AETIOLOGY OF FIBROSITIS: A REVIEW

BY

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From a review of systems of classification of fibrositis (National Mineral Water Hospital, Bath, 1940 ; Devonshire Royal Hospital, Buxton, 1940 ; Ministry of Health Report, 1924 ; Harrogate Royal Bath Hospital Report, 1940 ; Ray, 1934 ; Comroe, 1941 ; Patterson, 1938) the one in use at the National Mineral Water Hospital, Bath, is considered most valuable. There are five divisions of fibrositis as follows : (a) intramuscular, (b) periarticular, (c) bursal and tenosynovial, (d) subcutaneous, (e) perineuritic, the latter being divided into (i) brachial (ii) sciatic, etc.

Laboratory Tests

No biochemical abnormalities have been demonstrated in fibrositis. Mester (1941) claimed a specific test for "rheumatism", but Copeman and Stewart (1942) did not find it of value and question its rationale. The sedimentation rate is usually normal or may be slightly increased; this is confirmed by Kahlmeter (1928), Shackle (1938), and Dawson and others (1930). Miller and Gibson (1941) found a slightly increased rate in 52.3% of patients, and Collins and others (1939) found a (usually) moderately increased rate in 35% of cases tested.

Case Analyses

In an investigation Valentine (1943) found an incidence of fibrositis of 31.4% (60% male) at a Spa hospital. (Cf. Ministry of Health Report, 1922, 30.8%; Buxton Spa Hospital, 1940, 49.5%; Bath Spa Hospital, 1940, 22.3%; Savage, 1941, 52% in the Forces.) Fibrositis was commonest between the ages of 40 and 60; this is supported by the Spa Hospital Report, Buxton, 1940. Among the under forties it was commoner in females, the reverse being the case in the over sixties. Radiological changes were found in 50% of men and 80% of women examined, and there was a familial tendency to "rheumatism" in 23% of cases. In men the sciatic site was the commonest affected. (38.8%) followed by the lumbar area, shoulder girdle, and periarticular regions, 18.8, 17.2, and 15.5% respectively. In women the sciatic site was affected in 25.4%, the figures for the other areas being 18.6, 23.6, and 18.6%. Jews formed 3% of all patients in the consecutive series of 481 cases, and of these 85% had been diagnosed as suffering from fibrositis.

Bacteriological Theories

Llewellyn and Jones (1915) collected a large amount of material from very scattered sources and concluded that there were primary and secondary factors in the production of fibrositis. The primary causes they took to be either bacterial or auto-intoxicative ; they alleged that most fibrositic subjects suffer from focal sepsis, and that some of them improve dramatically after this has been eradicated, and also following the use of autogenous vaccines or colonic wash-outs with the Plombière technique. The experiments of Goadby and of Rosenow are quoted,

whereby the lesions of "rheumatic myositis" were produced in animals by the inoculation of a streptococcal strain. Collins (1940), however, thinks that animal experiments are unlikely to be helpful in elucidating the cause of human fibrositis, which is essentially a subjective phenomenon, and draws attention to the difficulty of deciding whether experimentally produced lesions may not have been brought about by other causes, e.g. incidental trauma.

This view, that the majority of fibrositic subjects suffer from focal sepsis, does not now meet with general agreement (vide Douthwaite, 1940; Slot, 1940; Collins, 1940; Savage, 1941; Ellman and others, 1942; Valentine, 1943) and the results of removing any foci present are frequently disappointing (Gordon, 1940; Slot, 1940); colonic lavage and the use of autogenous vaccines have now been for the most part abandoned. Crowe (1926) claimed good results in all types of "rheumatism" with vaccines, but it is seldom that a patient's improvement can be attributed directly to such treatment. Vaccines are often prepared from septic teeth, but the work of Fish (1943) shows that there is comparatively little absorption from septic teeth, as the inflammatory area becomes walled off. There is more toxic absorption from gingivitis. Another point is that any tooth will produce a growth (usually mixed strains of *Strep. viridans*) on a culture plate, unless the gums were previously cauterized, because non-sterile saliva is forced between the tooth and the periodontal membrane in the process of extraction. This is another fact casting doubt on the value of such vaccines. Vaizey and Clark-Kennedy (1939) have also questioned the widely-accepted relation between dental sepsis and the rheumatic diseases. However, there are occasional cases which appear to justify vaccine treatment, such as the one quoted by Myers (1939).

