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PHYSICIAN CONNECT

Dear Doctor,

Greetings from Micro Labs. We are happy to bring you "Physician Connect" which contains latest and interesting news.

This issue contains:

1. The effect of pulmonary tuberculosis on diabetic patients' immunological and metabolic characteristics
2. Correlation of patients Hypothyroidism, with dyslipidemia and protein contents with different metabolic disorders
3. A Systematic Review of the Effect of Alpha-lipoic Acid in the Treatment of Diabetic Neuropathy
4. A case of Streptococcus suis meningitis complicated by acute cerebral infarction.

Should you require any article or, medical information, please feel free to contact us at medicalservices@microlabs.in

Looking forward to your valuable feedback.

Best Regards,

Medical Team, Micro Labs Limited.

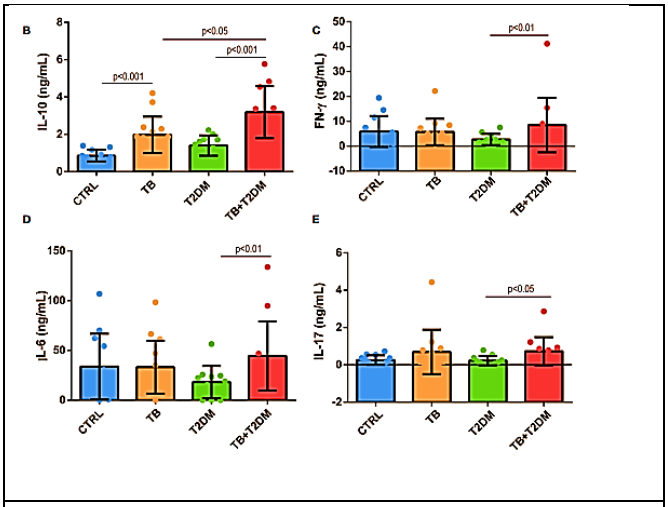
The effect of pulmonary tuberculosis on diabetic patients' immunological and metabolic characteristics

There are approximately 10 million patients with pulmonary tuberculosis (PTB) worldwide, with China accounting for 8.4%. Notably, diabetic patients are at a higher risk of PTB infection than non-diabetic subjects. Clinical data show that patients with diabetes and PTB have sustained hyperglycemia and prolonged pulmonary infection compared to patients with PTB only, all of which impair drug treatment and lead to a significant increase in recurrence rate and mortality. As a result, understanding the mechanisms by which diabetes drives PTB progression is critical for therapeutic purposes. [1] Lung-resident macrophages function as innate immune cells that protect against infection. In the lungs, clearance is primarily mediated by phagocytosis and inflammatory cytokines. Pathogen-associated molecular patterns (PAMPs) on PTB allow foreign pathogens to be recognised by receptors expressed on resident macrophages. NLRs (nucleotide oligomerization domain-like receptors) are cytosolic pattern recognition receptors that recognise PTB PAMPs after phagocytosis, activating inflammasome formation. PTB also expresses Toll-like receptor (TLR) ligands. TLR-2, TLR-4, and TLR-9 are known to be involved in recognition among all TLR ligands. The innate immune response is propagated further downstream of the TLR signalling pathways. For example, interleukin (IL) is one of the main cytokines involved in anti-tuberculosis immunity regulation. [2] Patients with T2DM alone, PTB alone, and PTB co-morbidity with T2DM were enrolled in the study to answer this question. As controls, healthy people were used. The fasted plasma samples were subjected to panels of cytokine/chemokine measurements using the Quantibody Human Inflammation Array. The relationship between the cytokines of interest and the features of the diseases was investigated.

This study enrolled patients with T2DM (n=12), PTB (n=14), or PTB combined with T2DM (n=12). Twelve healthy people served as controls. All study participants were age and gender matched. Basic information was gathered, such as medical records, gender, age, body weight, and height. Body mass index (BMI) was calculated by dividing weight in kilogrammes by height in metres squared. A systolic blood pressure of at least 140 mmHg, a diastolic blood pressure of at least 90 mmHg, or the use of antihypertensive drugs was considered hypertension. T2DM was defined as a fasting blood glucose level of at least 7.0 mmol/L or a blood glucose level of 11.0 mmol/L or higher after 2 hours of oral administration of 75-g glucose. When any of the following three conditions were met on sputum samples prepared by direct smear, PTB was diagnosed: (1) two sputum samples were acid-fast bacilli positive under microscopic examination; (2) one sputum sample was acid-fast bacilli positive on microscopic examination, and signs of active pulmonary tuberculosis were found on pulmonary imaging examination; and (3) one sputum sample was acid-fast bacilli positive on microscopic. All patients had fasting peripheral blood samples taken. After centrifugation at 3000 rpm for 10 minutes, the serum was collected and the supernatant was stored at -80°C. Following the manufacturer's instructions, equal amounts of Fried Ewald plasma samples were subjected to cytokine/chemokine measurements. Chip drying, gradient cytokine

