the pigmentation was fading, while the nail bands were as before, but of a paler shade of brown. His weight had increased by 10 lb. (4.5 kg.).

COMMENT

The diagnosis of Addison's disease, despite normal salt-tasting, was confirmed by inability to excrete a water load, reduced urinary steroids with lack of response to stimulation with corticotrophin, and by a dramatic improvement on oral cortisone. There is nothing specific about the pigmentary abnormalities of Addison's disease (Addison, 1868), but there was no evidence of liver or kidney disease, haemachromatosis, anaemia, or malabsorption, which may all give similar pigmentary changes. No tuberculosis being found, the suprarenal failure was presumably due to a primary atrophy (Prunty, 1964). Lerner *et al.* (1954) consider that the mechanism of pigmentation in Addison's disease is due to increased release of melanocyte-stimulating hormone (M.S.H.) from the pituitary as a result of decrease in adrenal cortical function, and that this is a distinct hormone whose only function is stimulation of melanin production.

Lerner and McGuire (1961) showed that both α and β M.S.H. produced darkening of human skin, and they thought

Pigmentation of nail.

that the darkening effect of corticotrophin itself was not established, as it might contain some M.S.H. Sulman (1956) found increased chromatophorotropic activity in the blood of all patients with Addison's disease. The pigmentation of the nails was a most striking feature in this case (see Fig.), and there was no history of ingestion of drugs that cause various colour changes in nails, such as phenothiazine (Satanove, 1965), chloroquine (Tuffanelli *et al.*, 1963), amodiaquine (Maguire, 1962), or phenolphthalein, silver, or arsenic (Samman, 1965).

Pillsbury *et al.* (1957) state that the nail usually lacks melanin pigment, but the occasional development of transverse bands

following the administration of a large quantity of M.S.H. or x-ray shows that non-functioning melanocytes may be present and released from inhibition by the appropriate stimulus. They agree with Keeley (1962) that longitudinal bands of melanin are common in negroes, but consider them so uncommon in white persons that their sudden appearance strongly suggests the development of a junctional naevus in the matrix. Because of the risk of a malignant melanoma arising in a lesion at this site they consider amputation of the distal phalanx to be the soundest course. This view is shared by Harvey (1960) and Gibson *et al.* (1957). Longitudinal pigment in nails has been reported in Peutz-Jeghers syndrome by Kyle (1961), and as familial congenital naevi by Caron (1962), but in Addison's disease is not mentioned by Spence (1953), and Wiener (1947) says the white colour of the nails is a most striking feature. Soffer (1956) notes pigmentation as infrequent and mentions only one patient with longitudinal streaks.

We consider that the pigmented streaks in our patient most probably arise by stimulation of resting melanocytes in the nail bed, though stimulation of junctional naevi is a possibility. This physical sign is infrequent, but the case is of value as the nail pigment may be noted before generalized colour change or signs of Addison's disease are apparent and so be considered as a subungual melanoma and treated by radical surgery as potentially malignant.

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C. F. ALLENBY, M.B., M.R.C.P.,

Senior Registrar in Dermatology, United Oxford Hospitals.

P. H. SNELL, B.M.,

Senior House Officer in Medicine, United Oxford Hospitals.

REFERENCES

- Addison, T. (1868). *Collection of the Published Writings*, edited by Drs. Wilks and Dady, p. 211. New Sydenham Society, London.
 Caron, G. A. (1962). *Lancet*, **1**, 508.
 Gibson, S. H., Montgomery, H., Woolner, L. B., and Brunsting, L. A. (1957). *J. invest. Derm.*, **29**, 119.
 Harvey, K. M. (1960). *Lancet*, **2**, 848.
 Keeley, K. J. (1962). *Ibid.*, **1**, 866.
 Kyle, J. (1961). *Scot. med. J.*, **6**, 361.
 Lerner, A. B., Fitzpatrick, T. B., and Shizume, K. (1954). *Amer. J. Med.*, **17**, 105.
 — and McGuire, J. S. (1961). *Nature (Lond.)*, **189**, 176.
 Maguire, A. (1962). *Lancet*, **1**, 667.
 Pillsbury, D. M., Shelley, W. B., and Kligman, A. M. (1957). *Dermatology*. Saunders, Philadelphia.
 Prunty, F. T. G. (1964). *Chemistry and Treatment of Adrenocortical Diseases*. Thomas, Springfield, Ill., U.S.A.
 Samman, P. D. (1965). *The Nails in Disease*. Heinemann, London.
 Satanove, A. (1965). *J. Amer. med. Ass.*, **191**, 263.
 Soffer, L. J. (1956). *Diseases of the Endocrine Glands*. Kimpton, London.
 Spence, A. W. (1953). *Clinical Endocrinology*. Cassell, London.
 Sulman, F. G. (1956). *J. clin. Endocr.*, **16**, 755.
 Tuffanelli, D., Abraham, R. K., and Dubois, E. I. (1963). *Arch. Derm. Syph. (Chic.)*, **88**, 419.
 Wiener, K. (1947). *Skin Manifestations of Internal Disorders*. Kimpton, London.