The theory of sub-infection put forward by Adami is also cited by Llewellyn and Jones (1915) as a possible cause. According to this hypothesis, bacteria are carried in the blood stream from septic foci or from the alimentary tract, and produce chronic interstitial fibrositis without setting up suppuration. This is stated to be brought about by the proliferation of connective-tissue elements caused by the endotoxins released upon lysis of the bacteria. According to more modern investigations, however, toxins usually enter the system via the lymphatics, and such bacteria as enter the blood stream are, unless in the case of an overwhelming dose, destroyed in quite a short time by the leucocytes. Auto-intoxication was also advanced as a possible cause, but need not further be discussed.

Secondary or exciting factors Llewellyn and Jones (1915) state to be low temperature, excessive or deficient muscular exercise, and the influence of occupation or strain. The same writers include traumatic fibrositis in their classification, but reserve it for cases in which direct injury plays an obvious part, as in muscular strains and tears.

Five years after the publication of the work of Llewellyn and Jones, Stockman (1920) distinguished (i) original causes, including acute rheumatism, rheumatoid arthritis, sore throats and influenza, gonorrhoeal septicaemia, mucous colitis, and chronic *B. coli* infections of the bladder; and (ii) subsequent attacks, precipitated by exposure to cold, or damp, unusual or violent muscular exercise, acute indigestion, and slight febrile attacks.

Hypersensitivity to Diet

Thomson and Gordon (1926) regard strain and exposure to chill as contributory causes, and postulate as the essential feature of fibrositis a toxin, derived usually from the colon or from focal sepsis. In support of this view they put forward the questionable statement that fibrositic subjects possess a functional inferiority of the lower digestive tract and are particularly liable to gastro-intestinal disorders.

Writing of lumbago, Llewellyn (1927) makes out a case for hypersensitivity to certain protein foods, and invokes a similar explanation for the action of septic foci, viz. one transient bacteraemia sensitizes the system, and a second produces an anaphylactic reaction, of which the lumbago syndrome is a symptom. In the same article he stresses the importance of trauma in the history of lumbago, and also makes one of the earlier references to vasomotor defects in the rheumatic diseases. Such infective theories have been criticized by Douthwaite (1938), who considers that deep massage might be expected to worsen the condition rather than improve it, as it does in actual fact, were the tissues invaded by bacteria. Collins (1940) finds that painful states of muscle and connective tissue may result from toxæmia or septicaemia, but does not consider that fibrositis comes under this category at all.

Hypothyroidism

Douthwaite (1938) criticizes Llewellyn's suggestion of a subthyroid state as being an important precursor of chronic rheumatism, stating that if any abnormality exists in fibrositis it is more likely to be hyperthyroidism. This criticism is in part supported by the investigations of Potter (1938), who found "no great degree" of hypothyroidism in the majority of ninety female cases of chronic rheumatic disorder. In general, the state of the thyroid seems to have little influence in fibrositis.

Vascular Deficiency and Metabolites

Copeman (1933) mentions sensitivity to diet and possibly to bacteria or their toxins; also the poor adaptability of the skin capillaries to changing atmospheric conditions, with secondary accumulation of metabolites and effusion of serum. He also mentions chronic strain, and stresses the importance of fatigue. Douthwaite (1938) criticizes this suggestion of allergy and considers that fibrositis differs too much in its onset and course from the proved allergic diseases to be considered in the same category. Ray (1936) holds similar views to Copeman, mentioning capillary stasis, sensitivity to articles of diet, and chronic postural strain. Also discussing the metabolic factor, Gordon (1936) expresses the view that in the inefficient circulation, metabolites will tend to collect in fibrous tissue, this being relatively less vascular, especially in parts where the greatest muscular activity occurs.

Reconciliation between the infective and metabolic schools is attempted by Patterson (1938), who postulates an infective "seed", which for implantation depends on suitable "soil" prepared by metabolic processes and endocrine constitution. He also makes an early reference to psychological factors, claiming the importance of over-work or mental strain.