standard dilution, serum sample dilution, chip sample addition, cleaning, detection of antibody mixture incubation, and other procedures were carried out.

Patients with T2DM and PTB have higher fasting blood glucose levels and monocyte counts in circulation than those with T2DM alone. Among the four groups, patients with T2DM and PTB had the highest levels of circulating IL-10. In these patients, univariate linear analysis revealed that serum IL-10 levels were positively associated with myeloid cells but negatively associated with lymphocyte counts. Patients with T2DM plus PTB had 1.6-fold higher serum IL-6 levels than those with T2DM alone.



Serum levels of IL-10, IL-6, IL-17 and IFN-gamma among all groups. (A) PCA plot. (B–E) serum levels of IL-10, IFN-g, IL-6 and IL-17 in the study subjects. CTRL, healthy controls; PTB, pulmonary tuberculosis; T2DM, type 2 diabetes mellitus; PTB+T2DM, pulmonary tuberculosis combined with type 2 diabetes mellitus.

The study's main findings are summarised below: (1) Patients with PTB had low BMI and skewed myelopoiesis, as evidenced by increased myeloid cell number but decreased lymphocyte count when compared to healthy controls. Furthermore, serum triglyceride levels were higher, but total cholesterol and HDL-c levels were lower in PTB patients compared to healthy controls. (2) When combined with T2DM, patients with PTB had increased myelopoiesis and lower total cholesterol and HDL-c levels compared to T2DM alone. As shown in Table 1, T2DM combined with PTB resulted in greater hyperglycemia and liver dysfunction than T2DM alone; and (3) Serum IL-10 levels peaked in patients with T2DM combined with PTB among all groups. Taken together, these findings suggest that the presence of PTB reduces T cell-mediated immune responses while promoting the progression of skewed myelopoiesis and metabolic disorders in T2DM patients.

Obesity, lipid disorders, and low-grade chronic inflammation were all symptoms of T2DM. A growing body of evidence suggests that immune cells are the primary cause of metabolic disorders and inflammation, which lead to poor vascular outcomes. Diabetes patients and mice have consistently shown an increased ratio of myeloid cells to lymphocytes, which increases with disease progression. Furthermore, GM-CSF and IL-6 intracellular levels were higher in db/db bone marrow cells than in age-matched wild-type controls, indicating a pathological basis for skewed myelopoiesis. In line with this, infiltrating macrophages produce cytokines such as IL-1b, which promotes b cell dysfunction under chronic inflammatory conditions. [3]

PTB replicates in alveolar macrophages and then spreads to macrophages, myeloid dendritic cells, and neutrophils recruited from the periphery to promote phagocytosis. The activation of complementary component C3 improves M. tuberculosis adherence and uptake, allowing mononuclear phagocytes and B-lymphocytes to be activated for antibody production. Antigen-specific T-cell clones are expanding and migrating to the lungs in response to

antigen-presenting cells. To combat PTB, the innate and adaptive immune systems work in tandem. [4]

After infection with M. tuberculosis, peripheral blood monocytes aggregate in the alveoli and develop into macrophages. As a result, interleukins are produced, which play a role in the protective immune response of the body. [5]

In this study, patients with T2DM and PTB had significantly higher serum IL-10 levels than those in the other groups. IL-10 is secreted by a variety of cells in the body and has the ability to effectively regulate the function of monocytes/macrophages, act as a negative regulator of cell-mediated immune response, and inhibit the production of pro-inflammatory factors by monocytes and macrophages.

