma. We report a case of an adenoma of the duodenal papilla which presented with acute pancreatitis.

Case report

A 68-year-old widow was admitted as an emergency after severe epigastric pain and vomiting for 20 hours. She gave a three-year history of upper abdominal pains, mostly after meals, which had become more severe during the preceding six months, when she had also lost 3 kg. Her epigastrium was very tender, and her serum amylase activity was raised at 6470 IU/l (normal range 86-268 IU/l). Results of liver function tests were normal apart from a raised serum alkaline phosphatase activity of 186 IU/l (normal range 22-92 IU/l). Plain abdominal radiographs and ultrasonography of her upper abdomen showed no abnormality. After three days' conservative management she was much improved, her serum amylase activity had fallen to 698 IU/l, and she went home. An oral cholecystogram three weeks later showed no abnormality, and her serum amylase activity was normal.

Two weeks later her symptoms returned, in particular epigastric pain, usually after meals with occasional vomiting. Findings of barium-meal examination were reported as normal, and examination of the stomach and duodenal bulb with the standard end-viewing gastroscope showed no abnormality. Her symptoms continued to fluctuate in severity over the next two months, and she lost 6 kg. Her serum alkaline phosphatase activity remained raised at 305 IU/l, but she was not jaundiced and at no time was a mass palpable in her epigastrium. A pancreatic cause for her symptoms was strongly suspected, and endoscopic retrograde cholangiopancreatography showed an irregular, mobile mass (2 cm in diameter) in the second part of the duodenum, in the region of the ampulla of Vater. Four biopsy specimens were taken and these showed fragments of villous mucosa with no evidence of malignancy. Cannulation of the ducts was not possible owing to the mass. At laparotomy (8 July 1980) the duodenum was opened to confirm a tumour 2 cm in diameter arising from the papilla. The tumour was completely excised with diathermy, and the common bile duct and pancreatic ducts were reimplanted as an extended sphincteroplasty. Histology confirmed the mass to be a benign adenomatous polyp with the main ducts running through its centre.

The patient made a good recovery apart from a wound infection, her symptoms have gone, and all investigations have since shown no abnormality.

Comment

This is the first reported case to our knowledge of acute pancreatitis caused by an adenoma of the duodenal papilla. At no time was the patient jaundiced, though the serum alkaline phosphatase activity remained persistently raised. The long history of upper abdominal pain, weight loss, nausea, and vomiting is typical of periampullary adenomas, but the most consistent abnormality is usually jaundice attributed to varying degrees of biliary duct obstruction.

Fourteen cases of duodenal adenomas not affecting the periampullary region have been described; these were similarly associated with upper abdominal pain, nausea, and vomiting, and symptoms persisted in only four patients after excision of the tumour.[2] This suggests that the symptoms of periampullary adenomas may to some extent be explained by partial obstruction of the duodenum by the tumour.

Post-mortem studies have shown that the prevalence of periampullary adenomas in the elderly may be as high as 4%,[3] of which most remain undetected clinically. Symptomatic cases tend to occur in middle to late life with no sex predilection.[2] Barium studies and cholangiography, though informative, rarely provide a clear diagnosis. Without doubt the best diagnostic tool is endoscopic retrograde cholangiopancreatography, which affords an opportunity to take multiple biopsy specimens. Complete excision is recommended since the condition is premalignant.[2 4]

[1] George P. Disorders of the extrahepatic bile ducts. *Clin Gastroenterol* 1973;**2**:127-46.

[2] Charles RN, Kelley ML, Campeti F. Primary duodenal tumours. *Arch Intern Med* 1963;**111**:23-33.

[3] Baggenstross AH. Major duodenal papilla. *Archives of Pathology (Chicago)* 1938;**26**:853.

[4] Seifert E. Endoscopic polypectomy: upper gastrointestinal tract. *Clin Gastroenterol* 1978;**7**:749-55.

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Pneumocystis carinii pneumonia as presenting feature of lymphoma

Pneumocystis carinii is an opportunistic pathogen that has previously been reported to occur only in patients who are immunosuppressed, either therapeutically to prevent transplant rejection or as a result of treatment of a myeloproliferative disorder. We report on a patient, who presented with pneumocystis pneumonia and had oral candidiasis as the only suggestive evidence of immune depression. Only after histological examination of necropsy specimens was lymphoma diagnosed.

Case report

A 53-year-old engineer who had been working in Saudi Arabia for eight years had deteriorated in health over the past 18 months. Shortly after