Nail Damage Associated with Handling of Paraquat and Diquat

PETER D. SAMMAN,* M.D., F.R.C.P.; E. N. M. JOHNSTON,† M.B., B.CHIR., M.R.C.P.

Brit. med. J., 1969, 1, 818-819

Summary: Damage and discoloration of the fingernails probably caused by contamination with concentrated solutions of paraquat and diquat are described in three patients. The cause of this damage is unknown, but is probably a local action.

Introduction

Paraquat and diquat are dipyrilidium compounds used as weed-killers. They have the great advantage of being non-persistent. They kill all foliage with which they come in contact but are quickly inactivated on contact with the soil. They are widely used in this country and overseas. For home use paraquat is put up in granular form under the name of Weedol, but for commercial use they are available as concentrated solutions under the names of Reglone (diquat), Gramoxone (paraquat), and Preeglone Extra, and require dilution before use. Preeglone Extra contains both chemicals. The makers stress the need for care in handling the concentrated liquids, and recommend the wearing of face shields and rubber gloves when doing so. Cases of fatal poisoning from accidental ingestion (Bullivant, 1966; Campbell, 1968; and others) and after subcutaneous injection (Almog and Tal, 1967) have been reported, as also have eye injuries (Cant and Lewis, 1968), epistaxis (*Brit. med. J.*, 1967), and irritation of the skin (Swan, 1967). The pathological effects of ingestion and skin contamination in experimental animals have also been reported in some detail (Clark *et al.*, 1966). Damage to the nails has been mentioned (Barnes, 1967) but has received scant attention so far.

The following three case reports describe extensive nail damage almost certainly due to the chemicals.

Case 1

A man aged 46 was referred on 12 January 1967 by Dr. W. H. Gray on account of a white discoloration of his finger-nails, which was suspected to be due to the use of a spray in his work in the previous September. The patient had been a forester all his life. He had a few days' course of instruction in the use of a weed-killer in August 1966. He continued in this new work for two months.

The weed-killer Gramoxone was sprayed on to the ground around young fir trees which were protected from the spray by a cone of polyethylene. One pint (470 ml.) of Gramoxone was poured by the patient into a drum, to which he added 5 gallons (22.7 litres) of water. He used a milk bottle to measure the pint of neat weed-killer. Protective rubber gloves, leggings, and a mask were supplied, but were somewhat irregularly used at first, as the need for special care in the use of the weed-killer was not fully appreciated. He thought that neat weed-killer may well have come into contact with his fingers. At the end of September, while he was still on spraying work, he noticed a white discoloration at the proximal part of seven finger-nails, and this whiteness was present as a transverse band in the middle of each affected finger-nail when he attended hospital in January 1967 (Fig. 1). All four right finger-nails and the left middle, ring, and little nails were involved. Apart from the discoloration, the hands and fingers

showed only the mild roughness which had been present on his hands and fingers for years. The toe-nails were not affected. By the spring of 1967 the nails had returned to normal and have remained so.

FIG. 1.—Case 1. White patches on nails.

Case 2

A man aged 75 was referred by Dr. I. A. B. Ross on 13 August 1968 complaining of nail damage. He was a farmer who had been using Preeglone Extra for a number of years without trouble. He admitted that he took no special precautions when diluting the concentrated solution and often was splashed with it.

Several finger-nails were damaged to a greater or less extent. On the right hand the middle and index fingers showed subacute paronychia, and the nail of the ring finger was partly destroyed from the base. On the left hand the index nail was almost completely lost but new nail was forming. The middle nail showed partial loss from the base. Two weeks later (3 September) the nail of the right middle finger had been shed but showed some regrowth. The nails of the right index and ring fingers were discoloured and partly broken, and there was some haemorrhage below the nails. The left thumb-nail had a depression across it (Beau's line). Three weeks after this (24 September) the nails of the right index, middle, and ring fingers had all been shed but the beds looked healthy. On his final visit one month later (22 October) all nails were regrowing satisfactorily. The toe-nails remained unaffected throughout.

Case 3

A man aged 50 was referred by Dr. K. M. Tomlinson on 29 November 1968 on account of nail deformities. Previous examinations for fungus and monilia had been negative. The patient was a market gardener who used Preeglone Extra and diluted the concentrated solution himself. He said he was unable to wear rubber gloves because he developed a rash between his fingers when doing so. He poured the liquid with his right hand and admitted that the left got splashed at times.

He first noticed trouble with his nails in February 1968, when there was discoloration at the base of the nail of the left ring finger. This progressed and infection developed in the nail-fold. The discoloration extended to the whole nail-plate, and loosening started at the base of the nail. The nail was shed in September or October, and had not regrown. The nail of the left little finger became discoloured at the base, a yellow colour, and rather different from the ring finger two weeks before attending.

