MEDICAL MEMORANDA

Sialadenitis after Intravenous Pyelography

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British Medical Journal, 1971, 3, 351–352

We present an account of a patient who developed iodine sialadenitis after infusion pyelography, together with estimations of total plasma iodine levels. As a control study we report the measurement of plasma iodine levels in a patient whose renal function was investigated before and after surgical treatment for ureteric obstruction producing renal failure.

Case 1

A man aged 53 was admitted to hospital on 12 April 1968 with progressive dyspnoea and weakness. Fourteen years previously he had attended hospital as an outpatient complaining of polyuria and nocturia and was found to have hypertension, anaemia, and proteinuria. Chronic glomerulonephritis was diagnosed.

On admission in 1968 he was lean and moderately anaemic, with a sinus tachycardia of 110/min and a blood pressure of 210/120. Laboratory investigations confirmed the presence of a normochromic, normocytic anaemia, with a haemoglobin of 9 g/100 ml. A 24-hour urine specimen contained 2·4 g of protein. He was shown to be in renal failure, with a blood urea of 120 mg/100 ml and a creatinine clearance of 10·8 ml/min. Protein-bound iodine at that time was 3·7 μg/100 ml. Despite the blood urea being in excess of 100 mg/100 ml excretion pyelography with 30% Urografin by the infusion technique was attempted on 19 April, during which a total dose of 36·5 g of iodine was used. The kidneys were not visualized. Four days later he complained of discomfort over the submandibular glands. He was found to have a tender enlargement of both submandibular salivary glands but no enlargement of other salivary glands. The enlargement of the salivary glands subsided without treatment within three days, and on 27 April they were neither palpable nor tender.

This was recognized as a probable iodide sialadenitis, and arrangements were made to estimate total plasma iodide. The results of these estimations are as follows: April 23rd, 10,000 μg/100 ml; 25th, 21,100 μg/100 ml; 26th, 11,900 μg/100 ml; and 27th, 1,630 μg/100 ml. From these figures there seems to be a good correlation between the clinical course of this complication and the plasma iodide levels, as shown for the first time by Harden (1968). He reported that the salivary gland enlargement occurs at a plasma concentration of 10,000 μg/100 ml or above and subsides again when the plasma level drops below this.

Case 2—Control Study

A 53-year-old woman was admitted to the gynaecological ward with a history of acute vaginal blood loss. Her general condition was fair, pulse 108/ min, sinus ryhthm, and blood pressure 190/110. After vaginal examination and subsequent biopsy carcinoma of the cervix was diagnosed.

At that time her blood urea was 202 mg/100 ml and potassium 8·1 mEq/1. She was treated with a low-protein diet, insulin and glucose, and sodium polystyrene sulphonate retention enemas.

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On this regimen her blood urea gradually rose to 330 mg/100 ml but the potassium level remained between 4 and 6 mEq/1. Urinary output varied from 350 to 500 ml/day. A diagnosis of renal failure due to ureteric obstruction arising from carcinoma of the cervix was made. As retrograde pyelography seemed unlikely to prove successful, on 27 August 1970 infusion intravenous pyelography was attempted, the same material (30% Urografin) and technique as described in Case 1 being used. Renal outlines were visualized but no pyelograms were obtained. Blood samples were collected for total plasma iodide estimations. On 29 August a bilateral ureterostomy was performed, which was followed by a brisk diuresis of 7,550 ml in 24 hours.

The blood urea returned to normal within five days. Daily detailed examination of the salivary glands did not show any evidence of tenderness or enlargement. There was no evidence of any other side effects.

*Levels of Total Plasma Iodides in Case 2**

Date	Total Plasma Inorganic Iodide (μg/100 ml)	Blood Urea (mg/100 ml)	Urine Output (ml/24 hours)
	Before Ureteric Obstruction was Relieved		
28/8/70	196,000	333	340
28/8/70	192,000		
	After Ureteric Obstruction was Relieved		
29/8/70	148,000	291	7,550
30/8/70	10,600		4,310
1/9/70	442	26	2,620
	After Renal Function had Returned to Normal		
7/9/70	58	28	1,990
9/9/70	50		2,000
10/9/70	30		1,310

*Dr. R. McG. Harden reports that these estimations are accurate to within 1 μg/100 ml.

On 7 September, when it was thought that renal function had returned to normal, as judged by blood urea, electrolytes, and urinary output, a further infusion intravenous pyelogram was performed using the same technique and material. On this occasion good contrast pyelography was achieved, showing some residual dilatation of the renal pelvis.

Levels of total plasma iodides and their relation to the renal function before obstruction was relieved, immediately after relief of obstruction, and again later when renal function had returned to normal are shown in the Table.

Comment

The salivary glands trap substantial amounts of iodide but they do not exert any important overall effect on iodine metabolism. There is evidence to suggest that the site of concentration is the salivary ducts (Mason *et al.*, 1966). The salivary accumulation of iodine was found to be independent of thyroid function (Fellinger *et al.*, 1956; Gabrielsen and Kretchmar, 1956).

