Tuberculosis in the pediatric age group: An emerging disease - Imaging findings

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Learning objectives

To define the radiological findings and establish the differential diagnosis of the tuberculosis in the pediatric age group in the respiratory system, the central nervous system (CNS), the muscle-skeletal system, abdominal and head-neck locations.

Images for this section:

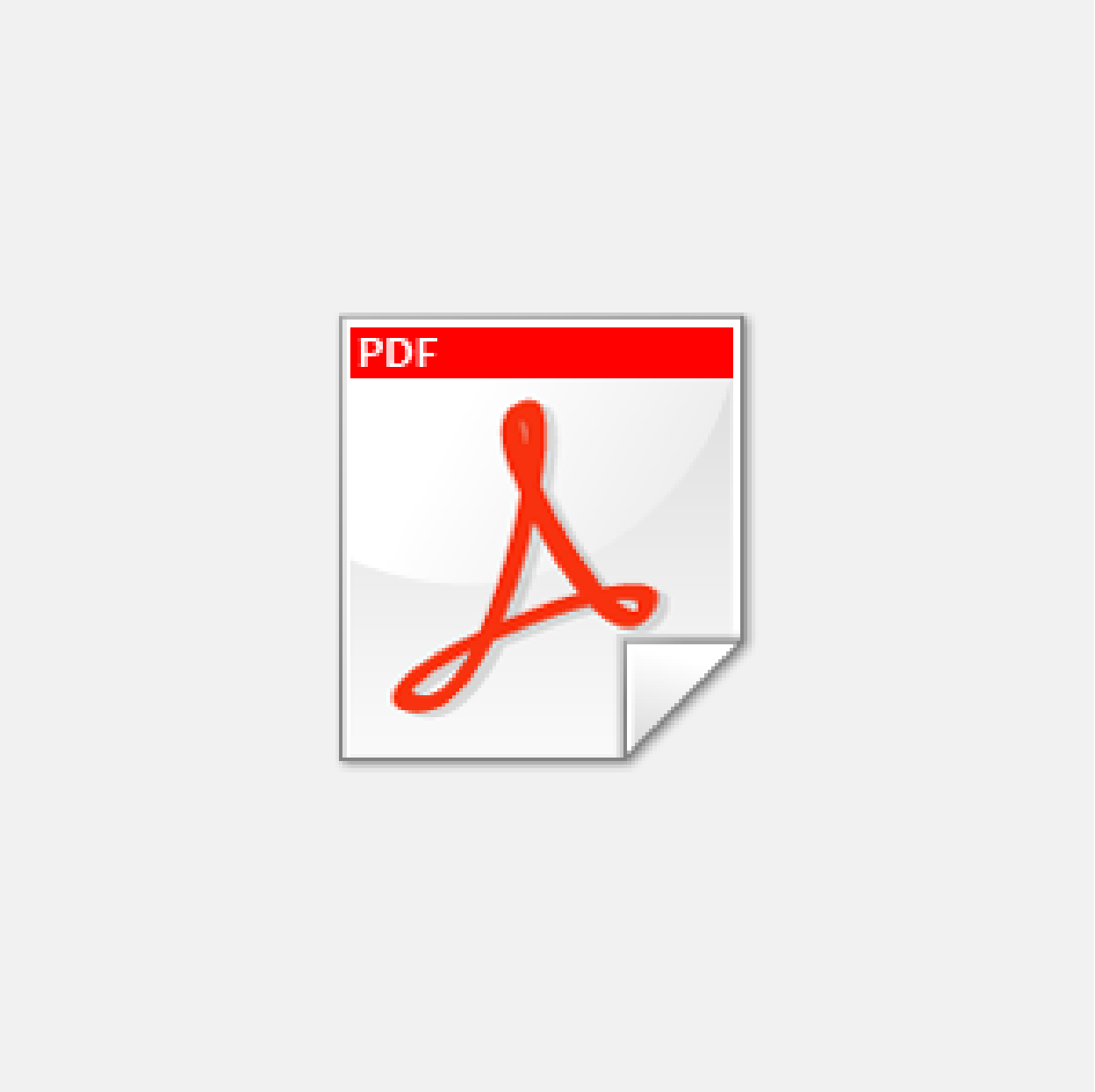

Fig. 1: Complete EPO in pdf.

Background

Tuberculosis is reappearing in non-endemic population due to the increase of immigration, immunosupression and resistance to the treatment. It presents a high rate of morbidity if an adequate and early treatment is not used.

The prevalence of the tuberculosis infection in our community is increasing. We find in our region a rate of 13/100 thousand population in patients of 0-5 years-old and 10.3/100 thousand in 5-10 years-old.

Infection by TBC shows a great variety of clinical finds and through images that can simulate a big number of diseases. Even though the thorax is the area most frequently affected, the disease may affect any other organs.

Therefore, it is important to become familiar with the most characteristic radiological findings in this entity.

We show different samples collected from May 1999 to September 2009 and their radiological characteristics in simple x-ray, ultrasound (US), TC and MR.

Imaging findings OR Procedure details

Tuberculosis usually affects the respiratory system. However, it can appear in any organ system, especially in immunocompromised patients.

PULMONARY TUBERCULOSIS.
It has been divides into primary and postprimary tuberculosis.

PRIMARY PULMONARY TBC

It is seen in patients with no previous exposure to M. tuberculosis.It is the most common form in childhood (higher prevalence in children less than 5 years-old).It is radiologically distinct from postprimary tuberculosis (the most common form in adults).
Simple Rx is normal in up to 15% of patients with proved tuberculosis.

At radiology, primary tuberculosis can manifests as four main entities:
- Parenchymal disease.
- Lymphadenopathy.
- Miliary disease.
- Pleural effusion.

Parenchymal disease (Figure 1 on page 11)
It manifests as dense and homegeneous lobar consolidation (with predominance in the lower and middle lobes).
It is often indistinguishable from that of bacterial pneumonia, however,in TBC there is limphadenopathy and lack of response to conventional antibioctics).
In children under 2 years of age, lobar or segmental atelectasis can appear, most seen in the anterior segment of an upper lobe or the medial segment of the middle lobe.
There is a total resolution without sequelae in approximatey two-thirds of cases, though it can take up to 2 years. In the remaining cases, a radiologic scar persists and can calcify (in 15% of cases, known as Ghon focus). In 9% of cases tuberculomas can be seen (they can cavitate and undergo calcification).

Lymphadenopathy (Figures 2 on page 12)
They are seen in up to 96% of children. It is typically unilateral (bilateral in about one-third) and right sided, involving the hilum and right paratracheal region.
Any nodes greater than 2 cm in diameter usually have a low-attenuation center due to necrosis and are highly suggestive of active disease.
They can be associated with other manifestations of TBC, though they can be the unique radiographic finding (especially in children).
CT is more sensible than chest radiography.
With treatment there is slower resolution than parenchymal disease and they usually calcify (6 months or more after the initial infection).
It is frequently seen a Ghon focus with calcified hilar node (Ranke complex).

Miliary disease (Figure 3) on page 13

It affects between 1-7% of patients with all forms of tuberculosis. It is usually seen in the elderly, infants and immunocompromised patients, and it manifests within 6 months of initial exposure.
Chest radiography is usually normal, and the classic imaging finding are diffuse small 2-3 mm nodules with lower lobe predominance. HRCT is more sensible than conventional chest Rx.
The nodules resolve in 2-6 months with treatment, and they can coalescence to form consolidations.In 2/3 of cases they associate pleural effusion, peritonitis or meningitis.