The serum level of IL-10 in PTB patients is higher than in healthy controls, which inhibits the production of pro-inflammatory factors by monocytes and macrophages, weakens macrophage antigen presentation, and reduces immune clearance of M. tuberculosis. In T2DM, IL-10 is associated with an inflammatory response and insulin resistance, but it is unclear whether higher IL-10 levels prevent T2DM by reducing the production of pro-inflammatory cytokines, or whether higher IL-10 levels in T2DM patients lead to a compensatory response to increased pro-inflammatory mediators (mainly tumour necrosis factor-a and IL-6)

In conclusion, the presence of a PTB infection maintained greater hyperglycemia and monocyte numbers in patients with T2DM than in patients with only T2DM. When patients are infected with PTB, the treatment of T2DM becomes more complicated and difficult. Better precision medicine-based therapies are needed to control hyperglycemia and inflammation in patients with T2DM and PTB.

When compared to PTB or T2DM alone, PTB infection in patients with T2DM had distinct inflammatory profiles and sustained hyperglycemia. Patients with T2DM who are infected with PTB may have elevated IL-10 levels and monocyte counts.

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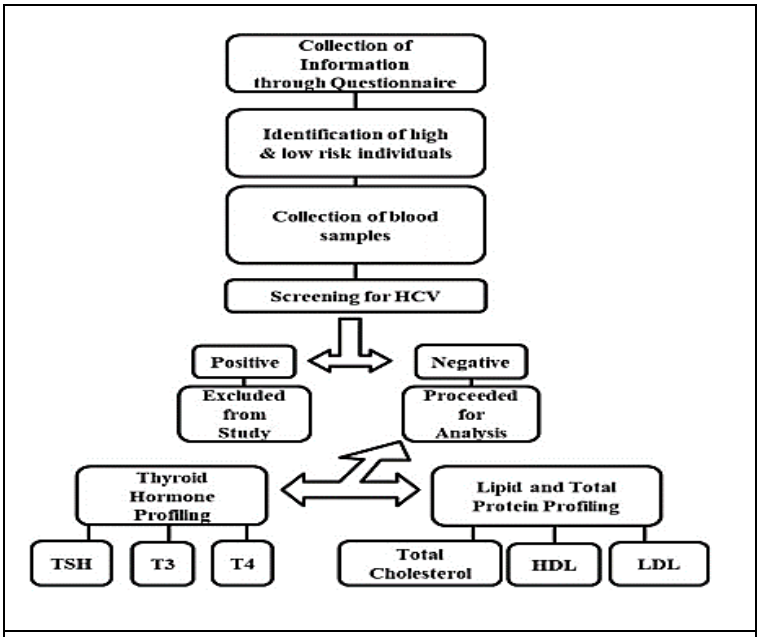
Correlation of patients Hypothyroidism, with dyslipidemia and protein contents with different metabolic disorders

Thyroid hormones (THs) regulate nutrient metabolism, blood sugar, cholesterol, pregnancy, and menstruation, among other things. Thyroid-related abnormalities have been studied in over 110 countries, with 1.6 billion people identified as being at risk of developing thyroid disorders. The prevalence of thyroid-related disorders differs greatly between men and women. Similarly, the severity of thyroid disorders varies with age. [1] Cholesterol is divided into three types: low-density lipoprotein (LDL), high-density lipoprotein (HDL), and very low-density lipoprotein (VLDL). Dylipidemia is a condition characterised by elevated lipid levels in the blood. Thyroid problems contribute to dyslipidemia. [2] Diabetes is increased by irregular thyroid levels. Diabetes and thyroid abnormalities are thus considered risk factors. Thyroid disorders are significantly more common in people with diabetes, ranging from 6.9% to 16%. The primary goal of this study was to look for any abnormalities in the levels of thyroid hormones (TSH, thyroxin (T4), and triiodothyronine (T3)), lipid profile parameters (TC, LDL, and HDL), and total protein (TP) in patients with various metabolic disorders.

The study's aim was to analyse the study parameters, which included enzyme-linked immunosorbent assays, Bradford assays, and standard clinical kits and methods. The data was analysed using the appropriate statistical tests.



Methodology flow diagram of the study.

Thyroid-stimulating hormone, total cholesterol, and low-density lipoprotein were all elevated in all subjects except those with hypotension, while triiodothyronine levels were decreased. Thyroxin levels were lower in people with diabetes and symptoms of thyroiditis, but higher in people with hypertension. High-density lipoprotein was upregulated in diabetic men only, while total protein was downregulated in hypotensive men only. Dylipidemia was associated with hypothyroidism in patients with diabetes, symptomatic thyroiditis, and hypertension. It was associated with total protein in hypertensive subjects.

