capillaries and thin-walled veins. The endothelial tissue was lightly infiltrated with mononuclear cells, and in places this tissue was retracted, leaving between it and the osseous trabeculæ a space occupied by a slightly acidophilic serous substance.

We believe that the osseous cystic lesions in congenital generalised osteodystrophy are not fortuitous but form part of the clinical picture of this condition in the adult.

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REGRESSION OF BURKITT'S LYMPHOMA IN ASSOCIATION WITH MEASLES INFECTION

SIR,—There have lately been reports suggesting a beneficial effect of natural measles infection on the course of acute lymphoblastic leukæmia.[1,2] During the past few months we have observed a complete regression of histologically diagnosed Burkitt's lymphoma in an untreated African child who contracted measles shortly after diagnostic biopsy.

Case-report

An eight-year-old African boy was admitted to Mulago Hospital on Dec. 1, 1970, with a four-month history of painless right orbital swelling. Six months before admission a much smaller right orbital mass had been excised at another hospital. At that time the clinical diagnosis was lipoma, although no histological examination of the mass was performed. On Dec. 1 physical examination was remarkable only for right-sided proptosis with conjunctival œdema, loss of vision, and absence of all extraocular movements on the right side (fig. 1). A biopsy specimen of the right retro-orbital tumour taken on Dec. 3 was histologically diagnostic of Burkitt's lymphoma. No blood or blood products were administered at the time of surgery nor at any time thereafter. On Dec. 13, before institution of any form of therapy, the patient was noted to have a generalised measles exanthem. On the same day, the right orbital tumour was first noted to be regressing; over the course of the next two weeks both the exanthem and the tumour disappeared (figs. 2 and 3). Measles-antibody titres, measured by complement fixation, were absent on Dec. 16, 1/64 on Jan. 8, and 1/16 on March 5. The patient remains in complete remission four months after the measles infection, having received no antineoplastic therapy.

Immunological evaluation included intradermal testing, on Jan. 9 and April 12, with intermediate strength P.P.D., streptokinase-streptodornase, mumps antigen, and dermatophyton O. On neither occasion was a reaction to any of these antigens observed. Attempted sensitisation to topical dinitrochlorobenzene applied on Jan. 9 was proven unsuccessful at the time of challenge on Feb. 4 and April 12. Iliac-crest bone-marrow aspirations on Dec. 20 and Jan. 11 disclosed normal cellularity with 20% to 22% erythrocyte precursors. Only 7% lymphocytes was found in the first aspirate; this figure rose to 28% during the three-week period preceding the second bone-marrow aspiration.

Discussion

The ability of measles virus to suppress established cellular reactivity to tuberculin has been demonstrated both in vivo [3] and in vitro.[4] This suppression has also been observed in patients vaccinated with live, attenuated measles-virus vaccine [5]—an effect that has been applied to the treatment of allergic disorders.[6] An agent capable of suppressing cellular reactivity would be expected to affect adversely the course of Burkitt's lymphoma since a cellular immune reaction against the tumour cells has been shown to correlate [7] with a favourable prognosis. The complete

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Fig. 1—Patient on admission, Dec. 1, 1970.

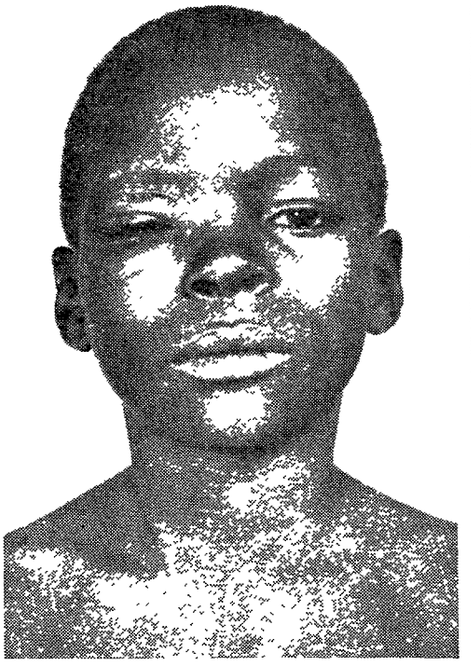


Fig. 2—Patient on Dec. 21, 1970, with resolving measles exanthem.

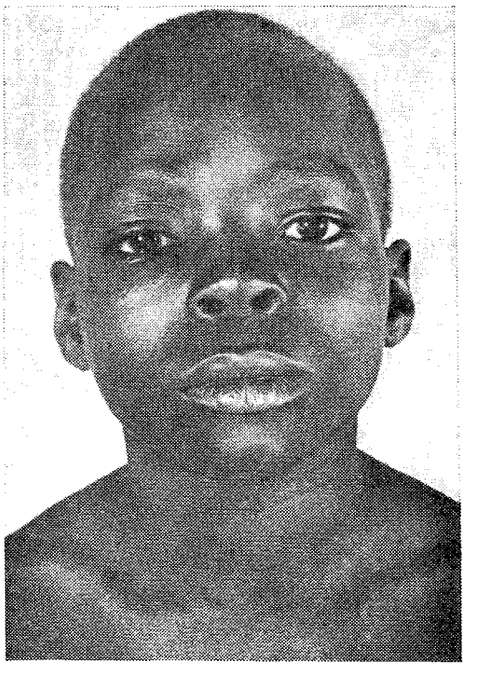


Fig. 3—Patient on Jan. 6, 1971. Tumour and measles have resolved completely.

regression of tumour concurrent with natural measles infection seen in this case was therefore unexpected. Some possible explanations are discussed briefly below.