Infection with Adult *Toxocara canis*

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Toxocara canis and *T. cati* infections in man have latterly assumed increasing importance in view of the recently acquired knowledge that a large reservoir of infection with them exists in dogs and cats respectively in Britain and elsewhere, and that these infections are not infrequently transmitted to man

(Woodruff and Thacker, 1964). Virtually all the toxocariasis reported to date, however, has resulted from the presence of toxocaral larvae in tissues. The infection is acquired by swallowing ova passed in animals' faeces, and, though these develop into larvae in man, it is rare for these larvae to complete their cycle of development in the human host and for infection with adult *T. canis* or *T. cati* to result. The following case is believed to be only the second in which *T. canis* adults have been demonstrated in man.

CASE REPORT

A 16-month-old female child, born of English parents in Southampton, was seen as an outpatient in the Medical Unit, Hospital for Tropical Diseases, in August 1965 with a history of having passed a worm in the stool. A month previously an immature female worm (see Fig. 1) had been passed, and had been suspected of being a *T. canis*. A second worm had also been passed a few days prior to the child's attendance at the Hospital for Tropical Diseases.

There had been no residence overseas, but the mother had observed the child eating soil in the garden. The family possessed a cat, and a relative owned a dog with which the child had been in contact. Examination of the faeces of each revealed no toxocara ova.

The child when seen was well, and there was no history of cough or fever. No clinically detectable abnormalities were present, and the optic fundi were clear. In view of the history of passing worms the child was admitted to hospital; haemoglobin was found to be 12.3 g./100 ml.; the white blood cells numbered 13,000/c.mm. (neutrophils 32%, lymphocytes 60%, monocytes 5%, eosinophils 3%); the erythrocyte sedimentation rate was 5 mm./hr. (Westergren). Six stool examinations by centrifugal concentration revealed no helminthic ova or protozoan cysts. The chest x-ray picture was normal. Toxocara skin test was positive. The toxocara fluorescent antibody test was positive up to a dilution of 1/2,560.

The child remained well, and after a week in hospital was given 2.5 g. of Pripsen (1 g. piperazine phosphate and 7 mg. standardized senna) as a single dose, and produced a worm which on microscopical examination was identified as an adult male *T. canis* (Fig. 1). Its length was 9.5 cm., its breadth 2.3 mm. With the object of eradicating any toxocara larvae which might have been present in the tissues, 100 mg. of diethylcarbamazine (Banocide) was given daily for 21 days.

The Parasites.—The anterior extremity of both the adult male and the immature female worms are bent ventrally and have characteristic lips, without denticular ridges (see Fig. 2). Cervical

FIG. 1.—Immature and mature *T. canis* passed by the patient (lengths 4.5 and 9.5 cm. respectively).

FIG. 2.—Anterior extremity of the mature *T. canis* passed by the patient. ($\times 150$.)

alae and a distinct posterior muscular ventriculus to the oesophagus are present. The male "tail" is bent backwards and the posterior extremity is probular (Fig. 3); the coils of the testis reach the anterior of the body; caudal alae are absent. There is a group of five papillae on each side of the conical tail, and on each side a double subventral papilla behind the anal opening and in front of the tail, with a row of over 20 pre-anal papillae. Spicules are present.

FIG. 3.—Posterior extremity of the adult male *T. canis* passed by the patient. ($\times 150$.)

DISCUSSION

Infection with adults of the genus *Toxocara* have been thought to be very infrequent in the past, but the small number of cases reported may have resulted from their being mistaken for immature *Ascaris lumbricoides*, the common human roundworm (Leiper, 1907; Mendheim *et al.*, 1952). The exact number of cases of infection in man with adult *T. cati* is not clear from the literature, but it seems that only one with adult *T. canis* has previously been reported. No doubt with increasing awareness of the condition more cases will come to light, but a close examination of parasites is needed to ascertain their exact taxonomic status.

Beisele (1911) reported a human *T. cati* infection in Germany, and Swartzwelder (1941) reported another from the Southern United States in a child aged 7 years, who after a spell of coughing vomited two male worms. Mendheim *et al.* (1952), surveying the literature, found 18 records of *T. cati* infections in man and one of *T. canis*. Some of the reported human infections as tabulated by Mendheim (1952) are open to question, and may be instances of spurious parasitism. The related ascarid *Toxascaris leonina*, which occurs in both Canidae and Felidae, was reported by Leiper (1907) from human intestine in Egypt.

B. BISSERU, M.SC., PH.D., M.B., M.R.C.P.ED., D.T.M.&H.,
D.OBST.R.C.O.G.

A. W. WOODRUFF, M.D., PH.D., F.R.C.P., F.R.C.P.ED.,
D.T.M.&H.

R. I. HUTCHINSON, M.D., D.P.H.

Department of Clinical Tropical Medicine, London School of Hygiene and Tropical Medicine, the Medical Unit, Hospital for Tropical Diseases, London, and Public Health Laboratory, General Hospital, Southampton.

REFERENCES

- Beisele, J. (1911). *Munch. med. Wschr.*, 58, 2391.
Leiper, R. T. (1907). *Brit. med. J.*, 1, 1296.
Mendheim, H., Scheid, F., and Schmidt, J. (1952). *Z. Tropenmed. Parasit.*, 3, 368.
Swartzwelder, J. C. (1941). *J. trop. Med. Hyg.*, 44, 61.
Woodruff, A. W., and Thacker, C. K. (1964). *Brit. med. J.*, 1, 1001.