Pleural effusion (Figure 4) on page 14

It is seen in approximately 25% of patients with proved primary tuberculosis The effusion is often the sole manifestation of TBC and usually manifests 3-7 months after initial exposure.

It is a very uncommon finding in childhood.

It is unilateral and complicantions are rare (empyema, fistula, bone erosion).

Residual pleural thickening and calcification can result.

Ultrasound demonstrates complex septated effusion.

CNS TUBERCULOSIS

It occurs in 5% of patients with TBC. It results from hematogenous spread.

CNS tuberculosis can manifest in different forms: meningitis, tuberculomas, abscesses, cerebritis adn miliary tuberculosis.

Parenchymal tuberculosis (Figure 5) on page 15

The most common form is the tuberculoma.

The etiology is Hematogenous spread and the pathological anatomy are granulomas with central caseous necrosis (tuberculoma).

Intracraneal tuberculoma is uncommon in developed countries, though it can be seen in immigrants from endemic areas and immunocompromised patients.
It can develop in all ages.

Location: In children, cerebellum is the most frequent location, it appears in cortical and subcortical location. Tuberculomas are usually solitary. Multiple tuberculomas are found in 10-35% of cases.

CT findings:
- Acute stage: Hypodense area due to cerebritis.

- Immature tuberculomas: Iso or hyperdense with rim enhancement, nodular or irregular after contrast.

- Mature tuberculomas: Round or oval masses well defined, with rim enhancement. "Target sign": central calcification or punctate enhancement with surrounding hypoattenuation and rim enhancement.

- Cured tuberculomas usually calcify.

MR imaging:
Tuberculomas: Isodense images in T1. Central hyperintense area with hypointense rim in T2 and enhancement after contrast.

Solitary tuberculomas can not be distinguished from encapsulated abscess, malignant astrocytoma or metastasis.

Tuberculous meningitis (Figure 6) on page 16

It represents less than 5% of bacterial meningitis in children.
It is caused by miliar seeding, with granulomatous meningitis of the basal cisterns. Meningeal involvement is due to cortical tuberculomas rupture to subarachnoid space.

Inflammatory changes can induce a lenticulostriate and thalamoperforating arteries vasculitis, causing basal ganglia and thalamic infarcts.

Etiology: Hematogenous spread from pulmonary tuberculosis to the meninges.

Pathological anatomy: Caseous necrosis, chronic granulomas, endarteritis and perivascular inflammatory changes. Infarcts (¾ parts of infarcts are located in regions irrigated from medial lenticulostriate and thalamoperforant arteries. 70% of cases are bilateral infarcts).

Hydrocephalus and enhancement of basal cisterns.

Most of children with tuberculous meningitis also have CNS miliar infection (11% have parenchymal and meningeal lesion).

Location: There is predilection for the basal cisterns.

Natural history: Total mortality rates of 25-30%.

Sequelae: pachymeningitis, ischemia and infarct, atrophy and calcifications.

CT findings:
- Dural thickening. Dural calcification ("popcorn"), around basal cisterns.

- Abnormal meningeal enhancement (seen years after treatment of initial infection).

RM imaging:
Findings are better seen at gadolinium-enhanced MR imaging than at CT.

- Calcified meningeal nodules are hypointense in all sequences.

- Peripheral meningeal enhancement.

SKELETAL TUBERCULOSIS

It affects patients of any age.

Locations: vertebral column, hip and knee.

It is caused by hematogenous spread from a primary pulmonary focus (Pulmonary tuberculosis is found in 50% of bone TBC).

Urogenital and skeletal tuberculosis coexist in 20-45% of cases.

Spondylitis tuberculous (Figure 7) on page 17

Caused by hematogenous spread via plexus of Batson.

There is no reactive sclerosis or periosteal reaction (dd with pyogenic infections of the spine).

Without treatment it can develop a vertebral collapse and anterior wedging.

In developing countries, it is a childhood disease. 50% of skeletal TBC involves the spine (lower thoracic and upper lumbar levels more frequently).

Predisposing conditions: General weakness, immunosupresion.

There is no sex predominance.

Location:

In 90% of cases usually are 2 vertebral bodies affected, in 50% 3 or more levels.

It affects the anterior part of the vertebral body adjacent to the end plate --> adjacent intervertebral disk --> additional spinal segments -->paraspinal tissues (paravertebral abscess-Pott).

Paraspinal abscesses are found in 55-95% of cases.

Simple Rx:
- Bone destruction.
- Soft tissue mass.
- Loss of intervertebral disc height in 25%.
- Vertebral body fusion.

CT:
- Extensive bone destruction.
- Paraspinal abscesses.
- Epidural extension.

MR:
It is the preferred imaging modality.
- Extensive bone destruction with intervertebral discs unaffected. Posterior elements rarely affected.
- Differential diagnosis: PYOGENIC INFECTION, metastatic disease.

Tuberculous arthritis (Figure 8) on page 18

It tends to affect large weight-bearing joints (hip and knee). Is tis characteristically a monoarthritis.

There is usually synovial involvement with thickening. The end result is a fibrous ankylosis of the joint.

Classical triad of Phemister:
- Yuxtaarticular osteoporosis.
- Pheriferal bone erosions.
- Joint space narrowing (though there is relative preservation in the early stages).

ABDOMINAL TUBERCULOSIS

The abdomen is the most common focus of extrapulmonary tuberculosis.

The solid viscera more often affected is the gastrointestinal tract.

CT is the principal imaging technique for investigating possible abdominal TBC.

Lymphadenopathy (Figure 9) on page 19

It is the most common manifestation of abdominal tuberculosis (55-66% of patients).

There is mesenteric and peripancreatic lymph nodes enlargement.

Different patterns:
- Enlarged nodes with hypoattenuating centers and rim enhancement.
- Conglomerate mixed-attenuation masses.
- Enlarged homogeneus-attenuation nodes.
- Increased number of normal homogeneous nodes.

Hepatosplenic Tuberculosis (Figure 10) on page 20

It is common in patients with disseminated disease.

It can be micro-nodular-miliary or macronodular, 0.5-2 mm nodules (if is is micronodular).

THey are seen as hypoattenuating lesions at CT with irregular ill defined margins and hypoechogenic at US (liver can appear hyperechoic).

Hepatic tuberculomas eventually tend to calcify.

HEAD AND NECK TUBERCULOSIS (Figure 11) on page 21

It represents about 15% of cases of extrapulmonary tuberculosis.

The most common affectation are NECK NODES, often manifesting as bilateral painless cervical lymphadenitis (SCROFULA), with central necrosis and peripheral rim enhancement at CT and MR. Nodal calcification may also be seen.
DD: necrotic nodes seen in some metastatic carcinomas.

It can also be seen inflammatory soft-tissue thickening and bone erosions.

Extranodal tuberculous disease is rarely seen.

