MEDICAL PRACTICE

Clinical Topics

High pressure paint gun injuries

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British Medical Journal, 1977, 2, 1333-1335**Summary**

Despite their use for the past 20 years the dangers of injuries from high pressure paint guns are not widely known. Two cases treated incorrectly through ignorance in our casualty department resulted in amputation of digits. Paint solvents are far more damaging than paint or grease injection. All cases should be treated urgently by an experienced surgeon as fairly extensive surgery may be needed.

Introduction

This report illustrates the dangers of incorrect management of high pressure paint gun injuries. Two cases initially treated incorrectly in a London teaching hospital accident department showed ignorance of the condition. This may be partly due to lack of publicity, since no paper has appeared in a broad circulation British journal since Morley's original description of the injury in 1967.¹ One case concerned an injection of paint solvent into the hand. The effects of this are far more damaging than paint or grease injection, and more extensive surgery may be needed in its initial treatment.

Case 1

A 56-year-old painter was cleaning his gun and accidentally injected paint solvent (toluene 67%, isobutanol 7%, ketones 26%) into the ulnar aspect of the middle segment of his left middle finger

at a pressure of 246 kg/cm² (3500 lb/in²). The site was marked by a tiny puncture wound.

The finger became painful, then partially anaesthetic and cyanosed; the finger, palm, and dorsum of the hand rapidly became swollen. Two hours after the injury a casualty officer made a small incision under local anaesthetic at the puncture wound and drained a small amount of paint-contaminated fluid. He then told an orthopaedic registrar, who under a general anaesthetic performed an emergency decompression of the whole middle finger, its flexor sheath, and the distal palm. Tetanus toxoid, broad-spectrum antibiotics, and steroids were given and the arm was kept raised postoperatively. The hand continued swelling, and 36 hours later the incision was extended proximally to the distal border of the flexor retinaculum. Five days later he was referred to a hand surgeon as the whole forearm, dorsum of the hand (fig 1), ring and little fingers were grossly swollen and the index and middle fingers gangrenous (fig 2). He also had symptoms of median nerve compression in the carpal tunnel.

A decompression of the index, ring, and little fingers, the carpal tunnel, and the dorsum of the hand to mid-forearm level was performed, which disclosed widespread fat necrosis, large collections of

FIG 1—Grossly swollen forearm and dorsum of hand five days after accident.

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FIG 2—Swollen ring and little fingers and gangrenous index and middle fingers.

FIG 3—Front view of palm incision showing widespread fat necrosis, large collections of sterile pus, and flecks of paint in all fingers.

FIG 4—Back view of arm incision showing widespread fat necrosis and large collections of sterile pus on dorsum of forearm.

sterile pus on the dorsum of the forearm, and flecks of paint in all the fingers and up to the proximal end of the arm incision (figs 3 and 4). The patient then rapidly recovered but the index and middle fingers were later amputated and there was a residual 90° flexion contracture of the ring finger. Sensation recovered fully.

Case 2

An 18-year-old rust proofer was hit by a jet of Ziebart anti-rust sealant* from a defective gun/hose connection causing a puncture wound on the volar aspect of the base of his right index finger. A local incision, drainage, and wound suturing was performed in the accident department. He returned eight hours later because of severe pain in the finger and was given antibiotics and analgesics. He was seen again but was only referred to the orthopaedic department six days after injury. The finger, palm, and dorsum of the hand were grossly swollen and erythematous. The other digits were normal. A wide decom-

*Ziebart XL5B Sealant: an aliphatic mineral spirits solution of the calcium salt of a petroleum wax oxidate with a small amount of carbon black for colour. Obtainable from Ziebart Great Britain Limited, Worthing, Sussex.

pression of the index finger, palm, and dorsum of the hand was performed, which released large amounts of sterile pus. He was given broad-spectrum antibiotics, silicone baths, and extensive physiotherapy. The finger survived and, though scarred, has recovered full function.

Discussion

Since high pressure paint guns were introduced in the 1950s, 47 injuries due to injection of paint,¹⁻¹⁰ lacquer,¹⁰ and materials such as vehicle anti-rust undercoating^{3 10} have been recorded in Britain. Including our case there have been 10 injuries when various types of hydrocarbon solvents have been injected (see table).^{5 7 9 11-15} Injection normally occurs from close range into a finger of the non-dominant hand at a pressure of 211-492 kg/cm² (3000-7000 lb/in²).

Number of amputations necessary after various substances had been injected by high pressure paint guns. Percentages in parentheses

Injected material	No of cases	Amputations		
		None	One digit	Two digits
Paints and lacquer	35	16 (45.7%)	19 (54.3%)	
Ziebart sealant	4	3 (75%)	1 (25%)	
Hydrocarbon solvents	10	2 (20%)	5 (50%)	3 (30%)
Total	49	21 (42.9%)	25 (51%)	3 (6.1%)

The two cases treated incorrectly in our own casualty department illustrate the need for wider publicity of this grave injury. Moreover, reports and the results described in case 1 show that paint solvent causes far more severe tissue damage than paint (see table). Researchers using cadaver hands^{11 16 17} and animals have described accurately the anatomical spread of injected substances and the response they elicit in the tissues.^{13 18} The most potent inflammatory agents are the paint vehicle soya alkyd, mineral spirits, and other hydrocarbon solvents.¹⁸ They cause rapid tissue necrosis, acute inflammatory oedema, and accumulation of sterile pus. They dissolve fat, thrombose veins, and dissolve the myelin sheaths of nerves, causing anaesthesia. In an enclosed space like the finger the injected material causes mechanical compression, which, coupled with arterial spasm and venous thrombosis, rapidly results in ischaemia and gangrene. Injecting local anaesthetic potentiates these effects and is absolutely contraindicated.

