INTRADURAL SPINAL GRANULOMAS
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Intraspinal granulomas are rare, but they do occur, and are most frequently the familiar extradural tuberculous granulomas associated with Pott's disease of the vertebrae. Occasionally there may be no associated vertebral involvement. Non-specific granulomas in the extradural location, though less common, have also been described.

A granulomatous lesion of the dura mater itself, hypertrophic spinal pachymeningitis, was described in the 1870's by Charcot and his pupil Joffroy. This condition is most common in the cervical region, but may occur elsewhere in the spinal dura mater. It is usually non-specific, but occasionally is said to be of tuberculous or luetic etiology.

Intradural granulomas are very rare and may be intramedullary or extramedullary. They may be of syphilitic origin in the form of a gumma, which now is a rare lesion anywhere in the body, or tuberculomas, which again are quite unusual lesions, or they may be of other etiologies. As a granuloma represents an intense reaction on the part of the tissues to a specific or non-specific infection or irritation, the varieties of granulomas possible are manifold. Among these are: granulomatous tumefactions of pyogenic, other bacterial and mycotic (blastomycotic, coccidioidal, torular, trichophytic) origin, as well as those caused by the irritation of non-infectious agents such as injected iodized oils, dusting powders, surgical cotton and other foreign bodies. All of these varieties, however, have not been found intraspinally. The eosinophilic granuloma, while reported as causing symptoms of the central nervous system, is of secondary interest in this discussion in that symptoms produced would probably be those of extradural pressure secondary to the bony involvement.

DISCUSSION

Tuberculoma of the spinal cord or its leptomeninges is a rare entity. In a series of 6000 postmortem examinations of the central nervous system at the Philadelphia General Hospital, only 80 (1.3 per cent) cases of tuberculomas of the central nervous system over 1 cm. in diameter were found. Of these, 78 per cent were in negroes, 22 per cent in white people, 58 per cent in males and 42 per cent in females. There were 40 solitary lesions and 40 showed 2 or more granulomas present per case. In only 6 cases were there tuberculomas of the spinal cord, 3 solitary lesions and 3 associated with granulomas elsewhere in the central nervous system. Tuberculomas were found in the central nervous system in 1 out of every 53 cases that showed

evidence of tuberculosis elsewhere in the body. In 8 cases tuberculoma of the 
central nervous system was the only tuberculous focus anywhere in the 
body. The correct diagnosis of tuberculoma was made clinically in only 1 of 
the 80 cases.

The disease is seen in childhood or early adolescence rather than in later 
life in the majority of cases. The lesions vary in size from that of a small 
nodule a few mm. in diameter to that of a hen’s egg, and produce the symp-
toms of any other space-occupying mass in the same location in the spinal 
cord. They are usually associated with foci of tuberculosis elsewhere in the 
body and are probably secondary to these. These tumors may be intra-
medullary or extramedullary. The latter often arise from an infection of the 
spinal nerve roots, intervertebral cartilages, the epidural tissues, or the 
vertebral bodies. Peculiarly, the involvement of the spinal cord produces an 
inflammatory reaction of the pia arachnoid and dura mater, which may fuse 
all of these membranes into one thick mass and cause changes in the epidural 
space as well. These changes are most marked at the level of the tuberculoma 
and may include the adjacent nerve roots. The meningeal involvement may 
not spread far beyond the level of the principal lesion. It is very likely caused 
by the discharge of tuberculous debris into the subarachnoid space. On 
gross inspection, a round mass with concentric layers is found. It may show 
a softened, necrotic center with some surrounding edema, but the center 
may be calcified or, on rare occasion, present the findings of a true abscess.

In unusual instances the lesion may be removed with good results, as 
was shown by Elsberg’s patient who lived more than 8 years after operation, 
but usually a spreading tuberculous meningitis is the sequel.

Even when examined grossly and microscopically the differential diag-
nosis between tuberculoma and gumma may be uncertain. The differentia-
tion from coccidioidal granuloma may also present difficulties.

The gumma occurs as a late development of syphilis, but is rarely seen 
now. Formerly it was considered the process to be ruled out first and fore-
most in any case of suspected space-occupying lesion of the central nervous 
system. It is only a part of a more generalized disease process but may be 
the only manifestation thereof even to the extent of negative serological 
tests. Nonne (1913) stated, “In general, one can think of the possibility of 
a gumma whenever the symptoms of an extra- or intramedullary tumor are 
present.” Today the rare occurrence of a gumma in the spinal cord or its 
meninges has so altered the situation that gumma warrants only slight con-
sideration in the differential diagnosis of neoplasm of the spinal cord.