Since spontaneous regressions of Burkitt's lymphoma have been reported,[8] it is conceivable that the chronological association with measles was fortuitous. Such regressions have, however, been attributed to an immunological reaction, and would be unlikely to occur in the presence of suppression of cellular reactivity by measles infection. Destruction of the tumour cells by measles virus is a possibility, though measles virus does not appear to lyse infected cells.[9] Another possibility is that the measles infection stimulated production of endogenous lymphocidal substances, such as adrenocorticosteroid hormones. Children with measles, however, are reported to have normal levels of these hormones.[10] Measles-induced interferon might have been responsible for the improvement; but interferon seems to protect against oncogenic viruses only when given before or at the time of infection.[11] Since measles has been shown to induce blastic transformation in vitro,[12] it might effect an antitumour response by stimulating reactivity against tumour antigen. Measles virus seems, however, to stimulate cellular immunity to itself at the expense of other antigens; non-specific stimulation would, therefore, not apply here. Conceivably an entirely different process is at work; in both Burkitt's lymphoma and acute lymphoblastic leukæmia the lymphoblasts closely resemble normal lymphocytes which have undergone blastic transformation in response to a recall antigen. Possibly, at least in an early stage of these diseases, the " tumour " cells are really non-malignant immunocytes responding to an as yet unidentified stimulus. Measles might have suppressed this reactivity. Measles suppression of reactivity rarely lasts more than eight weeks,[5] however, and our patient's remission has lasted sixteen weeks to date.

A more probable explanation is based on rejection of a measles/tumour-cell antigenic complex. Cells infected by viruses with lipoprotein coats—a group which includes the measles virus—have been shown to form filamentous microvilli containing viral antigen.[13] Wiktor et al.,[14] exploiting this finding, showed that tissue-culture cells infected with rabies virus were lysed following exposure to rabies antibody and complement. Webb and Gordon Smith [13] suggested that human neoplastic cells might be artificially infected with virus to achieve immunological lysis of the tumour cell as a byproduct of cytotoxic-antibody production against the virus. These workers were, however, quick to point out that the presence of a circulating antibody to Epstein-Barr-virus-related antigen in Burkitt-lymphoma patients has not been correlated with clinical evidence of increased tumour-cell destruction. Since cellular reactivity to autologous tumour extract is associated with a favourable prognosis in Burkitt's lymphoma,[7] Webb and Gordon Smith [13] concluded that tumour-cell lysis would more likely be due to cell-mediated than to humoral immunity. Though cell-mediated immunity is believed to be suppressed in measles, this effect may be more apparent than real: massive cellular reactivity to measles may temporarily exhaust the thymus-dependent lymphocyte pool. In the course of this reaction the measles/tumour-cell complex might be destroyed, even though there is apparent suppression of cell-mediated reactivity to non-related antigens. This explanation, of course, presupposes measles infection of the tumour cells —a hypothesis currently being investigated as part of a therapeutic trial of measles vaccination in Burkitt's lymphoma.

This work was supported by contract numbers PH 43-67-1343 and PH 43-67-67 from the National Cancer Institute, National Institutes of Health, Bethesda, Maryland.

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PARASITES AND CARCINOGENESIS

SIR,—Dr. Keller and his co-workers (April 3, p. 678) have shown that infection with *Nippostrongylus brasiliensis* may have a considerable effect on the development of transplantable tumours in rats and mice. Similar effects were noted during our own investigations on the morphological and metabolic alterations within the skeletal muscles of rats infected with *Trichinella spiralis* larvæ [1,2] in experiments on tumour formation in muscle.[3] In order to answer the question, " Does trichinellosis have any effect on the carcinogenic process ? " we observed three groups of hooded rats. In the first group of 7 animals 0·1 ml. of an aqueous suspension of powdered nickel sulphide (Ni_3S_2 100 mg. per ml.) with 0·3% carboxymethylcellulose was injected into the gastrocnemius muscles. In the second group of 6 rats the same dose of nickel sulphide was given, and methandrostenolone (metandienone) (5 mg. in 0·3% aqueous carboxymethylcellulose) was given subcutaneously every week for 12 months. In the third group, 12 animals were infected with *T. spiralis* larvæ (each rat received 3000 larvæ of the Krotoszyn strain, orally) and then, five days later, injected with nickel sulphide as above.

The results were as follows. In the first group none of the rats developed tumour up to the 13th month of investigation. In rats treated with methandrostenolone, the first palpable tumour was detected 4 months and the next 8 months after the injection of nickel sulphide. The remaining 4 animals in this group were killed after 13 months, and no evidence of carcinogenesis was found in their limbs. In the trichinellosis group no tumours were found in the 7 animals that died during the 1st month of the disease. All 5 survivors, however, developed tumours at the site of the nickel-sulphide injection within 8 to 11 months. Microscopically all the tumours in both groups were undifferentiated rhabdomyosarcomas. They did not produce any metastases even after 5 weeks of growth.

Nickel sulphide is a well defined muscle carcinogen [4,5] producing a high incidence of sarcomas in various strains of animals. There has been no evidence, so far, that the *T. spiralis* invasion by itself can cause tumours of striated muscle. Our results, however, seem to show that trichinellosis can increase the carcinogenic effect of nickel sulphide, to a greater extent even than methandrostenolone. Androstenolone, a myotrophic steroid, has already been found to accelerate the carcinogenic activity of nickel sulphide in Sprague-Dawley rats.[6]

It is difficult to explain the enhancing effect of tri-

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