Images for this section:

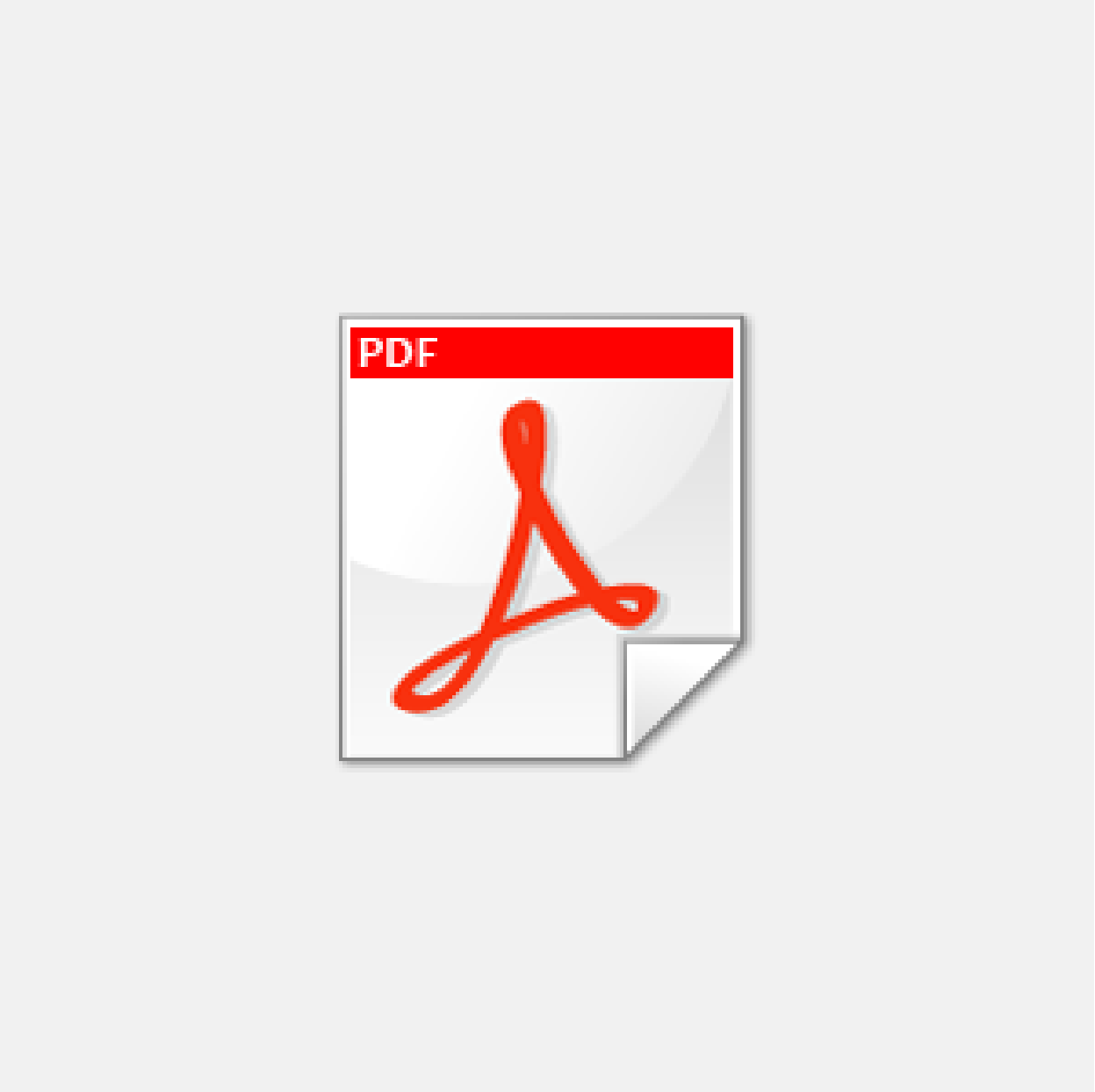

Fig. 1: Parenchymal disease.

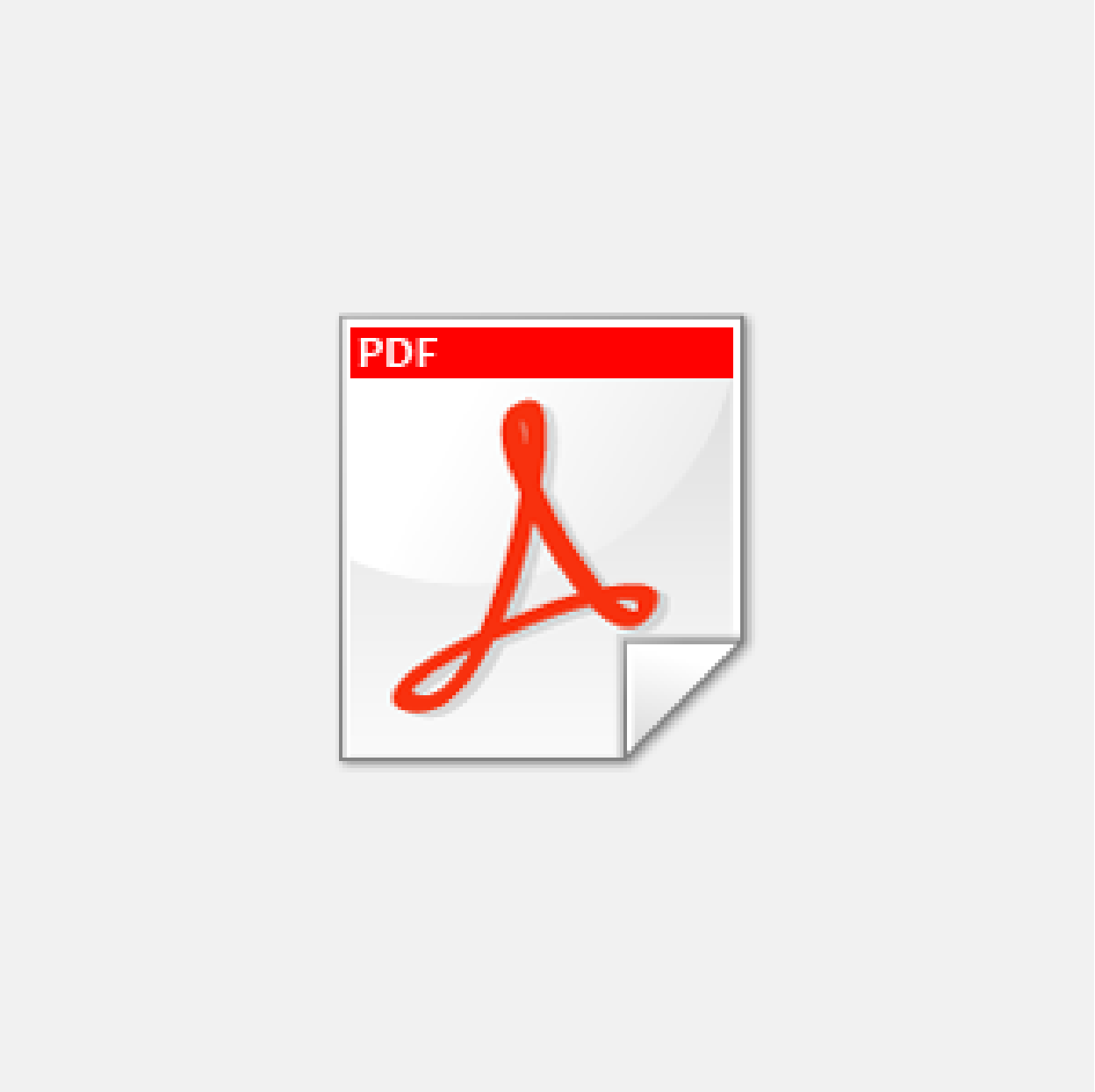

Fig. 2: Pulmonary lymphadenopathy.