In a large percentage of the few cases in which the central nervous 
system is involved, the gumma starts in the meninges and may encroach 
upon the substance of the brain and the spinal cord or may remain entirely 
extraparenchymal. The lesion is usually solitary but may be multiple and 
vary in size from 1 to 2 mm. to 4 or 5 or more cm.

In the spinal canal the extension of the tumefaction produces a fusiform 
enlargement of the cord and associated structures with fine adhesions
between the meninges and the cord proper. The mass and cord fill the dural sac, become unusually soft, and produce the signs of a true tumor of the spinal cord. Extramedullary gummata form flattened masses which compress or encircle the spinal cord, giving rise to symptoms of compression, subarachnoid block and Froin's syndrome with its xanthochromia, high total protein, and spontaneous coagulation of the cerebrospinal fluid on standing. In other cases the meninges may be involved minimally, the process being limited to the nerve roots, along the course of which a number of small enlargements may be found.

Coccidioidal granuloma in man is a chronic, progressive, often fatal fungus disease caused by the Oidium coccidioides (Coccidioides immitis). The central nervous system and meninges are often involved. Coccidioidosis was first reported by Wernicke in 1892 and in the United States for the first time by Rixford in 1894. The first report of coccidioidal infection of the central nervous system was in 1910 by Ryfkogel; the patient was a child with coccidioidal meningitis. Dickson, in 1915, described a coccidioidal destruction of the lumbar vertebrae and involvement of the paravertebral musculature, but it was not until 1930 that coccidioidal granuloma involving the spinal cord was reported. At that time Rand reported 2 cases simulating true cord tumor. In 1 case the symptoms were those of compression at the level of the 6th thoracic vertebra with paraplegia and complete sensory loss below the 9th dorsal dermatome. Laminectomy revealed an extradural granuloma at the level of the 6th thoracic vertebra communicating through an opening between the 5th and 6th ribs, with a pocket of caseous material in the right chest. In the 2nd case there was a subdural granuloma completely encircling the cervical cord. In Rand's case the histological picture was very similar to that of tuberculosis with giant cells, lymphocytes, epithelioid cells and plasma cells, but the giant cells contained the double-contoured spherules of Coccidioides immitis. The presence of these parasites was the only reliable means of differentiation. In general, the disease may be primary in the skin, where it is usually confined as an ulceration which may become rather large. In other instances it may occur in the lungs and give the clinical picture of pulmonary tuberculosis, or in the skeletal system, again to be almost indistinguishable from tuberculosis. A diagnosis is made on the absence of tubercle bacilli, on microscopic examination, and the presence of the fungus. The spherules are scattered throughout the pathological tissues and many of them are taken up by the giant cells. In some instances the fungus is surrounded by eosin-staining club-like processes to form a well defined pseudo-rosette.

Reports on torular infections of the central nervous system emphasize the meningitis produced by Torula histolytica and largely disregard the gross granulomas. That torular granulomas do occur in the central nervous system was shown by Smith and Crawford in 1930 and by Goodhart and Davison in 1937. In 1944 Swanson and Smith reported 2 such granulomas simulating cerebral tumors. As for torular tumor of the spinal cord, as far as can be
ascertained only 2 cases have been reported. One of these (Smith and Crawford) involved the thoracic portion of the cord and the undersurface of the dura mater. The tumor measured $\frac{1}{4} \times \frac{1}{2} \times 1$ cm. Goodhart and Davison reported the 2nd case, an intramedullary granuloma.

The diagnosis, clinically, may be difficult, especially when the cerebrospinal fluid examination is negative in spite of a proven torular pulmonary disease. Whereas the spinal fluid may be entirely negative with the granuloma, in torular meningitis the organisms may be grown with ease even on routine culture media.

The gross appearance of the torular granuloma is quite characteristic, but at first glance it may resemble a glial tumor. Section reveals a multiloculated cyst, filled with a gelatinous material which does not empty itself when opened. In this way it is unlike a true cystic tumor and this is its most striking feature. The lesions may be multiple but single ones with findings resembling those of an isolated neoplasm do occur. There is a tendency to sharp demarcation from the adjacent structures and an apparent absence of reaction of the surrounding tissues. This constitutes another striking feature of the process. It would seem that surgical removal could be accomplished easily, but only rarely is there a true absence of an adjacent meningeal involvement. In addition, there is always the danger of opening the cyst and causing a rapidly spreading, purulent meningitis.

