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MYOIDEMA

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Myoidema was first described in 1830 by Robert Graves and William Stokes, of Dublin. When they percussed the chest they noticed that "a number of little tumours appeared answering exactly to the number and situation of the points of the fingers." They did not regard the phenomenon as peculiar to pulmonary tuberculosis. Cornet (1899), Denny-Brown and Pennybacker (1938), and Smart *et al.* (1948) mistakenly credit Schiff (1858) with priority in its discovery. Schiff made an elaborate physiological investigation of its occurrence in the lower animals. He described how "a localized knot of contraction immediately forms at the site of percussion and a series of small waves of contraction leave the knot on either side and travel to the ends of the muscle fibres, sometimes being reflected back again."

Tait (1872), a Birmingham surgeon who was presumably unfamiliar with Schiff's work, coined the word "myoidema." He separated the condition into two distinct components—the instantaneous formation of a sulcus, and the "nodule or intumescence of Stokes." He used the term for the nodular variety only, which he regarded as a significant sign of pulmonary tuberculosis. The sulcus formation, he said, may be "frequently met with in health." He agreed with the finding of Stokes that myoidema was more pronounced in the pectorals on the diseased side in unilateral pulmonary tuberculosis. Broadbent (1934) extended this observation further by pointing out that if both lungs were involved myoidema was more readily obtained on the more diseased side. In 12 cases of lung tuberculosis in our hospital, of which six were radiologically unilateral, we found myoidema to be equally marked in pectorals and biceps on the two sides.

West (1879) regarded the sign of little value in the diagnosis of any one disease, as it frequently occurred in a wide variety of conditions, and might be physiological. He thought the two kinds of contraction—the nodular and the fascicular—need not necessarily be associated, and that the term "myoidema" was not accurate as there was no oedema at all. He also pointed out the importance of subcutaneous fat in the production of the sign. The findings of Labbé (1881), Klippel (1903), Jeanselme and Lermoyez (1885), Curschmann (1905), Shively (1905), and Langley and Hashimoto (1918–19) supported West in showing the sign to be non-specific.

Féré and Papin (1901) drew attention to the presence of myoidema in the muscles of fatigued cyclists, of epileptics following a fit, and in muscles undergoing atrophy in general illnesses, general paralysis, and mental deficiency. They concluded that myoidema was a sign of muscular fatigue from any cause. Ebbecke (1935) considered the swelling of myoidema to be similar in nature to cathodal swelling. Debenedetti (1930) believed that myoidema ceased to appear when the individual put on weight, and suggested that this finding may be of prognostic importance in pulmonary tuberculosis.

Most of the English textbooks do not distinguish the two distinct components of muscular irritability, and the local lump (termed variously knot, ridging, nodule, intumescence, or nodular contraction) for which the word

myoidema was originally coined is not even mentioned. The terms "muscular irritability" and "myoidema" are mistakenly used synonymously for the fascicular or linear contraction-bands produced by tapping the pectoralis major (Martin, 1946). This error may be one explanation of the sign's having lost favour.

In South Iraq in 1944–5 obvious myoidema was found in 82 cases of peripheral neuritis, and this led to a search for it in patients suffering from other diseases and in supposedly healthy soldiers on duty. A high percentage of the latter groups also demonstrated a marked degree of myoidema. We tentatively ascribed the sign to malnutrition, and in particular to thiamin deficiency.

Our first observations were made in 1944–5 at an Indian General hospital at Shaiba in the extreme climate of South Iraq. The local garrison consisted of Indian and British troops, but the former were in a large majority. In 1945–6 observations were made on British troops returning from South-east Asia and India, on men working in a troopship, and on Indian troops in Syria-Lebanon. The Indian soldiers consisted of two dietetic groups, the meat-eaters and the vegetarians; the vegetarians had never taken meat or eggs in their life, but otherwise the living, eating, and working conditions of the two groups were identical.

TABLE I.—*Distribution of Myoidema*

	Nationality	No. of Men Examined	No. of Cases of Myoidema				
			Severe	Less Severe	Moderate	Slight	Absent
Hospital patients in S. Iraq (1944)	Indian M.	1,075	817	73	145	35	5
	,, V.	320	260	20	29	8	3
		1,395	1,077	93	174	43	8
Men on duty in S. Iraq (1944)	Indian	409	270	50	70	13	6
	British	176	31	30	50	32	33
	Chinese	109	64	14	20	11	0
Men returning from India and S.E. Asia (troopship, 1945)	British	124	19	27	21	17	40
	Waiters and cooks	34	20	8	2	2	2
	Lascars—stokers	39	22	11	3	2	1
	Deck-hands	42	13	11	6	5	7
Hospital patients, Syria-Lebanon (1945–6)	Indian M.	410	146	84	64	104	12
	,, V.	51	25	8	10	5	3
		461	171	92	74	109	15
Men on duty in Syria-Lebanon (1945–6)	Indian M.	352	156	72	48	52	24
	,, V.	72	36	10	13	10	3
		424	192	82	61	62	27

M = Meat eaters. V = Vegetarians.