Physiological Reports

At this point the work of physiologists may be considered; Lewis (1932) has suggested the elaboration of a pain stimulant, which he calls the P-factor, in the ordinary course of muscular activity. This is normally removed in the blood stream but persists in the ischaemic conditions. The P-factor seems to depend directly on the state of the local circulation; if ischaemic muscles are exercised, pain is produced, the pain remains constant if the exercise is stopped and the

ischaemia maintained, and the pain disappears rapidly when the circulation is restored. Regarding the origin and distribution of pain, Kellgren (1939) observes the effects of stimulation of interspinous ligaments, and states that whether pain is segmental or local depends on the depth at which the tissue is stimulated, rather than on the nature of the tissue. According to Samson Wright (1939), fatigue from activity is not due to increase of the H-ion concentration nor to lactic acid. Pemberton (1935) mentions experiments not indicating any abnormal production or disposal of lactic acid in patients with arthritis. Pennington (1939) found increased lactic acid in the sweat after exercise in 100% of cases, and argues from this that there is strong presumptive evidence for the pathological identity of fibrositis with physiological fatigue sequelae in muscles.

These reports in general do not suggest any marked alteration of lactic acid metabolism in rheumatic disorders, and, should any local alteration be found, it will probably be secondary to local vascular deficiency. There is insufficient evidence to show whether this capillary defect is primary.

Gouty Fibrositis

Although the term "gouty fibrositis" is rarely used now, there is some support for the idea that fibrositis may on occasion be associated with an increased blood uric acid. Buckley (1940) mentions cases of this type and considers they should be treated on the same lines as the more common types of gout.

Myalgia and Epidemic Myalgia

Myalgia is defined by Good (1942) as "a muscular disease localized in well-defined parts of the muscle and in its appendages—tendon, ligaments, perimysium, fascia—corresponding anatomically to the origin, insertion, the course or the edge of the muscle". It thus appears to be essentially a fibrositic lesion.

Epidemic forms have been described of diseases bearing, in their symptoms at least, some resemblance to the condition which we ordinarily recognize as "rheumatic fibrositis". An epidemic form of brachial neuritis has been described by Wyburn-Mason (1941). He considers that there may be infection by a neurotropic virus, or an allergic reaction to infection, but thinks it unlikely that this is a "fibrositic" condition. Houghton and Jones (1942) describe a series of cases of persistent myalgia following sore throat. They suggest as a cause an unidentified myotropic strain of virus. Douthwaite (1937) quotes a report by Massell and Solomon (1935) of an epidemic of benign myalgia of the neck. It was unilateral affecting mainly the trapezius muscle. It usually lasted two or three days, and deep massage was the best cure. Beeson and Scott (1941) described epidemic myalgia of the neck, with an attack rate of 17.8%. It was commoner in women, and resembled acute fibrositis rather than Bornholm disease. Trail (1943) doubts whether such a disease as epidemic myalgia exists and does not consider it a disease entity. Beeson and Scott (1942) made very thorough investigations on 125 cases of epidemic myalgia of the neck and shoulders, and commented on their results with admirable precision and restraint. The sedimentation rate was not increased in any cases, but a few showed slight pyrexia or lymphocytosis; a history of common cold at the time of onset of the complaint was obtained in 41% of cases, and the myalgia was always worsened by exposure. Washings from the nose and throat of patients failed to reproduce the disease in other persons; transfusions of whole blood from patients, however, reproduced the myalgia in a significant proportion of volunteers, and reasons are advanced to explain failures of transmission.

The largely subjective nature of the complaint makes the assessment of the experiment difficult, but the disease is from all accounts dissimilar to fibrositis.

Subcutaneous Fibrositis

This term is rather loosely applied to superficial tenderness of a fibrositis-like type, which the writer has found common at the nape of the neck and around the scapula. Llewellyn and Jones (1915) mention this type of subcutaneous tenderness, occurring alone or in association with muscle-sheath fibrositis, and Stockman (1928) reserves the term panniculitis for a type of chronic inflammation of the areolar tissue of the panniculus adiposus, mentioning that subjects might be quite healthy in other respects, and that excessive fat or myxoedema were not essential features.

Panniculitis has a different significance for several other writers, who consider it in association with endocrine disease or pathological obesity. Ray (1936) blames endocrine factors, especially hypothyroidism, and states that it is difficult to distinguish from Dercum's disease (see Bochrach, 1902), Thomson and Gordon (1926) state that all cases are hypothyroidic, and Llewellyn (1927) speaks for a similar association, while Buckley (1940) also suggests an endocrine basis.