No cases of intradural pyogenic granuloma were found in the literature reviewed, but the possibility is obvious. The same statement holds for non-infectious foreign body granulomas. Alajouanine and Petit-Dutaillis did report an epidural granuloma occurring in a French soldier 16 years after a bullet causing paraplegia had been removed from the spinal cord. With the development of the granuloma the soldier again developed a paraplegia. Warren and Romano, Turnbull et al., and Watts and Mixter have reported intraspinal pyogenic granulomas but all were epidural. One case of Watts and Mixter presented minor arachnoidal involvement but this was apparently secondary to the dural involvement. Watts and Mixter pointed out the accompanying fever and leucocytosis as aids in the diagnosis of pyogenic granuloma simulating cord tumor, but the other observers made no mention of these points.

Various observers have reported instances of granulomas occurring in the central nervous system as the result of infestation with schistosoma. This is primarily a disease of the Far East and occurs in the Occident only in persons who have recently resided in the Orient, as with the former soldiers reported by Hunt, Abramson and Weaver, and by Swanson. Of the three varieties of schistosoma, S. japonicum involves the central nervous system much more frequently than do the others. This type is endemic in Japan, China, Formosa, the Philippines and the Celebes. Neurological complications caused by S. hematobium are extremely rare. This type is found exclusively in Africa (particularly Egypt) and causes urinary schistosomiasis. The third variant, S. mansoni, prevails in Africa, the West
Indies and northern South America. Instances of involvement of the central nervous system by this parasite are even more rare. It is said to favor the spinal cord. Müller and Stender presented a case of transverse myelitis of the dorsolumbar cord caused by S. mansoni. Gama and Marques de Sá reported a case of granuloma of the spinal cord caused by S. mansoni. There was a rapidly developing paraplegia in an otherwise healthy man of 42 years. A laminectomy revealed the true nature of the lesion, which was shown microscopically to be a granuloma containing the ova of S. mansoni. The past history of the patient revealed episodes of other symptoms suggestive of schistosoma infestation.

The possibility of this disease must be considered in the case of former service men or former residents of areas where the infestation is endemic. Episodes of other symptoms suggestive of such an infestation (rash, fever, diarrhea, abdominal cramps, stiff neck, changes in sensorium, muscular weakness, convulsive seizures, eosinophilia, positive intradermal tests and parasitic ova in the stools) may be of help in establishing the diagnosis. These findings, in all probability, will be of more value in retrospect than in foresight.

The cerebrospinal fluid findings are inconsistent, varying from normal to findings of slightly lowered sugar content, moderately elevated total protein, pleocytosis of slight degree and abnormal colloidal gold curves of the paretic type. The stool examinations may be negative for the ova of schistosoma. Eosinophilia may be absent.

Grossly, the granuloma varies in color from red purple to yellowish-gray and at times may resemble a glioblastoma multiforme. It may be of firm consistency and again may be quite soft. Microscopically, giant cells abound and contain the ova of the parasite. Plasma cells, lymphocytes and eosinophiles are found at the periphery. The center may be necrotic. The lesion may be a single granuloma or a coalescence of many smaller granulomas.

Surgical removal, if possible, should be followed by intensive therapy with the antimony derivatives, such as tartar emetic or Fuadin.

There are no clinical manifestations characteristic of compression of the spinal cord by granulomas that can be depended upon to differentiate them from those produced by a true neoplasm in the same location. To some degree, the compressive symptoms as produced by the granuloma may be overshadowed by manifestations of the myelitis or meningitis which may precede or accompany the granuloma. Again the possibility of a granuloma might be considered when evidence exists of a generalized disease process which might give rise to granulomatous formations. The symptoms vary greatly with the level involved and with the relation of the mass to the spinal cord, spinal nerve roots and meninges, as is obvious. Symptoms typical of a cauda equina tumor can be produced as shown by Ray who, in 1940, reported a case of gumma simulating such a lesion.

In the extramedullary location, the first symptoms will probably be those of an irritative sensory nature because of involvement of the posterior
roots. Eventually the complete picture of compression of the spinal cord with anesthesia and paralysis below the level of the lesion, alterations in the reflexes and in the function of the bowel and bladder will develop.