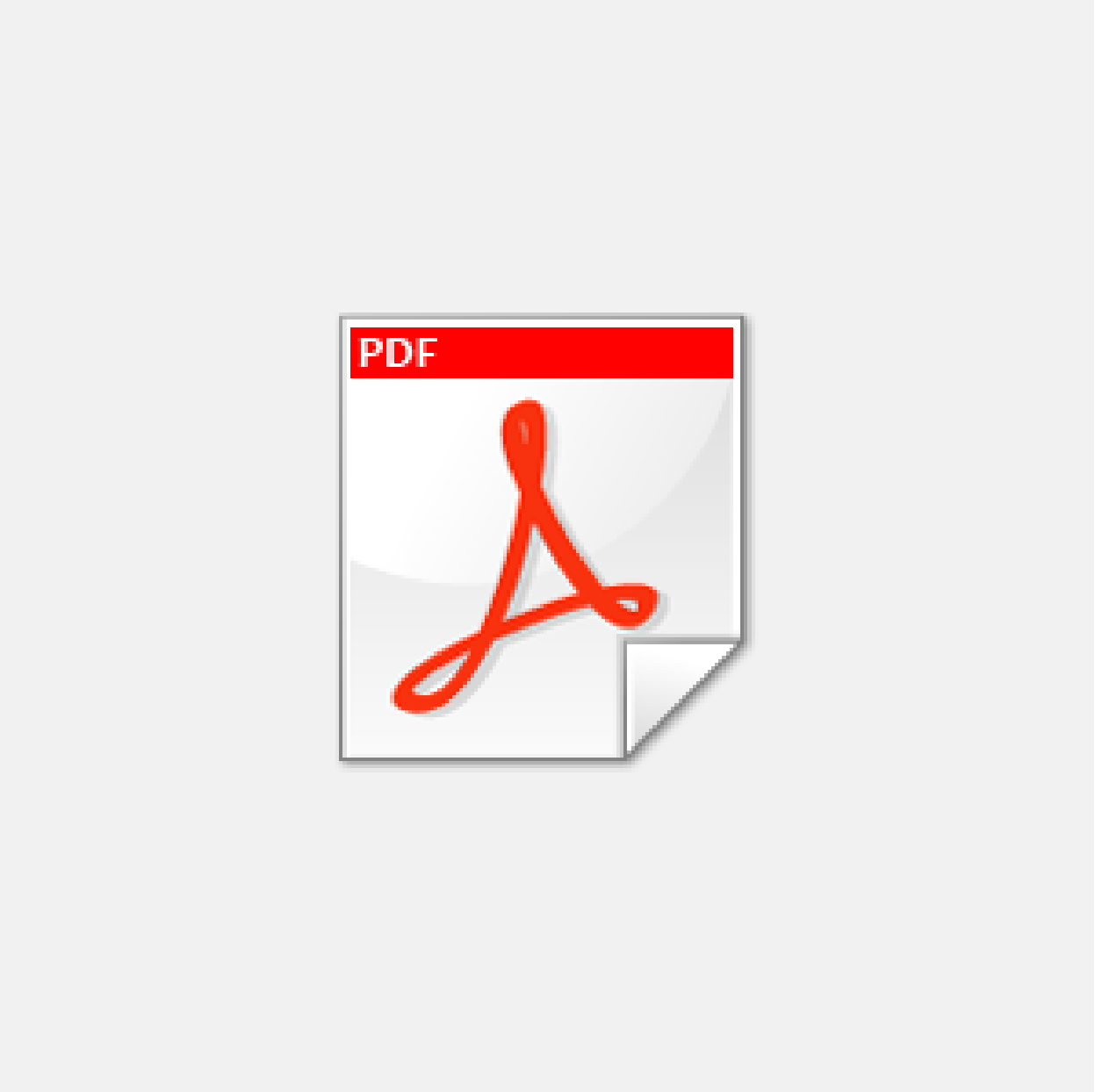

Fig. 3: Miliary tuberculosis.

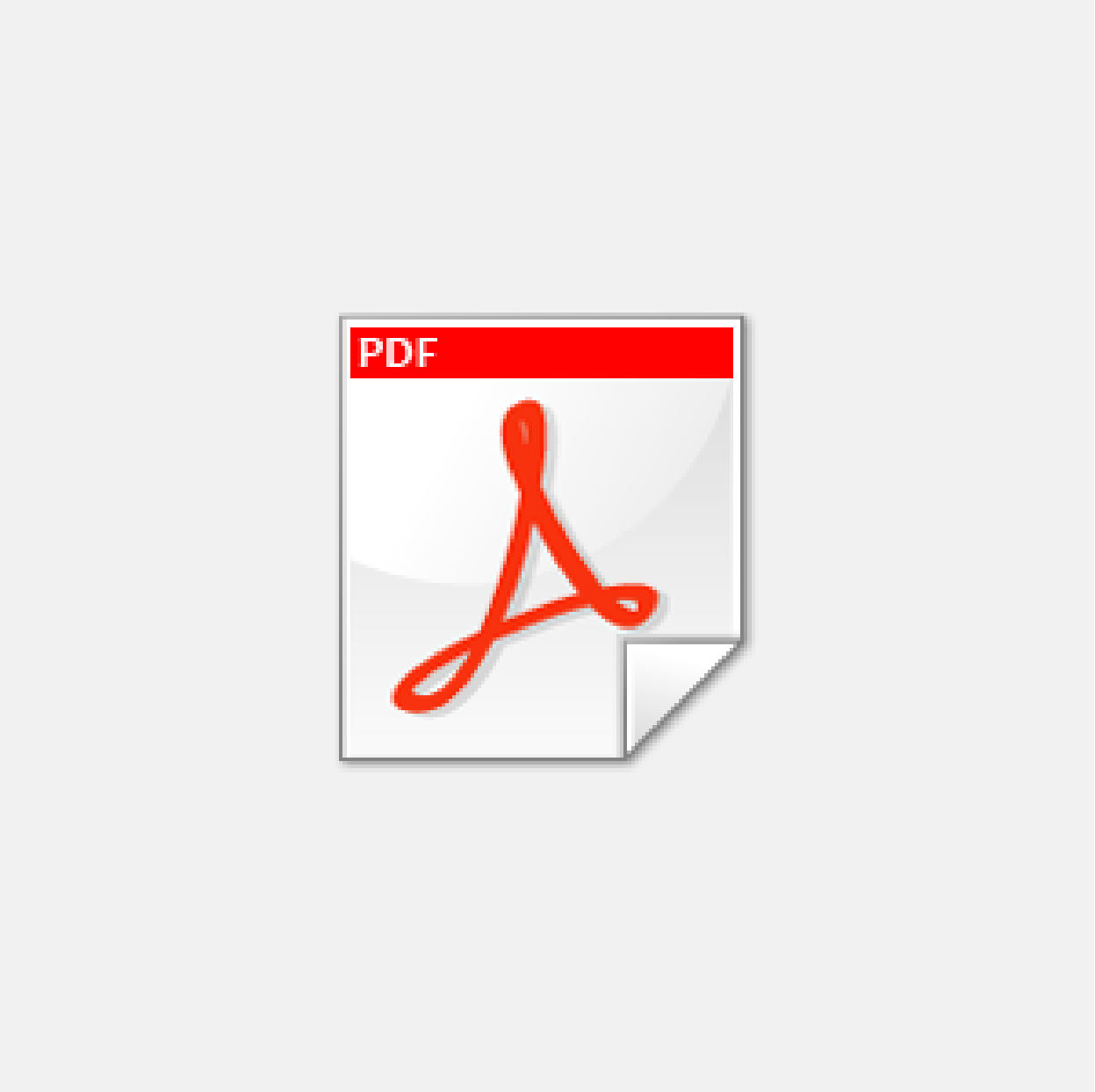

Fig. 4: Pleural effusion.