Table I shows the distribution of cases of myoidema. It demonstrates: (1) Indian troops on duty had a degree of myoidema less than those sick in hospital; (2) meat-eaters often showed a lesser degree of myoidema than vegetarians; (3) British troops had a lower incidence of myoidema than all the other groups examined; and (4) Indian men on duty and in hospital in Syria-Lebanon had a lower incidence of myoidema than their counterparts in Iraq.

Technique of Elicitation

The patient held the forearm extended by the side of the body with the muscles relaxed. The belly of the right biceps was strongly pinched between the observer's thumb and forefinger, letting the muscle slide between them. A constant maximum strength of stimulus was aimed at. Myoidema was considered to be absent if the lump subsided before it could be palpated.

Myoidema could be elicited in all skeletal muscles, though in small and deeply placed muscles, especially if they were covered with strong fascia and fat, the sign was difficult to see and had to be palpated. Tapping with the fingers or a patellar hammer, as well as squeezing, elicited myoidema. The stimulated fibres momentarily contracted as a whole as the lump appeared. There were thus two components: the sustained lump at the site of the stimulus, and the flickering contraction of the whole length of the muscle fibres that preceded it. In patients with little subcutaneous fat a wave of contraction started at the lump in the pectoralis major and spread in both directions along the course of the fibres. In a few of these patients the wave appeared to return from the periphery to the site of the stimulus, possibly owing to the earlier subsidence of the peripheral contraction. The muscles which showed marked myoidema were often tender and the squeezing was painful.

Myoidema was not influenced by general anaesthesia or by spinal and nerve-block analgesia. It was therefore not a reflex but a direct response to a mechanical stimulus.

Myoidema in Neuritis

Severe myoidema was present in each of the 82 cases studied. In the 51 patients treated with thiamin it decreased in intensity in all except one, who was receiving anti-syphilitic arsenical injections at the same time as the thiamin. The decrease was gradual, and was obvious after about two months' treatment. There was no constant decrease in controls receiving hospital diet and cod-liver oil but not thiamin injections. Patients with neuritis who had recovered after two to three months' treatment with thiamin were discharged to duty. In these patients the myoidema had decreased or completely disappeared. Two cases relapsed after two months' duty. Myoidema was pronounced in wasting muscles, but not in those severely wasted. It was not more obvious in the muscles affected by neuritis.

Myoidema and Vitamin Deficiency

Deficiency of vitamin B (and possibly of vitamin A) was often seen in troops under our care (Table II). The presence or absence of these deficiency signs and of

TABLE II.—*Incidence of Vitamin Deficiencies*

	Nationality	No. of Men Examined	No. of Cases				
			Angular Stomatitis	Bitot's Spots*	Cheilosis	Generalized Glossitis	Geographical Tongue
Hospital patients in S. Iraq (1944)	Indian M.	1,448	1,108	85	1,218	11	62
	,, V.	415	325	18	329	9	13
		1,863	1,433	103	1,547	20	75
Men on duty in S. Iraq (1944)	Indian	409	362	15	465	2	8
	British	176	58	—	82	1	13
	Chinese	109	98	—	99	—	—
Hospital patients, Syria - Lebanon (1945–6)	Indian M.	410	186	7	114	—	5
	,, V.	51	23	5	19	—	3
		461	209	12	133	—	8
Men on duty in Syria - Lebanon (1945–6)	Indian M.	352	129	13	50	—	5
	,, V.	72	21	3	3	—	2
		424	150	16	53	—	7

* The relation of Bitot's spots to vitamin A deficiency is not established.

myoidema was recorded at the same examination, except for the troopship population, in whom myoidema only was looked for.

Analysis of the incidence of vitamin deficiency signs showed: (1) Indian troops on duty in Syria-Lebanon had a lower incidence of deficiency signs than those in hospital; this was not so in Iraq. (2) The meat-eating patients in hospital (Syria-Lebanon) showed a lower incidence of deficiency signs than vegetarian patients. (3) The British troops had a lower incidence of deficiency signs than the Indian troops. (4) Indian troops and hospital patients in Syria-Lebanon had a lower incidence of deficiency signs than those in Iraq: the hospital patients in Syria-Lebanon showed a lower incidence than men on duty in Iraq. These findings suggested that severe myoidema and vitamin deficiency were possibly related.

Myoidema and Chest Pain

The commonest complaints of medical out-patients in our general hospital in Iraq in 1944 were pain on each side of the chest and loss of appetite, general weakness, and general aches and pains. Men with these complaints often had painful severe myoidema and active tendon-jerks. Some patients complained of giddiness, and others of cramps in the calf muscles and sciatic pain. Scores of such men were seen at the end of the hot weather, and were sent to hospital with such diagnoses as myalgia of the chest, neuralgia, fibrositis, pleurodynia, pleurisy, and sciatica. Many were sent for investigation of supposed early tuberculosis or cardiac disease and were unnecessarily submitted to *x*-ray and other examinations. Some were given procaine injections for supposed fibrositis.