The granulomas themselves offer definite differential diagnostic problems, even when examined grossly and microscopically. A selective diagnosis of gumma, tuberculoma or other granuloma will often rest upon evidence or lack of evidence of one or another disease process elsewhere in the body.

Since the differential diagnosis between granuloma and neoplasm is virtually impossible clinically, the therapeutic course, which should always be followed closely, is obvious. To subject the luetic patient, for example, to a course of antiluetic therapy before considering operation is not wise for two reasons. First, the lesion may be neoplastic and thus not amenable to such therapy. Second, even though it be a gumma it may not respond to such treatment, while in the meantime the spinal cord will be further and possibly irreparably damaged by compression. The first step should always be laminectomy and decompression of the lesion and spinal cord. Later, antiluetic or other specific or supportive therapy may be administered.

Although, in the main, the prognosis can not be regarded as good with a group of diseases that are typified by widespread involvement of the body and for which (with the exception of syphilis) there is no specific therapy of dependable value, the case of intradural granuloma of the spinal cord reported here is incontrovertible evidence that adequate decompression of the cord by laminectomy and partial removal of the granuloma may, on occasion, permit the body to overcome the infection and go on to a satisfactory readjustment and clinical recovery from the disease.

The following case is unusual and of interest for several reasons. In this patient an intradural granuloma developed in intimate relation to the leptomeninges and the spinal cord. It was apparently tuberculous in origin although no other loci of tuberculous infection have been demonstrated. Most surprising of all, its partial removal has been followed by a complete recovery of the patient from all of the symptoms of involvement or compression of the spinal cord. In the 3 years that have elapsed since her operation there has been no evidence of extension of the lesion or of the infection. Specifically, a tuberculous meningitis did not develop.

CASE REPORT

L.O., a 44-year-old widow, referred by Dr. F. L. Jenkins of Chicago, was admitted to the Chicago Memorial Hospital on Feb. 6, 1946. She had been in good health until July, 1945, when she arose one morning with a "toothache-like pain" in the lower thoracic and upper lumbar region of her back. The discomfort lasted all morning and then disappeared, only to recur about 1 hour later to last for the rest of the day. This was a daily cycle for 1 week, after which the pain left her and never recurred. There were no accompanying symptoms.

Following this brief episode she was well until mid-November, 1945, when she noticed "numbness" and coldness of her feet. This peculiar feeling progressed upward
slowly and was accompanied about 3 weeks later by weakness of the legs. The weakness gradually progressed and was more marked in the right leg. Soon she required a cane and by Jan. 31, 1946, she became unable to stand or walk.

In late November, 1945, a band of burning sensation developed at the level of the umbilicus with radiation to the right and left around to the back. The pain was constant and was aggravated by twisting of the trunk. She denied having any dysfunction of bowel or bladder, but a uriniferous odor was noted during the examination. She denied ever having had syphilis or gonorrhea.

Examination. There was a slight bloody discharge from the cervical os, which showed minimal erosion. There was slight dorsal kyphosis.

On neurological examination the cranial nerves were entirely negative, as were the upper extremities. Hypalgesia and hypothermaesthesia were present bilaterally below the 10th thoracic dermatome. Vibratory sensibility was absent below the 2nd lumbar spine. Position and localization sense and two-point discrimination were normal in the upper extremities but absent in the lower. Deep pain sense in the tendo achillis was present. The lower extremities, particularly on the right, showed definite weakness in all movements and were spastic. Heel-to-knee tests were impossible. Beevor’s sign was positive. Knee jerks were hyperactive, the right more so than the left. Ankle jerks were absent, as were abdominal reflexes (obese, flabby, abdominal wall). Babinski, Chaddock, Gordon and Gonda signs were all positive bilaterally. There was no nuchal rigidity. Kernig, Lasègue, Patrick and Naffziger tests were negative.

Lumbar puncture in the 4th interspace revealed an initial pressure of 150 mm. of fluid and an almost complete block of CSF flow on bilateral jugular compression. Abdominal compression caused a rapid rise to over 200 mm. with a rapid fall following release. Four cc. of xanthochromic fluid were removed: there were 5 rbc/c.mm.; Pandy test 4+; total protein, 6800 mg.

Roentgenograms of the chest were not unusual. The thoracic spine showed a minimal degree of osteoarthritis. Measurements of the interpedicular spaces showed a widening of the spinal canal at the level of the 5th, 6th, 7th and 8th thoracic
vertebrae (Fig. 1). The pedicles otherwise appeared normal with no apparent evidence of atrophy or erosion.

