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Plantar Warts

During the past 40 years there has been a remarkable increase in the number of patients with warts referred to dermatological clinics, and this applies to the plantar types as well as others. We now know much about plantar warts except for one important item: there is no specific method for curing them easily and quickly.

The wart virus can be readily identified under the ultramiscroscope and is killed by intense heat and cold, but no known virucidal drugs appears to have much effect on it. In spite of much endeavour the wart virus has so far proved impossible to grow in any culture system other than by implantation in the living human skin. The usual incubation period between implantation of the virus and development of the wart is from 1 to 6 months, though longer intervals have been reported. Whether a wart develops depends on the immunological state of the patient. Protective IgG antibodies to wart virus have been detected by M. M. Ogilvie,[1] not only in persons with a past history of warts but also in those with no remembered wart infection. An attack of plantar warts does not give permanent protection against further infections.

Undoubtedly the infection spreads mainly in swimming pools and probably in changing-rooms used for athletic exercises, but so far there is no proof that barefoot gymnastics and dancing have been responsible. In fact there is some evidence to the contrary.[2 3] The virus is implanted after injury to the plantar epidermis, and, as M. H. Bunney has pointed out,[4] swimming pools abound in rough surfaces on the surrounds and diving boards, which remove the water-softened, horny hyperkeratosis of warts together with virus particles. Individual susceptibility plays a part also, for some people appear to have excellent immunity against the virus, while others do not. The patient on immunosuppressive and steroid drugs may be at particular risk.

The diagnosis of plantar warts is usually simple, but some other dermatoses should be considered. They include corns; foreign-body reactions; inclusion cysts; molluscum contagiosum; various tumours such as malignant melanoma, neuromata, and haemangiomata; arsenical keratoses; linear warty naevi; and keratosis punctata; while the pitting in severe hyperhidrosis may simulate multiple warts. When the horny layer is gently shaved away with a scalpel blade, a wart can be diagnosed by the presence of tow-like papillary projections, which with further shaving show pinpoint bleeding of the tips of the papillary processes. Corns are painful keratotic masses, extremely tender on lateral pressure, and when they are pared down the smooth keratin core is disclosed, differing from the opaque tissue of the wart with its oozing capillaries. They usually occur beneath the metatarsal heads and are primarily an orthopaedic problem.

The number of plantar warts varies from solitary to dozens, and they often tend to occur in areas most liable to trauma. The most tedious type is the mosaic wart.[5] These small, usually painless growths are often aggregated in plaques of a mosaic pattern, so that the disease is not diagnosed. They are the most resistant kind known, but are caused by the same virus as other warts. Pain is a common symptom of the ordinary plantar wart, especially if it is at a site subject to pressure. When warts which have been relatively painless become suddenly exquisitely tender, this is often an indication that haemorrhage into them has occurred and that they are undergoing spontaneous cure.

As spontaneous cure occurs in approximately 20%,[6] it is advisable to avoid the use of treatments which can cause undesirable side effects. Thus radiotherapy, once so widely used, has been abandoned by almost all dermatologists, partly because of the possibility of radiodermatitis years later and partly because in general the less radiation a person receives the better. Smallpox vaccination for the treatment of verrucae is inadvisable, as untoward reactions may occur. Podophyllin resin, in various strengths and in various vehicles, has proved useful, but it must be borne in mind that intense reactions may occur, leading to large sterile abscesses. Likewise, freezing with carbon dioxide snow or liquid nitrogen still has an important place in the treatment of small warts in young children, but for large deep-seated lesions prolonged freezing is required, resulting in an intensely painful reaction.

In the routine treatment of plantar warts it is best to start with simple measures such as strapping with Elastoplast.[7] The area to be treated should be tightly bound with two layers of Elastoplast round the foot, and this should be renewed once weekly. Simple paints and lotions may be highly effective. Solution of formaldehyde 3%, applied either by soaking the affected area in a doll's saucer every night for ten minutes or by applying it just before bedtime on a small piece of cotton-wool kept in place with strapping, has often achieved cures, though I. Anderson and E. Shirreffs,[8] from the results of a controlled trial, cast some doubt on its efficacy.

Possibly the most useful paint is the old prescription: salicylic acid 1 part; lactic acid 1 part; flexible collodion 4

parts. It is applied every night after the surface of the wart has been gently removed with an emery board and the foot soaked in hot water for about five minutes. Four drops of this paint are applied to the wart, and when it is dry the area is covered with Elastoplast.[9] Other preparations such as alkyl dimethyl benzyl ammonium halide dibromide can be used in the same way.