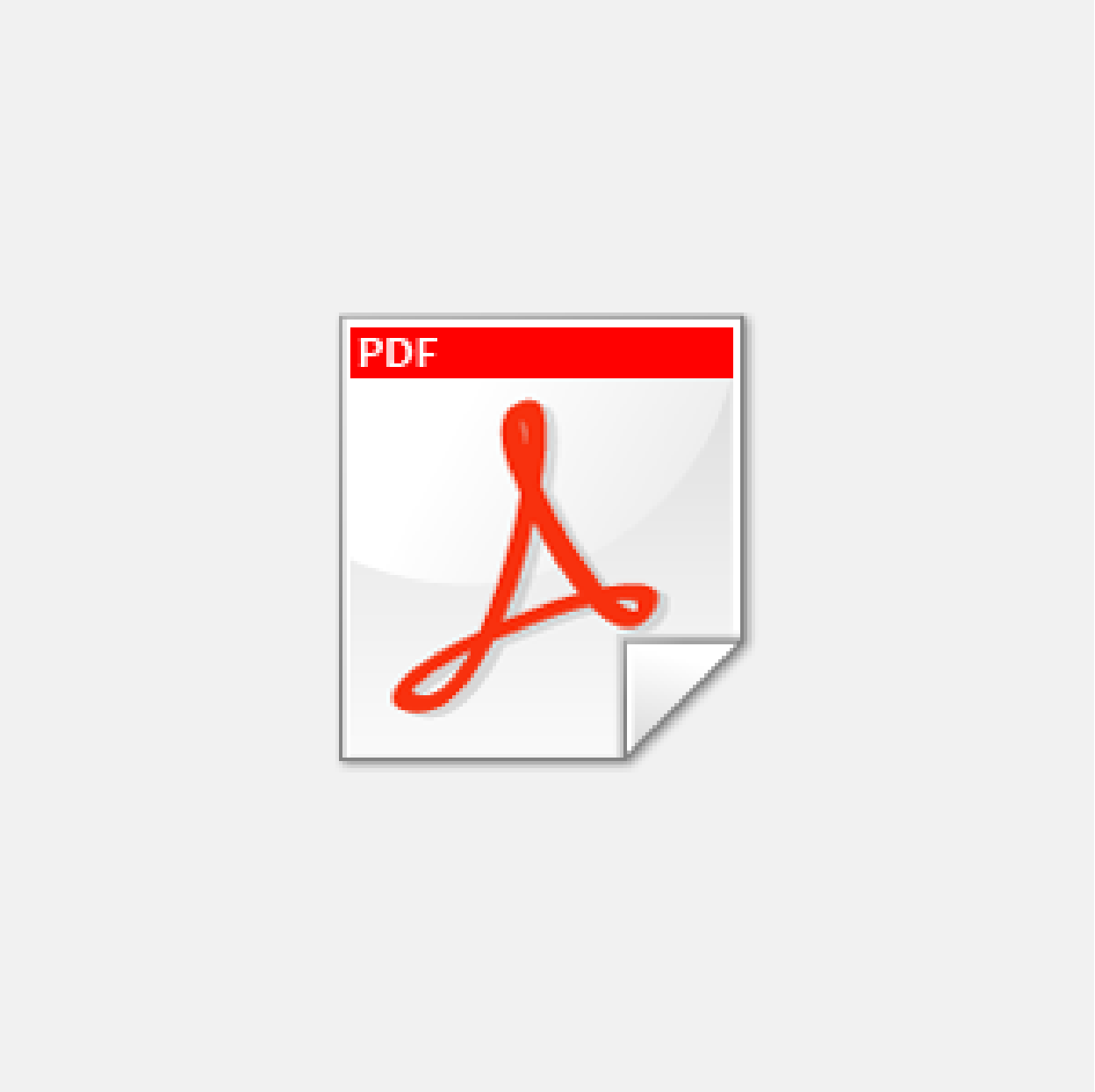

Fig. 5: CNS parenchymal tuberculosis.

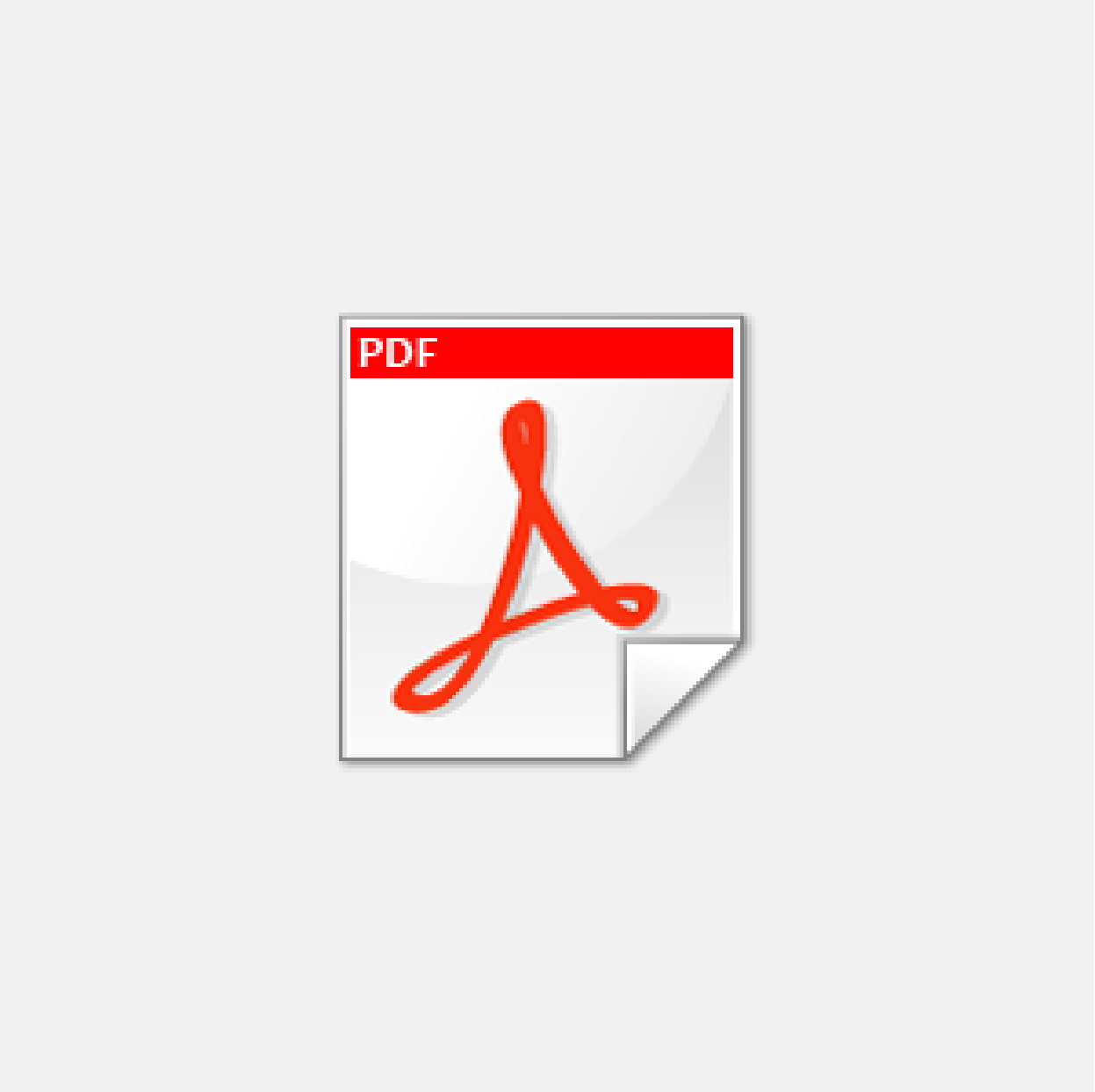

Fig. 6: Tuberculosis meningitis.