Similar to other studies, the findings of this study indicated a significant increase in TSH and a decrease in T4 and T3 levels. According to previous research, the study found significantly elevated TC and LDL levels. [2]

According to the findings of this study, most men and women with hypotension had euthyroidism, but only one woman with hypotension had unusual hypothyroidism due to high

levels of THs (T3 and T4) and elevated TSH. Previous research has found that hypothyroidism develops in older patients (>65 years) with hypotension, but the patients in the study were not older and did not frequently experience hypothyroidism. [3]

A previous study found similar results to the current study, indicating that TC and LDL levels fluctuated instead of HDL in hypothyroid patients. Other studies, like this one, found that patients had higher TSH, TC, and LDL levels than healthy controls, but normal HDL levels. [4]

This study found similar results, with postmenopausal women exhibiting no significant change in TSH levels. An earlier study found that pre- and post-menopausal women had higher TSH levels, but the current study found no significant change in TSH levels. Another group discovered higher TC and LDL levels in postmenopausal women, as observed in the current study. Another group of researchers, however, discovered high levels of LDL and HDL. Similarly, a previous study found that HDL was negatively correlated with TSH in postmenopausal women. [5]

The levels of the studied parameters in healthy subjects were within the internationally recognised standard ranges. Except for those with hypotension, the TSH level was increased in all patient groups studied. T4 levels were lower in diabetics and those with symptomatic thyroiditis, but higher in those with hypotension. Except for those with hypotension, T3 was downregulated in all of the subjects studied. Except for patients with symptomatic thyroiditis, all subjects had elevated TC levels. LDL followed a similar pattern and was discovered to be upregulated in all subjects studied. Only men with diabetes had higher HDL levels. Only subjects with hypotension had lower TP. TSH was found to have a strong positive correlation with TP in men with diabetes, as well as TC and LDL in men with symptomatic thyroiditis. TSH was discovered to have a strong positive correlation with T4 in female hypotensive subjects but a strong negative correlation with the same parameter in men with diabetes and women with hypertension. In diabetic men, T4 had a strong positive correlation with T3 and a strong negative correlation with TP. T3 was found to have a strong positive correlation with LDL in diabetic female subjects and HDL in postmenopausal women.

The current study found a link between dyslipidemia and hypothyroidism or hyperthyroidism in people with various diseases, but it should be expanded in the future to include a larger number of people with each type of metabolic disorder. Furthermore, this research should be expanded to investigate the underlying mechanisms and processes. The findings can help to improve disease management and reduce associated complications.

In patients with various metabolic disorders, this study discovered a correlation between hypothyroidism, dyslipidemia, and total protein.

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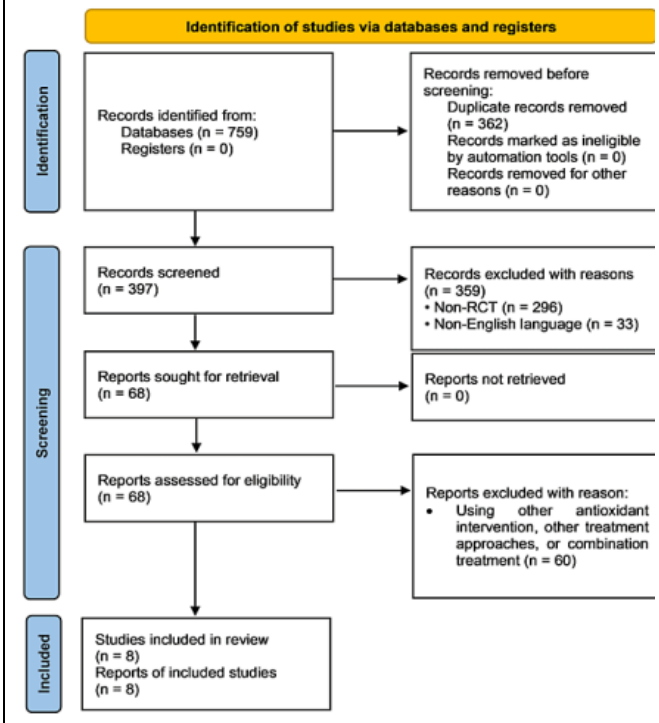
A Systematic Review of the Effect of Alpha-lipoic Acid in the Treatment of Diabetic Neuropathy