A diagnosis was made of extramedullary tumor within the spinal canal causing compression of the spinal cord at the level of the 6th to the 8th thoracic vertebrae.

Operation. On Feb. 8, 1946, a laminectomy was performed. The dura mater bulged backward with the apex at the level of the 7th thoracic vertebra. On palpation a firm mass was found at this level. The dura mater appeared normal. Immediately beneath it was a grayish, avascular mass which was attached to the dura mater at the level of the 7th thoracic vertebra, but extended upward to the top of the 6th thoracic vertebra and downward to the lower limits of the 8th. This mass, however, was largest in the middle. It covered the spinal cord completely. At the upper limits the mass merged imperceptibly with a somewhat thickened, opaque arachnoid membrane. Looking up the spinal canal beneath the 5th thoracic vertebra one could observe an irregularly thickened, milky arachnoid membrane as far as it was possible to see. When this was incised, the spinal cord and pia mater appeared normal. The mass arose from the arachnoid membrane and was much thicker and larger on the right side. An incision was made through the arachnoid membrane along the posterolateral part of the spinal cord on the left side. The mass was then reflected toward the right off the spinal cord, to which it was attached by numerous dense adhesions which had to be divided by sharp dissection. As this dissection proceeded around the right side, the mass became more and more densely adherent to the cord, which was markedly compressed, particularly from the right side (Fig. 2). All of the mass lying posterior and to the right of the spinal cord was removed. It was obvious, however, that there was still some of the mass lying anterior to the cord, which was pushed backward sharply at the level of the 7th thoracic vertebra. This part of the mass was left in place as it could not be removed without seriously endangering the already compressed and contused spinal cord.

A frozen section revealed a granuloma of unknown etiology. A piece of tumor was taken for culture and the remainder sent to the laboratory for microscopic study. The usual closure, including closure of the dura mater, was then effected.

Laboratory Studies. On Feb. 9, 1946, the blood serological findings were reported as Wassermann negative, Kahn positive and Eagle weakly positive. The CSF Wassermann was negative and the Lange colloidal gold curve was 000123000. A second examination of the blood showed Wassermann and Kahn tests both negative. Still another examination was again negative. Similar tests previously made were also negative. During operation a sample of CSF had been taken from the subarachnoid space above the point of obstruction; Wassermann test on this was negative and Lange was 000123000.

Postoperative Course. There was very rapid recovery of motion of the lower extremities, but rather slower recovery of position and vibratory sense. By the 6th postoperative day she was moving both legs well; position sense was still very poor.
but vibratory sensibility was excellent. The patient was able to extend and flex the legs well at both hips and knees. On the 12th postoperative day she sat up with help, and on the 14th day she was discharged to her home.

Antiluetic therapy was then started by Dr. F. L. Jenkins pending further investigation of the true nature of the granuloma. Therapy was discontinued when the pathologist’s final report was received.

Pathological Report (Dr. Otto Saphir). There was a diffuse granulomatous reaction characterized by scattered small foci of necrosis surrounded by epithelioid cells. Numerous multinucleated cells of the Langhans type were noted and lymphocytes were abundantly interspersed throughout the section. Strands of hyaline and fibrous tissue bordered the section, and more fibrous tissue was noted between the granulomatous areas. Except for several vascular areas in one region, blood vessels were very sparse (Fig. 3). Ziehl-Nielsen staining failed to reveal any tubercle bacilli. Impression: Tuberculous granulation tissue.

Bacteriological Report. Smears made for acid-fast organisms were negative; 6 weeks postoperatively the laboratory reported a growth, on culture, of colonies of acid-fast organisms, which colonies the bacteriologist stated were not typically those of the tubercle bacillus. These organisms were then inoculated into a guinea pig. Later, on May 2, 1946, the laboratory reported that the guinea pig had died and on autopsy was shown to have many typical tuberculous lesions. This diagnosis was confirmed by Dr. Otto Saphir, the pathologist.

Course. The patient showed gradual improvement. When seen on Mar. 18, 1947, she walked without assistance. She stated that from the time of her discharge she had no trouble with her bladder and only occasionally had required an enema. She
began to be up and about on crutches in May, 1946, and began to walk unaided by mid-July, 1946. She pointed out the right 10th thoracic dermatome as the site of occasional burning pain which lasted a day or two. She was able to walk at least two miles with no difficulty.