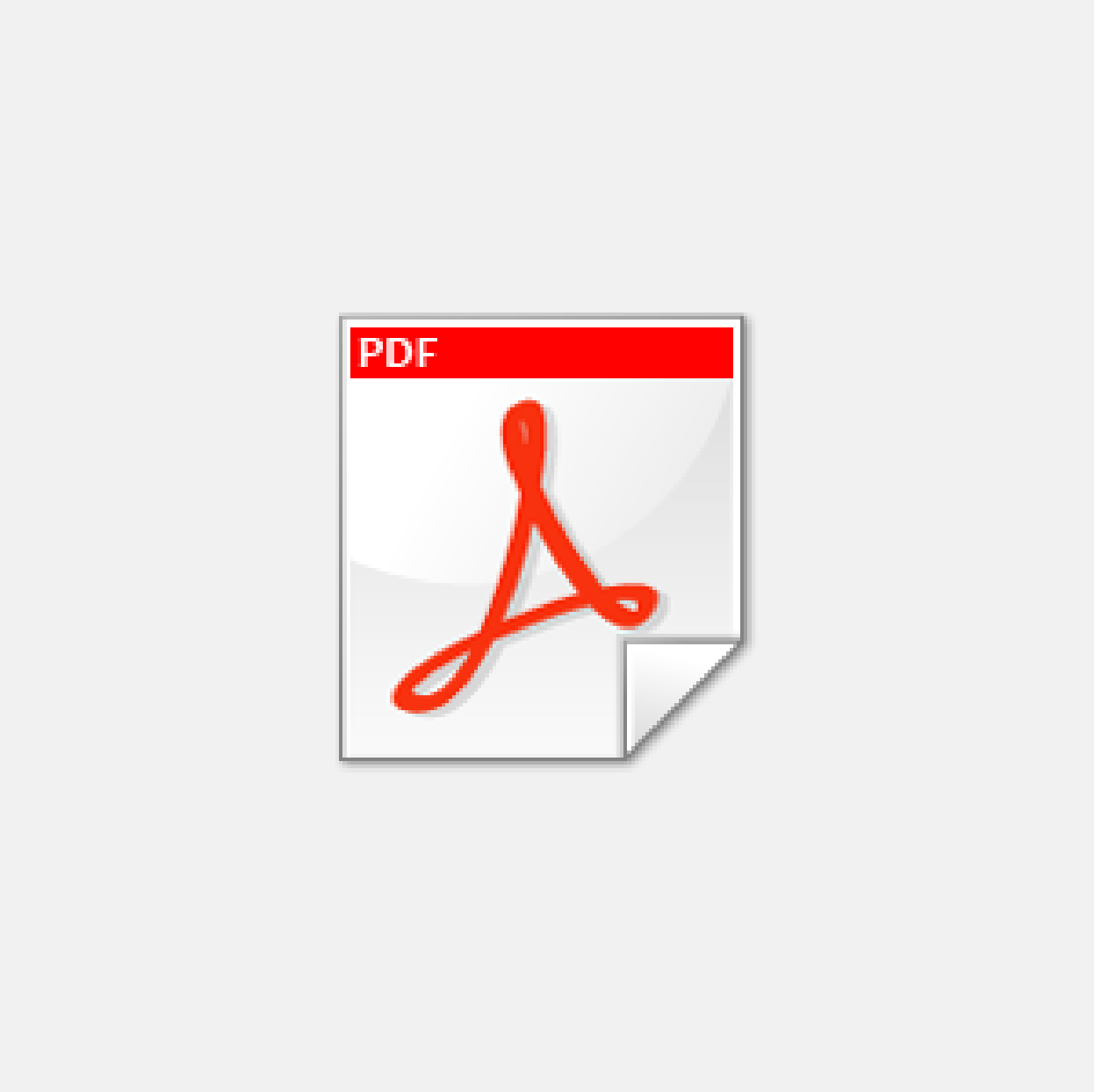

Fig. 7: Spondilytis tuberculous.

