

EOSINOPHILIA IN TUBERCULOSIS

BY

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In none of the English textbooks of medicine in popular use is tuberculosis given as a cause of eosinophilia, and a review of the literature in the English language has not revealed any reference to the condition. Equally barren has been a search for a reference to eosinophil pleural effusions in tuberculosis. On the other hand, both eosinophilia and eosinophil pleural effusions in tuberculosis are widely recognized in Europe, and there are many references in French, Spanish, Italian, Danish, Russian, and German publications.

Eosinophilia in Tuberculosis

This condition is noted in foreign literature under the following headings:

After Tuberculin Injections, Whether Therapeutic or Diagnostic (Michailow, 1933; Tsinman, 1936; Korostovskaya, 1936; Umanskiy and Landa, 1938).—On the Continent this is known as the Michailow reaction.

Occurring as Part of the Syndrome of Tuberculous Lymphadenitis.—Here it may closely resemble Hodgkin's disease, making diagnosis difficult and biopsy necessary (Puech, Vidal, and Martin, 1932).

Complicating Artificial Pneumothorax Therapy.—Soglia (1932) considers the eosinophilia to be due to vagal stimulation as a result of the pleural puncture. Riera and Jacue (1933) state that it is of no diagnostic or prognostic significance.

Complicating Sanocrysin Treatment.—Riera and Jacue (1933) find that eosinophilia sometimes precedes the symptoms and signs of intolerance to the drug and may therefore prove of value in such cases. Thomsen (1937) also mentions eosinophilia as a forerunner of skin and mucous membrane reactions in tuberculous patients during treatment with sanocrysin.

Occurring Spontaneously in Pulmonary Tuberculosis.—Rugiero and Tanturi (1934), from a study of fifty cases, are of the opinion that an eosinophilia is of prognostic importance and that the disappearance of a previous eosinophilia is of grave significance. They consider that it is not a factor in the defence mechanism against the tubercle bacillus, but is an allergic phenomenon. They quote several authorities in support of this view, including Appelbaum, Claude, and Zaky; Richard; Bezançon, de Jong, and de Sorbonnes; and de Asna. On the other hand, Tagliabue and Bonizzi (1936) state that eosinophilia bears no relation to the general or local condition, but to "the tone of the neuro-vegetative system." These workers used Danielopolu's method of studying the neuro-vegetative system.

Eosinophil Pleural Effusions

The occurrence of these effusions in tuberculosis is recorded in a few instances.

Occurring Spontaneously.—Pratsicas and Antoniou (1933) describe the case of a man aged 50 suffering from Addison's disease, pulmonary tuberculosis, and an eosinophil pleural effusion. The case came to necropsy. Cristiani's (1932) patient was also a man, aged 58. The effusion was haemorrhagic, and a count of the fluid showed the unique figure of 84% eosinophils. Cappellini (1929) records a blood-stained tuberculous effusion in a young man aged 22, the effusion containing 22% eosinophils.

Complicating Artificial Pneumothorax Therapy.—Such cases are described by Gavazzeni (1935), Kabaker (1935), and Vaucher, Kabkaer, and Zenguinoff (1935). Pavie, Lefèvre, and Rossignol (1937) state that eosinophil effusions often complicate artificial pneumothorax, both in experimental animals and in humans.

The following case illustrates many of the features noted above.

Case Record

A youth aged 19 was admitted to hospital under my care on February 20 with a history of sudden onset of colicky upper abdominal pain some two months previously. These pains occurred several times a day and lasted only for a few minutes. One week previous to admission he had developed sharp pains in the right chest, together with a dry cough and marked sweating. There had been no serious illness in the past.

On examination there were fever and tachycardia and the patient looked ill. The respiratory system showed the physical signs of a right-sided pleural effusion, while the abdomen was distended and diffusely tender, and some ascites was present. A clinical diagnosis was made of abdominal tuberculosis and tuberculous pleural effusion.

Radiographs of the chest confirmed the presence of a right-sided pleural effusion but showed no localized pulmonary lesion, and the blood sedimentation rate was raised to 26 mm. at one hour. A specimen of fluid was withdrawn from the chest: this was turbid and slightly blood-stained, and a differential count showed the astounding figure of 80% eosinophil polymorphonuclear leucocytes. No tubercle bacilli were found, and culture was sterile. A blood count showed a total of 9,200 leucocytes per c.mm., with 14% eosinophils. Further counts gave similar figures. These findings appeared to negative the original diagnosis, and further investigations were then made with a view to excluding the well-known causes of eosinophilia. Examination of faeces for worms and ova and of a blood film for parasites, the Casoni test, and the van Slyke urea-clearance test threw no further light on the aetiology of the condition.

The patient's general state steadily deteriorated: the fever was of a swinging sustained type, and the abdominal pains became worse and developed into those associated with a subacute intestinal obstruction. It was therefore considered justifiable to explore the abdomen, and on March 14 a laparotomy was performed by Mr. J. C. R. Hindenach. The intestines were found to be matted together and bound down by adhesions. Some fluid was present, and there were enlarged glands in the right iliac fossa. The general appearance was that of abdominal tuberculosis, and a small portion of peritoneum was removed for histological examination. The abdomen was then closed. The tissue removed was found to be tuberculous, thus upholding the original clinical diagnosis. The patient's general condition continued to deteriorate, and death ensued eleven weeks after admission. The last blood count showed the total white cells to have remained unchanged, while the eosinophils had fallen to 9%.