Diabetic neuropathy, also known as peripheral neuropathy, refers to a group of clinical pathologies caused by peripheral nervous system dysfunction in diabetic patients. The most common presenting symptoms are numbness, tingling, and burning in the extremities, with estimates ranging from 6% to 51% among adult patients with diabetes. [1] Although the precise cause of diabetic peripheral neuropathy is unknown, several studies have proposed underlying pathologies such as metabolic, neurovascular, and autoimmune mechanisms. The most widely accepted theory is that hyperglycemia causes oxidative stress in the mitochondria, which leads to hyperglycemic damage. As a result, endothelial and neuronal cells are damaged, compromising oxygen and nutrient supply to the nerves. [2] Neuropathic pain is difficult to treat because most standard analgesics do not provide adequate pain relief. The medication route of treatment primarily includes antidepressants, anti-epileptics, and opioids, with tricyclic antidepressants, serotonin-noradrenaline reuptake inhibitors, and anticonvulsants that target calcium channels serving as first-line therapies. Topical agents such as capsaicin and lidocaine may also be used. Recently, antioxidants such as flavonoids and alpha-lipoic acid (α-lipoic acid) have been proposed as effective treatments for diabetic neuropathy. Given the growing body of evidence supporting the role of α-lipoic acid in the treatment of diabetic neuropathy, the purpose of this systematic review is to assess the current literature and make recommendations for future research. The emphasis is on symptom reduction and the incidence of adverse events following α-lipoic acid administration in this population.



Reporting Items for Systematic Reviews and Meta-Analyses flow diagram.

The main goal of this systematic review is to evaluate the efficacy of α-lipoic acid in the treatment of diabetic neuropathy. Several secondary goals are also assessed, including the occurrence of adverse effects after α-lipoic acid administration. The following inclusion criteria set the parameters for study selection: (1) a study population of diabetes mellitus patients with neuropathic pain, (2) randomised controlled trials (RCTs) looking into α-lipoic acid, (3) and an appropriate comparison was made in the study Studies that were published in a language other than English were excluded. A thorough evaluation of the titles and abstracts of the database search results yielded studies that were independently identified for inclusion in the review. In accordance with the PRISMA recommendations, a data extraction form was created. This enabled the extraction of all relevant data from the reviewed literature. This included the author(s), year of publication, patient population, intervention, comparison, study period, outcome measures, results, and any conclusions drawn based on the evidence provided about the administration of α-lipoic acid to diabetic patients with peripheral neuropathy symptoms.

This systematic review looked at eight studies that included 1,500 diabetic patients. The literature on the effectiveness of α-lipoic acid in the treatment of diabetic neuropathy was inconsistent, with three trials (37.5%) observing significant improvements in symptoms and five trials (62.5%) not observing any notable results. α-lipoic acid was found to be a safe and tolerable intervention in all studies, with no reported adverse effects.

The purpose of this systematic review was to assess the efficacy and safety of α-lipoic acid in the treatment of neuropathic pain in diabetic patients. We discovered that administering α-lipoic acid is a safe and tolerable alternative intervention for the treatment of diabetic neuropathy. However, significant findings were only found in three of the trials included in this review, with the remaining literature failing to provide any noteworthy evidence. As a result, more research is needed to confirm or refute the hypothesis that α-lipoic acid is an effective intervention for the treatment of diabetic neuropathy.

A trial involving 148 type 2 diabetics that looked at the effect of α-lipoic acid as a combination treatment on diabetic peripheral neuropathy provided evidence in this regard. Following the administration of a combination of treatments, including αα-lipoic acid at 600 mg/day, gliclazide, sodium-glucose-linked transporter 2 inhibitors, metformin, and glucagon-like peptide 1 analogues, the peripheral neuropathy development score decreased significantly in all participants. The treatment was closely monitored for eight months. These findings highlight the beneficial effect of α-lipoic acid when used as part of a combination regimen in type 2 diabetes patients experiencing neuropathic pain. [3]

Based on the majority of the evidence in this systematic review, we conclude that using αα-lipoic acid alone does not provide a significant improvement in the treatment of neuropathic pain in diabetic patients. It is, however, regarded as a safe and tolerable treatment option that may result in some neuropathic symptomatic relief. Given its notable efficacy and good safety profile, future trials could include this intervention in combination with current treatments for diabetic neuropathy.