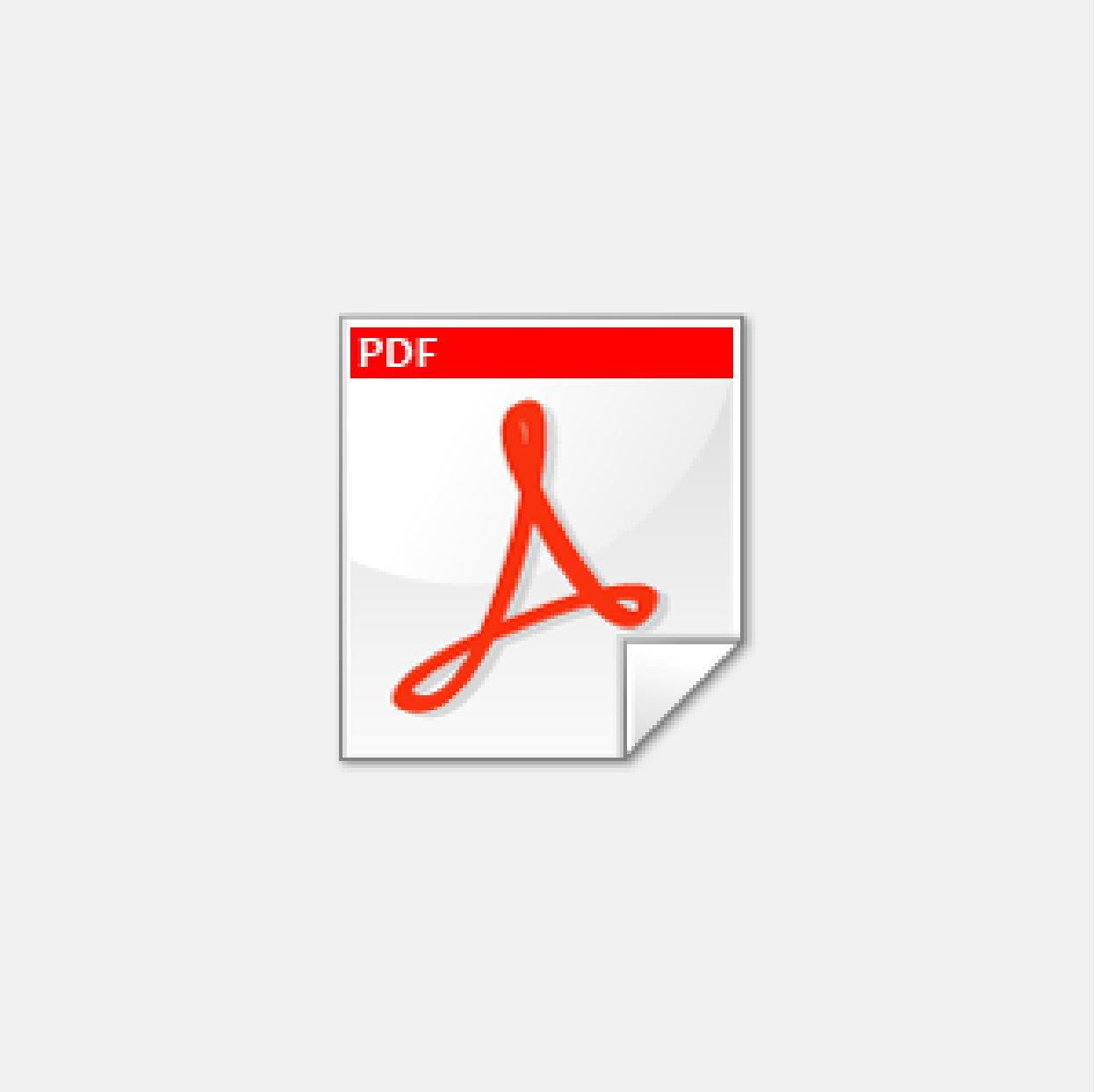

Fig. 8: Tuberculous arthritis

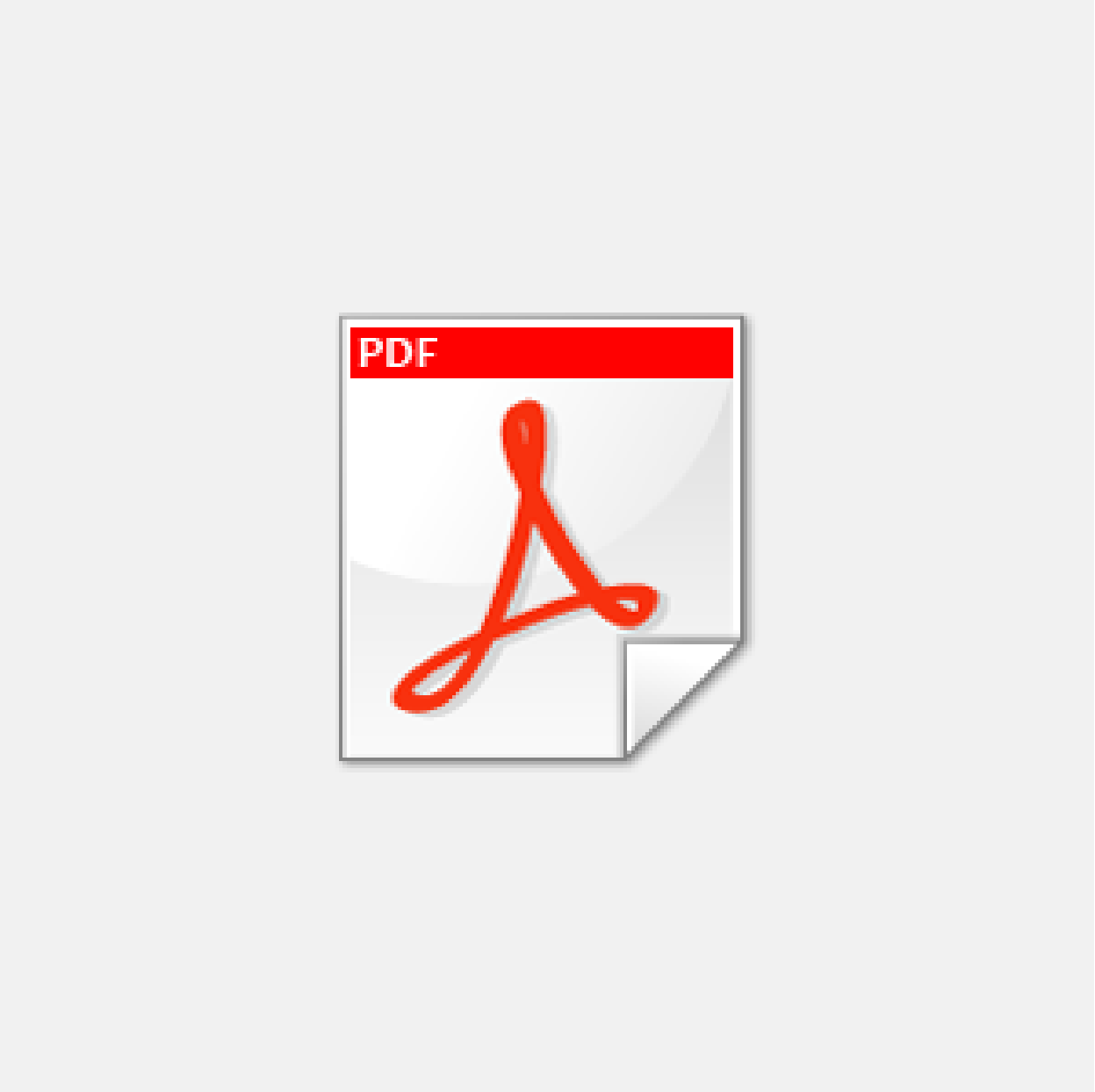

Fig. 9: Abdominal lymphadenopathy.