The post-mortem report was as follows: "Some adhesions at bases of both lungs. Flat tuberculous patches on the parietal pleura on both sides and on the parietal pericardium. No tuberculous pulmonary foci, but tubercles in the tracheo-bronchial glands. Intestines matted together. Serous coat studded with caseous tubercles. No tubercles found in liver, spleen, kidneys, pancreas, or suprarenals."

Commentary

The points to be noted are:

The doubt thrown on a clinical diagnosis by an unexpected laboratory finding, and the final vindication of the original diagnosis by biopsy and necropsy.

The high figure for eosinophils in the pleural fluid—approaching that recorded by Cristiani (1932).

The persistence of the blood eosinophilia in a patient whose general condition was steadily deteriorating (cf. Rugiero and Tanturi, 1934).

Summary

The literature of blood eosinophilia in tuberculosis and eosinophil tuberculous pleural effusions is reviewed and a case added.

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OSTEOMALACIA AND DENTAL CARIES

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In the *Journal* of May 6, 1939 (p. 919), we published the results of the examination of the teeth of ten children with severe rickets, radiologically confirmed, in whom an average of 1.4 cavities per head was found. The ten children had ninety-six temporary teeth and 135 permanent teeth. This group was taken as a typical sample of children with severe rickets from the Kangra District of the Punjab. Diet and health surveys have been carried out in this district by the Public Health Department of the Punjab and also by Dr. Curjel Wilson. These two surveys show a high incidence of rickets and osteomalacia and definite vitamin D, calcium, and phosphorus deficiencies in the diets of most of the population there.

We concluded that the finding of almost perfect teeth in children with severe rickets demonstrates that diets deficient in calcium and phosphorus and the fat-soluble vitamins and rich in "anti-calcifying" cereals do not necessarily lead to a greater susceptibility to dental caries, and that other factors, including the physical nature of the diet, play a more important part.

Investigation of Twenty-two Osteomalacious Women

We have recently revisited the district and examined women with severe clinical osteomalacia. The results recorded in Table I show that in twenty-two cases of severe osteomalacia only thirty-four cavities were found in 565 teeth, or an average of 1.54 cavities per head. In eight of the twenty-two cases no dental caries whatsoever was observed. Hypoplasia was noted in seven of the women. Pyorrhoea alveolaris was present in half the number of women examined—in eight cases the disease was severe—which accounted for the majority of the teeth missing.

The osteomalacia was of a severe degree. Caesarean section had been done in five of the cases at the Canadian Mission Hospital, Palampur. In all of the cases severe pains in the back, pelvis, and thigh were or had been present, and in most of them the gait was typical of osteomalacia and the back was bent. Tetany was present or there was a history of tetany in eight cases. The women were all stunted, the average height being approximately 4 ft. 6 in. The majority gave a history of long and difficult deliveries. Many of them had had treatment for osteomalacia at the local Mission Hospital, at which cod-liver oil and calcium are given for that condition. The pains are usually relieved by the treatment in a few weeks or months, but these return when the women go back to their homes and their poor diet and when they again become pregnant.

These women come from a hill district and live in primitive conditions. The preliminary report of the diet survey of the district states that "the diet consists mainly of carbohydrates with relatively small proportions of protein and fat. Meat and vegetables are almost entirely absent." Very small quantities of poor-quality milk from deficient animals are occasionally consumed. Sugar is rarely eaten. Two meals only are taken daily, of rice and "chapatti" made of unmilled wheat ground to flour by the women themselves in their homes. They not only live on this poor diet but breast-feed their children for usually two to three years. There is no "purdah" system in the district, the women working in the fields in sunshine for most of the year.

The cause of the severe osteomalacia is thus the very poor diet, often associated with frequent pregnancies and long-continued breast-feeding.

TABLE I.—Particulars of Twenty-two Osteomalacious Women

Case No.	Age	Children		Osteomalacia				Teeth Present	Caries	Hypoplasia	Pyorrhoea
		Alive	Dead	Typical Gait	Bent Back	Pain in Back, Pelvis, and Thighs	Tetany				
1	20	1	2	Cannot walk	Yes	Severe	Yes	23	1	+	++ +
2	25	2	—	Yes	"	"	No	27	1	—	—
3	22	2	1	"	"	"	"	24	2	—	—
4	45	2	—	"	"	Yes	History	21	1	+	++ +
5	25	3	—	"	"	Severe	Yes	19	—	—	++ +
6	35	4	2	"	No	Yes	No	30	5	—	++ +
7	24	5	—	"	Yes	"	Yes	32	5	++ +	+
8	20	2	1	"	"	"	"	28	4	++ +	—
9	28	—	3	"	"	Severe	"	25	1	—	++ +
10	20	—	—	"	No	Yes	No	28	1	—	++ +
11	22	1	2	"	Yes	"	"	30	2	—	—
12	25	5	—	"	"	"	Yes	30	1	—	—
13	40	—	2	"	"	"	No	15	1	+	++ +
14	32	5	6	"	"	"	"	20	—	++ +	++ +
15	33	3	—	"	No	"	"	30	1	++ +	++ +
16	30	3	—	"	"	"	Yes	15	—	++ +	++ +
17	19	—	—	Cannot walk	"	"	No	28	8	—	++ +
18	30	5	4	No	"	"	"	16	—	—	++ +
19	25	—	—	Yes	"	"	"	29	—	—	++ +
20	26	1	3	"	"	"	"	32	—	—	—
21	23	1	—	"	Yes	"	"	31	—	—	—
22	22	1	—	Cannot walk	"	"	"	32	—	—	—
Totals								565	34		

Note.—In 565 teeth 34 cavities were found—an average of 1.54 cavities per head.