For patients whose warts are intensely painful or have resisted simpler methods removal with a blunt curette is often indicated. Local anaesthesia is normally employed, and the pain of the injection can be mitigated by the preliminary use of an ethyl chloride spray. Any bleeding can be controlled by electrocautery or fulguration. Rarely, in patients with a great number of warts, general anaesthesia is indicated. Surgical excision is unwise.

The prevention of these warts is not an easy matter. It is notable that the incidence is low where a swimming pool is used by only one group of people and where supervision and inspection are adequate. Open-air pools and salt-water baths seem to lead to a lower incidence of infection. Protective footwear might greatly reduce the incidence of plantar warts if introduced widely.[10]

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Lithium-induced Diabetes Insipidus

There appears to be little doubt that administration of lithium carbonate is of benefit in the treatment of manic-depressive disease.[1-3] This is particularly true for the manic features of the condition, but long-term therapy also reduces the incidence of relapses of both the manic and depressive phases. In severe depressive illness it does not appear to be so effective. Since increasing numbers of patients are taking lithium for prophylaxis, consideration must be given to its possible toxic effects.

Apart from symptoms such as nausea, diarrhoea, and tremor, which may be transient, overdosage can lead to sleepiness, vertigo, slurred speech, or muscle hypotonia, and can lead on, in severe intoxication, to episodes of hyperextension of the limbs, epileptiform attacks, and coma. These toxic effects are related to the serum concentration of lithium and are more likely to occur if there is coexisting renal disease or if the patient is depleted of sodium. It is wise to check the blood level regularly to ensure that it remains in the effective and safe range of 0·7 to 1·3 mEq/l.[2]

Long-term complications include the occasional development of a diffuse goitre, but the patients usually remain euthyroid and the effect is reversible. The precise mechanism of the goitre is obscure, but there is some evidence that it results from impaired synthesis of hormone in the gland.[4-6] While stimulation of thyroidal adenyl cyclase by thyroid-stimulating hormone (TSH) is inhibited by lithium, this is not the full explanation of the effects, since the action of cyclic adenosine monophosphate (AMP) on the thyroid is also blocked.[7 8] The goitre seldom needs treatment, but if it is large, or if hypothyroidism occurs, there is a good response to thyroxine.

Polyuria and polydipsia have been reported in man and experimental animals.[9 10] Rats given toxic doses of lithium passed dilute urine, and the kidneys did not succeed in concentrating the urine despite the administration of vasopressin. Later the animals died in oliguric renal failure. These effects were more easily produced if the animals were salt-deficient. These observations suggested that lithium may interfere with the action of vasopressin on the nephron, resulting in an acquired form of nephrogenic diabetes insipidus such as may also be associated with hypercalcaemia and hypokalaemia. Now it has become clear that some patients may develop a disturbing degree of polyuria and polydipsia even when treated with conventional doses of lithium, and it seems probable that many others have these symptoms in a milder degree.[11-13] B. M. Angrist and colleagues[11] and T. A. Ramsay and colleagues[13] together have reported on five patients who developed persistent polyuria beginning between two weeks and seven months after starting long-term lithium carbonate therapy in conventional doses and who had safe plasma lithium concentrations. No patient had clinical or conventional biochemical evidence of pre-existing renal disease or electrolyte imbalance. During water deprivation tests or administration of a hypertonic saline stimulus these patients failed to concentrate their urine or reduce the flow normally despite haemoconcentration and loss of weight, indicating a diagnosis of diabetes insipidus. Since the urine did not become concentrated after administration of vasopressin either, the authors reasonably concluded that the patients had acquired nephrogenic diabetes insipidus, and the condition appears to be reversible when the drug is stopped.

The mechanism of this interference with the action of vasopressin is not known but may be similar to that produced experimentally by increased concentrations of magnesium, manganese,[14] and caesium.[15] There is evidence that in some of these instances the toxic agent inhibits the activation of cyclic AMP by vasopressin,[14] and this is probably true for lithium. Whatever the mechanism at work, it is important to remember that unlike other forms of nephrogenic diabetes insipidus it would be most unwise to treat the patient by salt depletion and diuretics, since salt depletion greatly enhances the toxicity of lithium.

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