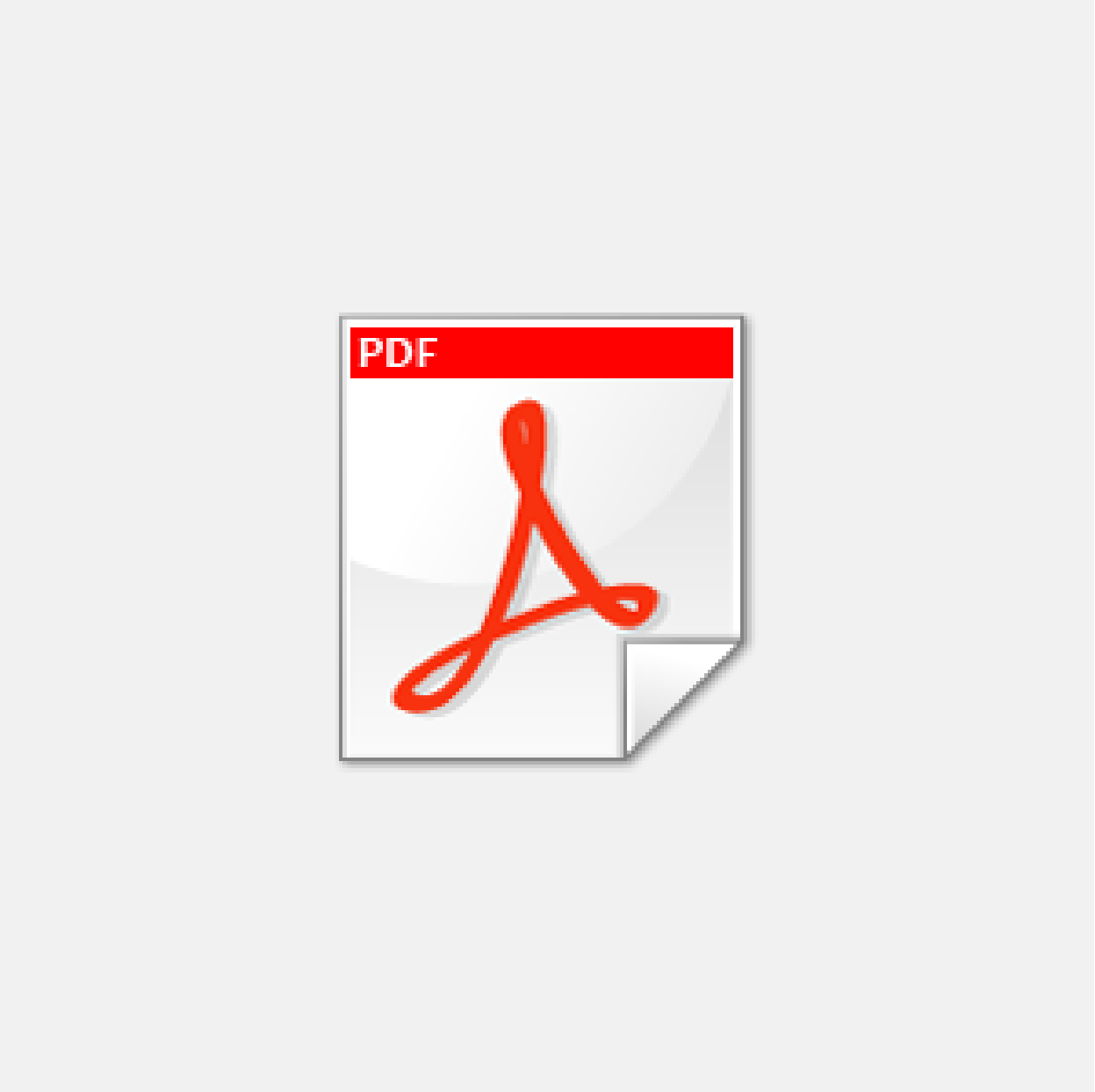

Fig. 10: Hepatosplenic tuberculosis.

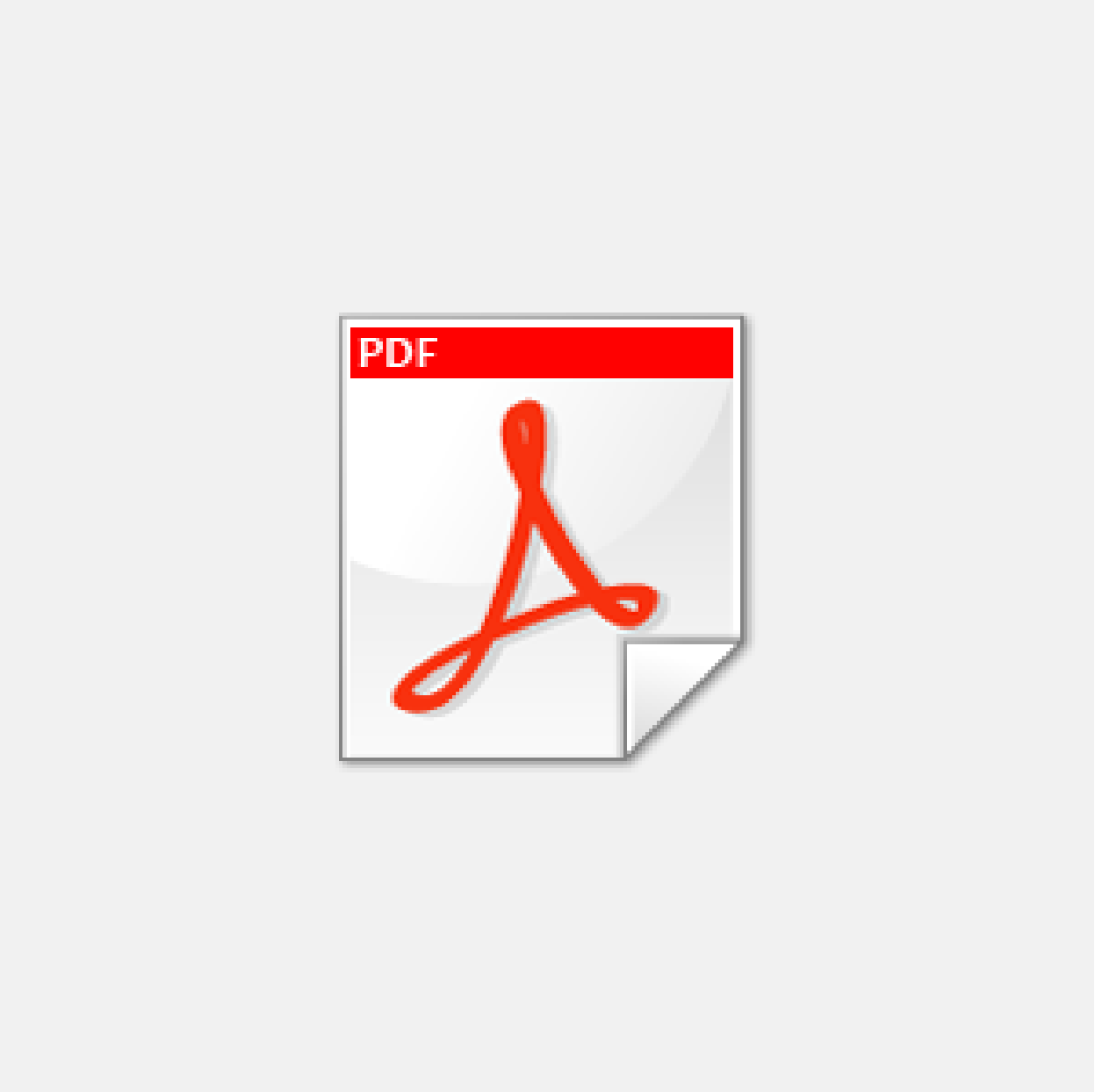

Fig. 11: Cervical lymphadenopathy.

Conclusion

Childhood tuberculosis is emerging in our region due to the increased immigration as the main factor.

There is high morbidity and frequent multiorgan involvement.

Its clinical presentation is nonspecific and can have a wide differential diagnosis, so it is essential an accurate knowledge of the radiological signs.

Personal Information

References

1. Burrill J, Williams CJ, Bain G, Conder G, Hine AL, Misra RR. Tuberculosis: a radiologic review. Radiographics 2007; 27:1255-73.

2. Andronikou S, Joseph E, Lucas S, Brachmeyer S, Du Toit G, Zar H, et al. CT scanning for the detection of tuberculous mediastinal and hilar lymphadenopathy in children. Pediatr Radiol 2004;34:232-6.

3. Marais BJ, Gie RP, Schaaf HS, Starke JR, Hesseling AC, Donald PR, et al. A proposed radiological classification of childhood intra-thoracic tuberculosis. Pediatr Radiol 2004;34:886-94.

4. Leung AN, Müller NL, Pineda PR, FitzGerald JM. Primary tuberculosis in childhood: radiographic manifestations. Radiology 1992;182:87-91.

5. Kim WS, Choi JI, Cheon JE, Kim IO, Yeon KM, Lee HJ. Pulmonary tuberculosis in infants: radiographic and CT findings. AJR Am J Roentgenol 2006 ;187:1024-33.

6. Kim WS, Moon WK, Kim IO, Lee HJ, Im JG, Yeon KM, et al. Pulmonary tuberculosis in children: evaluation with CT. AJR Am J Roentgenol 1997;168:1005-9.

7. Jeong YJ, Lee KS. Pulmonary tuberculosis: up-to-date imaging and management. AJR Am J Roentgenol 2008;191:834-44.

8. Harisinghani MG, McLoud TC, Shepard JA, Ko JP, Shroff MM, Mueller PR. Tuberculosis from head to toe. Radiographics 2000 Mar-Apr;20:449-70.

9. Van Dyck P, Vanhoenacker FM, Van den Brande P, De Schepper AM. Imaging of pulmonary tuberculosis. Eur Radiol 2003;13:1771-85.

10. Kim HY, Song KS, Goo JM, Lee JS, Lee KS, Lim TH. Thoracic sequelae and complications of tuberculosis. Radiographics 2001; 21:839-58.