The Fibrositic Nodule

The fibrositic nodule is by no means universally recognized to exist, and those who do recognize it do not always distinguish different types.

The fibrositic nodule was described by Stockman (1920) as an inflammatory hyperplasia of connective tissue in patches, containing numerous fibroblasts and a sero-fibrinous exudate; it is swollen, painful, and tender, tends to contract, and can be massaged away. Collins (1940) who has examined some of Stockman's sections, is convinced that no deductions concerning the cause of the nodules can be drawn from them.

Nodules were demonstrated by Collins (1939) in a case of traumatic tenosynovitis showing collections of lymphoid cells in the synovial villi of the tendon sheath. In another case Collins (1940) described a traumatic patellar tendon nodule showing central degeneration, lymphocyte cuffing, and lymphorrhages, but no intense fibroblastic reaction. He comments that traumatic causes may explain histological infective inflammations. Possibly lymphocytes are attracted to the site by the products of cell autolysis.

Elliott (1944) describes nodules, clinically similar to those found in fibrositis discovered in the leg muscles of cases of sciatica with prolapsed discs. Gordon (1940) declares the nodule to be the hallmark of the fibrositic subject but the tombstone of the fibrositic attack, and Copeman (1943) suggests that chill, trauma, and focal sepsis may reactivate nodules which have lain dormant since their inception during previous acute infections.

Fatty nodules are described by Collins (1940). Those had been diagnosed by physicians as "typical fibrositic nodules" and proved to be fatty masses, similar to those described by Stockman (1920) in panniculitis. Copeman and Ackerman (1944) describe oedematous fatty lobules whose removal resulted in the cure of painful tender nodules of the back. Copeman and Pugh (1945) demonstrated nodular fatty lobules enclosed within fibrous walls in the subcutaneous tissues, which constituted trigger points for fibrositic pain. These fat-herniae are considered liable to a type of non-inflammatory oedema with the production of pain

and tenderness; this is the rationale of the dehydration therapy suggested, which is carried out by restriction of fluid intake and intravenous injection of hypertonic saline. Considerable improvement was noted in a statistically significant number of cases in the fairly small series treated.

Areas of muscular spasm have been described by Llewellyn and Jones (1915) as typical of fibrositis; cases may show spastic patches about an inch in diameter, commonly in the trapezius, latissimus dorsi, and sacrospinalis. Elliott (1944) describes a "nodule" which proved under anaesthesia to be localized muscle spasm. This, however, is regarded by Copeman and Pugh (1945) to be a reflex response to irritation from a pathological process situated in tissues outside the muscle.

Collins (1937) describes the pathology of nodules found in rheumatoid arthritis and in acute rheumatism. He is not prepared to accept a common aetiology for the two types.

Postural and Occupational Stresses

Considering sciatica from an orthopaedic standpoint, Wesson (1938) contends that sciatic pain is referred pain from lumbo-sacral muscles, ligaments, and joints, usually from postural imbalance or occupational causes, and that it may only appear in states of anxiety and ill health. Cochrane (1938) argues from the infrequency of sciatica in childhood that it is only when the muscular support of the lower back is strained, or weakened by wear and tear, that lumbo-sacral anomalies and lumbar or sciatic pain occur. de Seze (1939) holds that sciatic pain is mainly caused by discogenetic disease of the space between the fourth and fifth lumbar vertebrae, brought about by deficient musculature of the abdominal wall consequent upon man's assumption of the erect posture.

Comroe (1941) puts forward an American point of view in holding responsible chilling of a part of the body previously over-heated, and trauma, either in the form of injury or chronic strain.

Weil (1939), the French authority, takes the view that continued strain, as indicated by the word "microtraumatisme", is the main cause of fibrositis. He regards infection as unimportant, but postulates an individual predisposition of an endocrine or sympathetico-vascular type, accompanied by abnormalities in the blood chemistry. The relation between rheumatism and occupational trauma has been dealt with in an interesting article by Duvoir and Desoille (1936), who demonstrate that certain trades cause distinctive types of rheumatic disability. Most French writers, although laying stress on the occupational factor, think there is an underlying individual susceptibility in addition.