The spread of injected material depends on the injection pressure, the tissues it strikes, and its viscosity. Paint solvents have a lower viscosity than paint and therefore spread much further. Experimental work and clinical reports do not note any cases of paint spreading proximal to the flexor retinaculum and in only a few cases does sufficient paint reach the dorsum of the hand to necessitate decompression. In the 10 cases of solvent injury swelling of the dorsum of the hand was a pronounced feature in seven, and anaesthesia in the hand of median and digital nerve distribution occurred often.

The laboratory findings, especially the effects of solvents on fat, blood vessels, and nerves have been confirmed clinically in our own case and in others that have been accurately reported.^{9 12 15} In case 1 it was noted at each decompression that arterial bleeding was free and unobstructed, but the hand and fingers remained cyanosed suggesting that extensive venous thrombosis was the cause of the cyanosis and ischaemia.^{9 12 15} Gangrenous digits were amputated in eight of the 10 patients with solvent injuries, and in three patients the injured plus an adjacent digit required amputation (see table). In the 39 recorded cases of injury with paint and anti-rust

materials only 20 needed amputations, and no patient lost more than one finger. The clinical cases confirm the experimental findings that paint and anti-rust sealant are not as tissue destructive as hydrocarbon solvents.

This report illustrates the dangers of delaying correct treatment. Local incisions in casualty departments are futile and merely waste precious time. Injection of local anaesthetics into digits increases tissue tension and worsens ischaemia. Although case 1 was decompressed early, the extent of decompression was inadequate. Low viscosity fluids spread into adjacent fingers, the palm, and thence to the dorsum of the hand and up the forearm. If the fluid is highly inflammatory the initial decompression should include adjacent fingers to the one injured (except the thumb, which is usually spared), the palm, the carpal tunnel, and the dorsum of the hand.

The principles of treatment for this type of injury are: (1) urgent referral to an experienced surgeon; (2) decompression of the digit and palm for paint injuries and wider decompression for solvent injuries under general anaesthesia or brachial block with local anaesthetic. A tourniquet should be applied after raising the arm for five minutes and kept on for as short a period as possible. An Esmarch's bandage should not be used as the pressure it exerts disseminates the foreign material further; (3) broad-spectrum antibiotics to prevent secondary infection of devitalised tissue—fortunately most solvents are bactericidal; (4) tetanus toxoid cover; (5) high doses of steroids, which may help in reducing the inflammatory response¹⁸; (6) analgesics; (7) very frequent postoperative observation of the hand, so that further decompression may be performed if

necessary; and (8) early intensive rehabilitation to prevent stiffness in the digits.

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Malaria in Nottingham children

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Summary

In five and a half years 16 children were admitted to hospital in Nottingham suffering from malaria. Thirteen cases were caused by *Plasmodium vivax* and two by *P falciparum*, and in one the type was not identified. All were children of immigrant parents, predominantly from Pakistan, although most were born in England but had been visiting Asia. Three children were ill on or soon after arrival, but in the others the onset of symptoms was delayed by up to nine months. All made an uneventful recovery, although two children with *P vivax* infections had a further relapse after chloroquine treatment only.

The question "Where have you been?" should be put to all immigrant parents whose children have a febrile illness. Agencies who provide travel for Asian immigrant families returning home for a visit should be encouraged to provide instructions about malaria prophylaxis.

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Introduction

In the past ten years malaria has been increasingly recognised as a hazard of international travel.¹⁻⁴ The World Health Organisation has recently prepared an up-to-date guide⁵ that gives information to travellers on the risks of malaria in various countries and advises on preventive measures, including chemoprophylaxis. Several series of imported malaria in Britain have been reported,⁶⁻⁸ all emphasising the importance of a high index of suspicion of the diagnosis in febrile patients who have recently returned from endemic areas, particularly Africa and Asia. There has, however, been no report of imported malaria in children in Britain. To show that early diagnosis and effective prophylaxis are equally important in children, I have reviewed all the recent cases of malaria in Nottingham children.

Clinical features

From January 1972 to August 1977 16 children (11 boys and five girls) were admitted to either Nottingham Children's Hospital or Nottingham City Hospital suffering from malaria. Their ages ranged from 11 months to 10 years—eight children were below school age. Thirteen children had benign tertian malaria (*P vivax*), two had malignant tertian malaria (*P falciparum*), and in one child the type was not identified.

All the children had immigrant parents, who originated from Pakistan (nine), India (five), Nigeria (one), and Ethiopia (one), although 10 children had been born in Britain. The malaria had been acquired in Pakistan (nine), India (five), and Nigeria (two). Both *P falciparum* infections had been acquired in Nigeria. The children