Chest pain was the commonest single symptom, and was complained of as a constant diffuse ache on both sides of the chest. This pain usually disappeared after thiamin injections had been given for 10 to 20 days, or after longer periods if yeast tablets were taken. The frequency with which pain in the chest responded to thiamin was striking, but under field conditions a controlled experiment was not possible. It was thought that this pain was associated with fatigue of the chest muscles, which were presumably in an early pathological condition, as shown by severe myoidema. Patients recovered when thiamin was given. Many of these patients were seen by a psychiatrist, because the symptoms were often excessive fatigue, exhaustion, and depression, but in most cases no psychiatric abnormality was found. The same symptoms, however, were often absent in men on duty who were found to have severe myoidema. We had the impression that the incidence of severe myoidema was greater at the end of the hot weather. Routine urine examinations showed no salt depletion.

Cause of Myoidema

Kollert and John (1925) reported that 40% intravenous glucose abolished or diminished myoidema; they suggested that the sign was related to failing carbohydrate metabolism. The addition of insulin increased the effect of intravenous glucose. John and Dattner (1926) considered that in a number of conditions producing vasomotor disturbance myoidema was always present, and the capillaries in these patients showed thickening, tortuosity, and absence of contractility. They thought that intravenous glucose improved the contraction of capillary walls, increased the blood flow, and brought about disappearance of myoidema. Grill (1927) found that a large number of patients showing myoidema had raised lactic acid levels in the blood.

Koumans (1928) reported myoidema to be a common finding in poor-class Javanese, Malays, and Chinese in the Tropics. Their well-to-do counterparts and the Europeans, even after a long stay in the Tropics, did not show myoidema. He found it had no relationship to any disease, to overexertion, to the blood-sugar level, or to deficient carbohydrate intake, but was related to unbalanced diet and undernutrition. He regarded myoidema as a sign of avitaminosis. John (1928), criticizing Koumans's paper, stated that myoidema did occur in cases showing no avitaminosis. Seto (1937), working in Japan, confirmed Koumans's findings.

Denny-Brown and Pennybacker (1938) made electromyographic studies of two cases showing myoidema and, citing the work of Gelfan and Bishop (1932, 1933) and Gelfan (1933–4), were of the opinion that the phenomenon was due to a disturbance of the excitability of muscle fibre, predominantly of its contractile contents. They postulated a disturbance of the potassium : sodium ratio and the raising of excitability of both sarcolemma and muscle fibre. These workers were of the opinion that in a normal muscle percussion induced a "small knot of contraction with tightening of the remainder of the fasciculus," and that both subsided in one to five seconds, according to the intensity of the stimulus. If the local contraction lasted more than 10 seconds they regarded it as abnormal. Smart and his collaborators (1948), studying Belgian workers who were receiving inadequate food, found the biceps "ridge" lasted longer in them than in healthy R.A.F. personnel (about 4.5 seconds and 2.1 seconds respectively), but the phenomenon decreased in duration after food supplements had been given. They could not indicate which biochemical lesion produced the hyperirritability.

Analysis of the association of severe myoidema with coincident illness, dietetic habit, and vitamin deficiency suggested that in our cases the sign denoted malnutrition. The evidence for associating myoidema with disordered thiamin metabolism was that in 51 cases of neuritis treated with thiamin myoidema decreased and in many cases disappeared. Thiamin also cured the associated chest pain and muscular tenderness. The part played by thiamin, however, may have been an indirect one, by restoring the appetite.

Under certain conditions severe myoidema may be a pre-neuritic phenomenon. Peripheral neuritis was common in our garrison, and all the cases had severe myoidema. The incidence of severe myoidema among troops on duty was also high: they often had tender muscles, pain in the chest and calf muscles, cramps, and exaggerated tendon-jerks, like those seen in early cases of neuritis in our series.

Since the men's rations were stated to contain normal quantities of thiamin (3 mg. daily) other causes for disordered thiamin metabolism were considered. Thiamin deficiency may have been due to loss of appetite, to diarrhoea, and to loss in sweat (especially during the hot weather, when sweat loss amounted to over 10 pints (5.7 litres) daily). An attack of fever, or extra marching, may have precipitated thiamin deficiency by increasing thiamin requirements.

Summary

Severe myoidema was found in South Iraq in a series of cases of peripheral neuritis in soldiers on duty and in patients suffering from other diseases.

The condition was related to the nutritional state and the dietetic habit of the groups of men examined. It accompanied signs of vitamin deficiency, and disappeared when the cases of peripheral neuritis were treated with thiamin. It is suggested

that in our cases severe myoidema was a sign of malnutrition and was probably related to thiamin metabolism.

An explanation is attempted of the growing disrepute of myoidema as a physical sign.

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