An interesting aspect of this question is the development of chronic rheumatism in workmen using pneumatic tools (Wessenbach and Francon, 1938; Copeman, 1940). In this country, Slot (1940) has shown that fibrositis affects those parts of the body which bear the brunt of the occupational strain; for instance, the neck muscles are affected in market porters who carry heavy weights on their heads; whereas in bus conductors, locomotive firemen, gardeners, and dustmen

the muscles of the arm and hand are often affected. Also, at the end of the last century, Arbuthnot Lane drew attention to the influence of occupation in determining the physical constitution of the individual. Van Breemen (1937) has also lent his support to this discussion, but remarks upon the difficulty of deciding if such cases can receive compensation under the existing regulations.

A high proportion of miners with fibrositis was found by Buckley (1933); and Savage (1941) states that most of his cases of fibrositis in the Services occurred in ex-miners. The high proportion of cases of fibrositis admitted to the Buxton Spa Hospital may be accounted for by the mining districts nearby. Conybeare and Glover (1938) stress the influence of occupation in causing postures favouring fibrositis, and the Buxton Hospital Report (1940) shows fibrositis to be commonest in outdoor labourers and miners.

Psychological Factors

Interest has recently been shown in the psychological components of fibrositis. Gordon (1940) distinguishes three varieties: (1) when pain is used as a symbol of emotional discontent; (2) when the whole sensorium is over-sensitized as a result of acute emotional disturbance; (3) when chronic emotional disturbances produce endocrine and autonomic disturbance through vegetative controlling centres in the hypothalamic region which are followed by fibrositis. He quotes cases illustrating the production of fibrositic symptoms by these types of psychological disturbances. In an investigation by Ellman and others (1942), 23 out of 50 patients diagnosed as fibrositis showed no physical abnormality.

Lindstedt, whose teaching has influenced the Scandinavian school, apparently thinks it impossible to overestimate the importance of the psychogenic aspect. Writing principally of sciatica (1938), he states that there is a depressive and psychoneurotic disposition in most cases. A person of this type is hypersensitive to pain, especially when he is over-fatigued from general constitutional causes. Thus muscular strain from faulty posture, working conditions, or from slight congenital or acquired deformities of the lower limbs such as lumbo-sacral anomalies, flat-foot, etc., will produce pain of a sciatic type; or sometimes muscular imbalance alone, without psychoneurotic tendencies, will give rise to pain. The tenderness in the leg, he says, is muscle tenderness, and is not due to neuritic irritability; similarly, he considers that the pain produced by Lasegue's sign is not from stretching the sciatic nerve, but from stretching the tender muscles.

In this connexion the writer suggests the following classification. First, conversion hysteria covering cases with fibrositic-like symptoms but with no physical signs or pathology; the symptoms serving a purpose not directly related to their type. This group also includes cases of fibrositic-like pain occurring as a hypochondriacal conversion in the course of an anxiety state, and cases in which the pain of a former or recent attack is reproduced or perpetuated as a hysterical manifestation. Second, psychosomatic fibrositis, some basis of fibrositis being unconsciously magnified, and to some extent representing the conflict, e.g. pain representing a feeling of injustice, and advanced as a reason for reparation.

There seems to be some justification for a third though uncommon group in which massive and generalized symptoms of fibrositis appear as the main and perhaps only manifestation of a neurosis, possibly from functional vascular and toxic causes. Mention is made of this type of case in the next paragraph.

The Peripheral Vascular System

The vascular changes in fibrositis have lately received much more attention. Pemberton and Pierce (Pemberton, 1935) found dysfunction of the smaller vessels of the skin in arthritis, and Pemberton believes that closed capillary areas, play an important part. Bisset and Woodmansey (1932), after photographing capillaries in normal and arthritic subjects, reported spastic capillaries in most cases of well-established rheumatic conditions. Pemberton (1935) also quotes experiments by Rhumann, who found capillary abnormalities, including a slow and interrupted flow, in 70% of patients with muscular rheumatism. Pemberton goes on to describe animal experiments by Goldhaft and himself, whereby arterial ligation, or venous stasis, produced bony proliferative changes.