The administration of αα-lipoic acid may result in symptom reduction and provides a safe and tolerable treatment option. However, there is limited evidence to support the positive outcomes of this approach. Further research is needed to confirm or refute the hypothesis that α-lipoic acid is an effective intervention for the treatment of diabetic neuropathy.

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A case of Streptococcus suis meningitis complicated by acute cerebral infarction.

Streptococcus suis is an animal pathogen that can cause acute infectious diseases in humans and animals, primarily through wound infection. It is most common in Southeast Asia and is the primary pathogen responsible for meningitis, hearing loss, skin damage, and bacteremia. [1] Streptococcus suis virulence genes include the capsular polysaccharide gene, the lysozyme releasing protein gene, the hemolysin gene, the cytokine gene, and the glyceraldehyde-3-phosphate dehydrogenase gene. These virulence genes are important predictors of Streptococcus suis virulence. [2] Streptococcus suis infection with acute cerebral infarction was identified. It states the following:

A 62-year-old farmer was admitted due to a headache and fever that lasted 8 days and worsened for 5 hours. The patient had a headache and a fever of 38.0 degrees Celsius. Binaural tinnitus, vomiting, right upper limb lifting pain, limited movement, and weakness of the left lower limb are all present. He had gone to a different hospital for treatment. MRI of the right shoulder joint revealed effusion in the descending capsule of the right acromion, the joint cavity of the right shoulder, and around the right biceps brachia tendon. The anti-inflammatory and analgesic treatment had a negative effect. Prior to admission, a severe headache, sweating, hearing loss in both ears, poor consciousness, and indifferent response occurred. On admission, the temperature was 38.2 degrees Celsius. Narcolepsy, binaural hearing impairment, grade 4 muscle strength in the left lower limb, four transverse fingers from chin to chest, and positive bilateral Kernig's and bilateral Brudzinski's signs. The patient works as a farmer, primarily cultivating fields and raising livestock. His left index finger had been scratched by trees prior to the onset, resulting in a 0.5 cm swollen wound. Streptococcus mitis was isolated from blood and cerebrospinal fluid cultures. Streptococcus suis typeII was discovered through the analysis of the second generation sequencing detection of pathogenic microbial infection in cerebrospinal fluid samples.

On the fluid attenuated inversion recovery (FLAIR) sequence, an enhanced MRI scan of the brain on the 14th of August revealed multiple cord-like slightly high signal shadows in the sulci of both cerebral and cerebellar hemispheres. On the enhanced scan, the pia mater was thickened and significantly enhanced, as seen in the flair enhanced series. The right semioval centre and basal ganglia saw spotted abnormal signal shadow, long T1 and long T2 signal, flair showed high signal, diffusion weighted imaging (DWI) showed high signal, the corresponding apparent diffusion coefficient (ADC) value decreased, the boundary was unclear, and there was no enhancement on the enhanced scan, according to brain MRI.

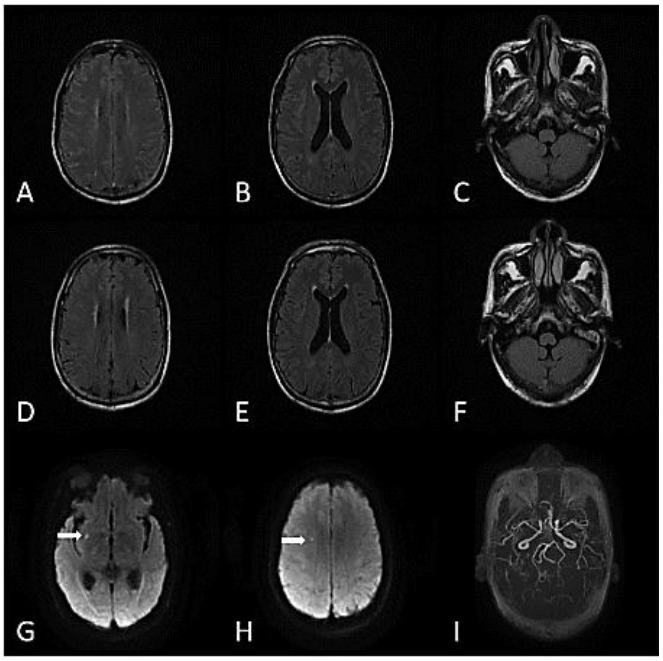

Figure 1. Imageological examination. FLAIR sequence of brain magnetic resonance imaging (before treatment (A, B, C) - after treatment (D, E, F)). DWI sequence and MRA of brain magnetic resonance imaging (G, H and I).