Neurological examination showed a band of hypalgesia at the 9th thoracic dermatome on either side but sensibility was otherwise normal everywhere. A slight weakness in flexion of the left knee was present. The abdominal reflexes again were not elicited because of a thick abdominal wall. Beevor’s sign was negative. The right knee jerk was slightly more active than the left and the ankle jerks were absent bilaterally. The Babinski and other signs showed a normal plantar response.

Her gait was slightly wider based than normal and the left leg was favored ever so slightly. Romberg’s test was negative.

X-ray examination of the thoracic spine showed the laminectomy and increase in the density of the pedicles at the site of the former tumor. The interpedicular distance was decreased as compared to the preoperative measurements (Fig. 4). A blood Wassermann and Kahn, as well as urinalysis and blood count, all proved entirely negative. No lumbar puncture was made.

On Nov. 15, 1948, at the time of her last examination, she stated that she had had no difficulties whatever. Gait and station were entirely normal. No muscular weakness was demonstrated. The tendon reflexes were equal bilaterally and no pathological reflexes were found. The sensory examination was likewise negative.

Roentgenograms of the thoracic spine centering on the 7th vertebra showed no essential differences from the previous interpedicular measurements of March, 1947 (Fig. 4), but the pedicles themselves appeared better calcified and more convex on their medial surface. No lumbar puncture nor serological blood study was made.

Inquiry several months later disclosed that the patient had worked as a kitchen helper and waitress at a tuberculosis sanatorium some 10 years prior to the onset of her symptoms. In her work she had contact with the ambulatory patients who had their meals served in the dining room of the sanatorium. Frequent chest examinations, including roentgenograms, were made on all employees of that institution.
but the patient was never found to have any objective evidence of tuberculosis. Neither did she have any subjective symptoms. Inquiry as to social or family tubercular contact was negative. She has always lived in Iowa or in Illinois and has never left these states.

COMMENT

There are several confusing points about this interesting and unusual case. In general, the symptoms and findings are clearly explained by the lesion, except for one point. We do not understand why this lesion at the level of the 6th to 8th thoracic vertebrae, which gave rise to hyperactive knee jerks and Babinski’s sign, was associated with absent ankle jerks. Apparently the one examination of the blood in which the Kahn test was positive and the Eagle test weakly positive was false and misleading, as all other examinations for syphilis were repeatedly negative. It seems, therefore, unlikely that the antiluetic therapy begun postoperatively before it was finally concluded that the lesion was not luetic in origin played any role in the patient’s recovery.

If, as finally seemed most likely, the lesion is tuberculous in origin, there are several facts that are difficult to understand. It should be recalled that the granuloma was only partially removed, that it lay intradurally and in intimate contact with the leptomeninges and the spinal cord. In such circumstances, one would ordinarily expect a fatal tuberculous meningitis to develop shortly. Here, on the contrary, not only did no meningitis appear, but the patient made a complete recovery which has been sustained now, some 3 years after the operation. We have no explanation for these facts. If we refuse to accept the tubercle bacillus as the responsible agent we have no grounds for acceptance of any of the other less common causes of granuloma of the central nervous system. Furthermore, in spite of the unusual aspects of the case, a tuberculous infection is by all odds the most likely for the following reasons: (1) Tuberculosis is the most common cause of a granuloma of the central nervous system in a resident of the central United States. (2) This patient had had extensive exposure to tuberculosis. (3) The tissue was diagnosed as a tuberculous granuloma on the basis of its microscopic appearance. (4) Acid-fast organisms were grown in cultures of the tissue removed at operation. (5) These organisms produced an acute disseminated disease in guinea pigs which was diagnosed as being tubercular.

SUMMARY

A case of intradural spinal granuloma is presented. The patient had a gradually progressive paraparesis with complete obstruction to the flow of cerebrospinal fluid and greatly increased cerebrospinal fluid total proteins. Laminectomy revealed a granuloma compressing the thoracic spinal cord. This was partially removed and a complete recovery effected. Later investigation proved almost certainly that this was a tuberculoma.

The various types of granulomas occurring subdurally in the central nervous system and especially in the spinal cord are discussed.

The difficulty of differentiation of granulomas from tumors is stressed.

It is recommended that the lesion, in cases where the spinal cord is
involved by a granuloma, should be exposed by a laminectomy and as thoroughly removed as possible. Systemic treatment should not delay the decompression of the spinal cord but where such specific treatment is possible it should be carried out postoperatively.

REFERENCES