The capillary spasm alleged to be present in fibrositis might be explained on the grounds that, owing to unfavourable or fluctuating conditions of temperature or humidity, the peripheral circulatory reflex protests against such abuse by refusing to function, or at least to function well. If, then, there is an angio-spastic state of the capillaries in fibrositis, it is possible that the osteophytosis which is fairly commonly found in chronic fibrositis is a function of the tissue ischaemia. The theory of halisteresis is no longer acceptable: only bone cells can elaborate or absorb bone matrix (Newton, 1939); venous stasis, with its retarded evacuation of waste products, would probably act as an irritant and cause deposition of bone (Fish, 1943). The typical picture of the capillaries in fibrositis is not of venous stasis, but of narrow, spastic vessels, some being closed altogether (Valentine, 1943). Possibly this could also bring about collections of metabolites such as would stimulate the embryonic resting osteoblasts found in fibrous tissue, with the resulting formation of osteophytes. Another theory might be that, if there is any connexion between fibrositis and osteophytosis, perhaps both fibrous tissue and bone are affected by the same vascular causes and react as their tissues are constituted; the fibrous tissue undergoing the changes leading to the production of fibrositis, and bone tissue producing osteophytes, much as Muir (1924) has put the case in osteo-arthritis. An attempt to discover whether any support could be found for these theories by the examination of case-records of fibrositis proved abortive, as many cases had not been radiographically examined on their first admission to hospital, and others had to be disqualified owing to the "wear and tear" osteophytosis found in most individuals after 55 years of age. It was thus impossible to say whether the fibrositis had run *pari passu* with osteophytosis (Valentine, 1943).

At present there is little or no support for the theory of primary vascular dysfunction in rheumatoid arthritis at least. Collins (1938) takes the view that these vascular phenomena are secondary to the joint disease, and in a further paper he and his co-workers (Collins and others 1939) state that the defect of vascular function is not an essential condition nor the cause of rheumatoid arthritis, but merely an inherent disability of certain subjects leading to aggravation of symptoms.

In cases of acute and fairly superficial fibrositis, pressure over the tender areas causes the appearance of a red flare similar to that produced by the local inoculation of histamine (Valentine, 1943). This phenomenon is often remarked during diagnostic palpation of the tissues, and does not occur, or at least not to the same extent, over unaffected areas. This leads one to think that the release of H-substance or histamine in regions affected by fibrositis might be a compensatory mechanism on the part of the body, designed to increase the sluggish circulation of the affected parts. The treatment of rheumatic complaints by histamine cataphoresis has received some attention (Copeman, 1935; Potter, 1935) and it is of interest to note that Kling (1934) found beneficial results from this line of treatment in the rheumatic group only in "myositis". Another interesting point is the low blood pressure sometimes found in generalized fibrositis. It is con-

ceivable that this is due to the mass release of "H-substance" in the manner suggested above, and similar in nature to, but milder, than the histamine shock described by Lewis (1927).

Eclectic Views

Telling (1935) postulates a complex aetiology but stresses chronic trauma, septic and toxic influences, and climate. Gordon (1940) suggests that fibrositis is a tissue reaction to a number of pathological causes, and Harman (1940) notes that the symptom of pain from deep fibrositis is similar to that from any lesion in a like situation (see also Kellgren, 1938, 1942). Fibrositis might also conceivably be a pattern reaction with a number of triggers, as Fletcher (1939) suggested was the case in osteo-arthritis. In view of its varying pathology, Collins (1940) has drawn attention to the need for fibrositis to be defined afresh in clinical terms, and stresses the diversity of underlying causal conditions.

Fibrositis seems to emerge as an inflammatory tissue reaction, largely from mechanical and physical and sometimes toxic causes. Some defect of circulatory functional response, probably in the peripheral reflex arc, may also render the individual more liable to fibrositis. For convenience the disease may be divided into two groups—the type apparently brought about by physical agencies, such as chronic strain and chill, and that caused by toxic influences such as influenza, gout, acute rheumatism, or focal sepsis, often showing a preference for tissues already damaged by physical means.

Summary

The classification of fibrositis in use at the Bath Spa Hospital is recommended; no diagnostic laboratory tests are of value in fibrositis; an analysis of case records is presented; epidemic myalgia appears to be a separate disease entity; subcutaneous fibrositis differs from the usually accepted meaning of panniculitis; the pathology of the fibrositic nodule is discussed; the role of the peripheral vascular system in fibrositis is considered important; from a discussion on theories of the causation of fibrositis by bacterial toxins, metabolites, postural and occupational stresses, and the psychological factors of the disease, fibrositis is considered to be a tissue reaction, of varying pathology, to several causal factors.

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