Acute lacunar infarction was a possibility. Magnetic resonance angiography (MRA) of the brain revealed no obvious abnormalities. After treatment, a brain MRI enhanced scan revealed that the pia mater thickening had decreased (Figure 1). Following admission, the patient was given empirical anti-infection treatment with cefotaxime sodium and sulbactam sodium. During this time, the temperature ranged from 38.5 C to 39.0 C. To reduce intracranial pressure and inflammation, dehydrant and dexamethasone therapy were used. The highest priorities of cerebral infarction treatment were antiplatelet aggregation, lipid regulation, and plaque stabilisation therapy. To increase blood supply to the ischaemic focus and nourish nerves, blood circulation drugs and cerebroprotein hydrolysate were used.

In addition, gastric mucosal protection, expectorant treatment, and nutritional support were given. Streptococcus mitis was found in the blood and cerebrospinal fluid bacterial cultures. Suppurative meningitis was the preliminary diagnosis. Meropenem was the improved antibiotic. The temperature had returned to normal. The patient's headache and neck stiffness were significantly reduced. The antibiotic was reduced to ceftriaxone sodium injection, and anti-infection therapy was continued until discharge. At the time of discharge, the patient had no headaches and grade 5 muscle strength in the left lower limb. Although no pathogenic microorganisms were discovered in the cerebrospinal fluid, he was left with severe binaural nerve deafness. In telephone follow-ups one and three months after discharge, the patient reported no special discomfort and the same binaural hearing loss as before.

Streptococcus suis can be isolated from sick pigs or the nasal cavity of healthy pigs. There are two types: type I only infects pigs and does not infect humans, and type II can infect and cause disease in humans. It primarily causes suppurative meningitis, which is frequently

accompanied by involvement of the eighth cranial nerve, manifesting as ataxia and deafness, with a 50%-70% incidence.

After coming into contact with livestock, the patient developed skin damage and disease. The patient suffered from severe hearing loss, which was caused by the pathogen entering the peripheral lymph from the subarachnoid space via the cochlear aqueduct. [3] When patients with bacterial infections have hearing loss, Streptococcus suis infection should be considered. Dexamethasone treatment, when administered on time and in sufficient quantities, can effectively improve the hearing impairment caused by Streptococcus suis infection. According to one study, the occurrence of hearing impairment in Streptococcus suis patients is not only related to the use of glucocorticoids, but may also be dependent on antibiotic use. The use of ototoxic drugs must be avoided. [4]

Dexamethasone was first used in this case at the time of admission. The treatment lasted more than ten days. There was no use of ototoxic antibiotics throughout the procedure, but there was still significant hearing loss. As a result, it is hypothesised that the hearing impairment caused by Streptococcus suis is unrelated to the use of ototoxic antibiotics, and the effect of hormone on improving hearing loss remains unknown. In addition to severe hearing loss, this patient had an acute cerebral infarction, which had not previously been reported. Patients infected with Streptococcus suis were reported to have sequelae such as deafness with mental retardation, hearing impairment, and sluggish response. [5] Other neurological sequelae may be related to cerebral ischemia, based on this case and relevant literature. Severe inflammation can cause hypoxia and necrosis. This case shows that Streptococcus suis meningitis can cause ischemic stroke.

Streptococcus suis infects humans with an acute onset and rapid progression. Identifying the pathogen as soon as possible and choosing effective antibiotics for treatment can help control the disease quickly, but more clinical research is needed to improve the sequelae and provide a more effective treatment scheme.

A case of Streptococcus suis-induced suppurative meningitis complicated by acute cerebral infarction was reported to provide a reference for the diagnosis and treatment of Streptococcus suis infection. The diagnosis, treatment, follow-up, and epidemiological materials in a case of suppurative meningitis complicated by acute cerebral infarction caused by Streptococcus suis were reviewed, as well as the relevant literature. This case's clinical manifestations were headache and fever, which progressed quickly. The patient improved and was discharged from the hospital after receiving effective anti-infection treatment, but he had severe hearing loss.

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