Examination showed some damage to all nails of the left hand. The nail of the ring finger was completely lost (Fig. 2) with no sign of regrowth, but the bed looked healthy. The thumb-nail showed slight loss of nail surface; on the nail of the middle finger there was slight discoloration at the base, and it was soft to the

* Physician for Skin Diseases, Westminster Hospital and St. John's Hospital for Diseases of the Skin, London; Consultant Dermatologist, Orpington and Sevenoaks Hospitals.

† Consultant Dermatologist, Nottingham and Mansfield Hospital Groups.

touch in this area. The nail of the little finger was discoloured at the base, and was starting to separate (Fig. 3). The nails on the right hand and the toe-nails were unaffected.

FIG. 2

FIG. 3

FIG. 2.—Case 3. Total loss of nail of ring finger. FIG. 3.—Case 3. Discoloration and early loosening at base of nail of little finger.

Comment

All three patients had frequent exposures to the concentrated chemicals, and had not taken special precautions to prevent contamination of the skin. The cause of the nail damage is

unknown, but it seems probable that the chemical reaches the nail matrix by entering the nail-fold and stimulates infection and also interferes with formation of the nail from the matrix. The damage is almost certainly due to local effects and is not the result of ingestion, because of the asymmetry of the lesions and the fact that the toenails are unaffected. The curious colour change and the softening of the nail at the base are rather characteristic, and should be a guide to the possible cause when seen. Infection seems to play an important part when the nail is actually lost. It should be stressed that it is the concentrated chemical which causes the damage.

Our thanks are due to the photographic department of the General Hospital, Nottingham, for Fig. 1, and to the photographic department of Westminster Hospital for Figs. 2 and 3. We also thank the doctors who referred the patients, and Dr. A. A. B. Swan for helpful criticism.

REFERENCES

- Almog, C., and Tal, E. (1967). *Brit. med. J.*, **3**, 721.
 Barnes, J. (1967). In *Third Symposium on Advanced Medicine*, p. 230, edited by A. M. Dawson. London.
Brit. med. J., 1967, **3**, 690.
 Bullivant, C. M. (1966). *Brit. med. J.*, **1**, 1272.
 Campbell, S. (1968). *Lancet*, **1**, 144.
 Cant, J. S., and Lewis, D. R. H. (1968). *Brit. med. J.*, **2**, 224.
 Clark, D. G., McElligott, T. F., and Hurst, E. W. (1966). *Brit. J. Indust. Med.*, **23**, 126.
 Swan, A. A. B. (1967). *Brit. med. J.*, **4**, 551.

Preliminary Communications

Blood Angiotensin II Levels of Normal and Hypertensive Subjects

Brit. med. J., 1969, **1**, 819–821

Summary: A specific radioimmunoassay for angiotensin II has shown that its normal concentration in arterial blood is 2.4 ± 1.2 (S.D.) $\mu\text{g./100 ml.}$; the venous level is consistently below this value, being usually 50–75% of it. Definite rises in blood angiotensin II levels were found in some patients with hypertension, both essential and secondary to renal disease. Extremely low levels were observed in two anephric women, and in one patient with Conn's syndrome. This radioimmunoassay offers a valuable alternative to renin bioassay in evaluation of the role of the renal pressor system in clinical disorders associated with hypertension and aldosteronism.

INTRODUCTION

Methods for the radioimmunoassay of angiotensin II in human blood have been described by Vallotton *et al.* (1967), Catt *et al.* (1967), Boyd *et al.* (1967), and Gocke *et al.* (1968). The values reported by these workers have shown some variation, though the ranges of plasma and blood concentration are similar for the latter three assays (Table I). It now seems probable that

the normal level of circulating angiotensin II is in the range 1 to 5 $\mu\text{g./100 ml.}$, a concentration range similar to that previously reported by certain of the bioassay methods used for the measurement of angiotensin II in blood (Mulrow, 1964). Radioimmunoassay of blood angiotensin II has now been performed in a much larger number of normal and hypertensive subjects in order to define more precisely the normal blood levels of the octapeptide and to assess its role in the development of hypertension.

TABLE I.—Radioimmunoassay Values for Blood Angiotensin II Concentration in Normal Human Subjects

Authors	Specimen	Angiotensin II Concentration ($\mu\text{g./100 ml.}$)	
		Range	Mean
Catt <i>et al.</i> (1967)	Arterial blood	0.5–4.7	2.1
Boyd <i>et al.</i> (1967)	Venous plasma	0.8–5.6	Not given
Gocke <i>et al.</i> (1968)	Venous plasma	1.8–11.0	5.4

METHODS

The previously described method for radioimmunoassay of blood angiotensin (Catt *et al.*, 1967) was used to measure circulating angiotensin levels in normal subjects and in those suffering from various forms of hypertension and vascular disease. All blood samples were taken after subjects had rested for at least 15 minutes in the recumbent position. Minor