Iodine sialadenitis, painful swelling of the salivary glands, during iodide administration has been reported, but the pathogenesis is uncertain. This complication has been described previously after the use of iodides in diagnostic radiography but seems to be rare, as found by Tucker and Di Bagno (1956). Sussman and Miller (1956) reported two cases of salivary gland enlargement after intravenous iodide contrast media but did not give plasma iodide levels.

The investigation of the patient with renal failure due to ureteric obstruction (Case 2) allowed us to observe plasma iodide levels during the phase of obstructive renal failure and after its relief, the patient in this instant acting as her own control. The information obtained shows two points of interest—(1) the fact that despite the extremely high levels of plasma iodide sialadenitis or any other side effects were not manifest, and (2) the sharp contrast in plasma iodide levels produced by infusion pyelography during renal failure and after recovery of normal renal function.

In conclusion, therefore, it is assumed that the complication of iodide sialadenitis is an individual response mechanism and not related to plasma iodide levels in general. In the two reports of this complication in which iodide levels are available it does appear that those individuals who develop this rare manifestation of iodide toxicity do so when the level exceeds 11,000 µg/100 ml.

The total plasma iodide estimations were kindly carried out by Dr. R. McG. Harden, of the Gardiner Institute, Western Infirmary, Glasgow.

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Acute Onychia and Onycholysis due to an Enzyme Detergent

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British Medical Journal, 1971, **3**, 352

It is recognized that nail damage may occur in irritant contact dermatitis. Recently some cases of contact dermatitis due to enzyme detergents have been reported (Ducksbury and Dave, 1970; Jensen, 1970). We report a case of acute nail damage which we attribute to enzyme detergent.

Case Report

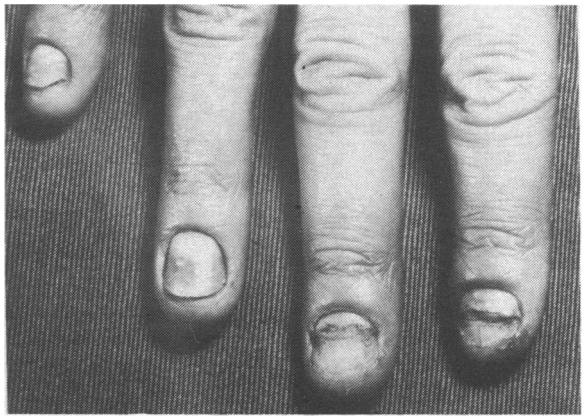
The patient, a 26-year-old housewife, had no previous history of skin disease or atopy. She first used enzyme detergents intermittently from a few small packets during the summer of 1970. She then used an enzyme detergent for her daily laundering for a month without wearing gloves. Her hands were exposed to the detergent solution for about an hour each day. No other detergents, bleaches, or disinfectants were used at the time. After two weeks she noticed dryness and scaling on the backs of her hands, together with soreness of the nail-folds and flaking finger-tips. The nails then became softened, discoloured, and raised. Next, on some of the fingers the distal half of the nails were shed. After the nails had become deformed and painful she applied false nails with an adhesive for half a day, removing them only because the soreness increased.

When seen on 30 November the backs of her hands showed areas of rough dry scaling, and acute paronychia was present. There was partial onycholysis of all finger-nails, with thickening, a brownish yellow discoloration, and elevation of the remaining distal third of the nail plate. The distal half of the nail had been shed on the index and middle fingers of both hands (see Fig.). The toe-nails were normal. There was no tinea pedis.

Patch tests (using Altest strips under Transpore tape) with 1% and 2% aqueous solutions of the detergent and with the artificial nail adhesive were applied for 48 hours. These were negative. Within a month of stopping the use of the detergent the dermatitis on the back of the hands had healed, the paronychia had settled, and the nails had started to grow normally again. A steroid ointment was used topically.

Comment

An outbreak of contact dermatitis due to enzyme detergents was reported by Ducksbury and Dave (1970). They considered this to be a primary irritant eruption. In all their 12 cases the reaction was severe, and in half of their cases the dermatitis developed within the first few days of exposure. Jensen (1970) also reported severe dermatitis with such detergents, but he found that patch tests to a 0·5% solution of detergent were positive in 4 out of 12 cases. Neither of these reports describe any associated abnormalities of the nails, but it would seem that the damaged skin is liable to digestion by proteolytic enzymes such as trypsin and chymotrypsin (Baden, 1970). In the present case the nail changes were severe in comparison with the relatively mild dermatitis on the hands.



Appearance of the finger-nails after use of an enzyme detergent.

While allergic contact dermatitis due to nail hardeners has been reported (March, 1966; Donsky, 1967) these were not used by our patient. Nail changes have also followed the use of artificial nails (Frumess *et al.*, 1952), but in the present case these were applied for only half a day after the onychia had developed. Nail damage caused by weed-killers (paraquat and diquat) has been described (Samman and Johnston, 1969; Clark and Hurst, 1970) but this patient had not handled such chemicals. Acute onycholysis and recovery is not a feature of an untreated fungal onychia or candidiasis. We therefore conclude that this patient's acute onychia and onycholysis resulted from her daily use of enzyme detergent.

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