A 30-year-old female presented with complaints of headache, dizziness, generalized seizures, and proptosis for the last 4 years. A family history of having pig farming in the neighborhood is present. No previous investigations were available and the patient was on antiepileptics. Currently, non-contrast Computed Tomography (NCCT) head revealed multiple small hypodense lesions having eccentric calcific foci within, involving the bilateral cerebral parenchyma, muscles of mastication, extraocular muscles, and neck muscles. To confirm the diagnosis, Contrast-Enhanced Magnetic resonance Imaging (CE-MRI) brain was done which revealed innumerable T2 hyperintense lesions with a hypointense eccentric focus in the brain parenchyma, head, and neck muscles. Many lesions showed mild to moderate perilesional edema and a thin peripheral enhancing wall. These resembled starry sky appearance in bilateral cerebral hemisphere (1). (Figure:1A, B) Disseminated Intramuscular lesions appeared like snakeskin, so we suggest naming it as a snakeskin appearance. (Figure:2A, B) Clinical and radiological imaging concluded as neurocysticercosis (NCC) and disseminated intramuscular cysticercosis. The patient was started on steroids and antiepileptics for initial 3 days during a hospital stay. Later, the patient was followed up on albendazole and steroids.

Discussion

Cysticercosis is caused by accidental ingestion of Taenia solium eggs and it is one of the common causes of acquired epilepsy. (2) Humans are definitive hosts and pigs are the intermediate or secondary hosts. Pigs ingest the tenia solium egg, which develops into infective tapeworm cysts. In the human small intestine, this tapeworm cyst develops into an adult worm which causes cysticercosis. The clinical presentation of NCC mainly depends on the size, number, location, and stage of the parasite. Common symptoms include altered sensorium, headache, seizures, meningitis, and blindness. The investigation includes cerebral spinal fluid enzyme-linked immunoassay (ELISA), neuroimaging, fundoscopy, histology, and enzyme-linked immune-electro-transfer blot (EITB) assay for the detection of antibodies (2). Radiological classification of NCC is based on the location and the disease stage and it is
divided into five stages: non-cystic, vesicular, colloidal vesicular, granular, and calcified granular. Inflammatory reactions occur in the colloid vesicular stage. The granulomatous nodule is found in granular NCC which later develops into calcified granular stage (3). In our case, all the stages of NCC were seen. Various stages of disseminated intramuscular cysticercosis were seen in bilateral ocular muscles, masseter muscles tongue, and neck muscles. It resembled a snakeskin appearance in MRI. Complication includes lacunar infarct, stroke brain hemorrhage, and midbrain syndrome. Hydrocephalus may occur by intraventricular cysts or secondary arachnoiditis or ependymitis in response to massive inflammatory responses. Close differential diagnosis includes intracranial tuberculomas, toxoplasmosis, and primary or secondary brain metastasis. Treatment includes antiparasitic drugs and symptomatic medication. Surgery is usually done for obstructive hydrocephalus. (4) NCC is the preventable and likely eradicable cause of seizure and epilepsy. The prognosis for parenchymal NCC is fairly good, however dismal for the extra parenchymal NCC. Prognosis depends mainly on the type of infection (3).

REFERENCE
**Figure 1A and B:** T2WI- Axial sections of CE-MRI brain shows innumerable cystic lesions with hypointense eccentric focus in bilateral cerebral hemisphere (starry sky appearance), orbital muscles, muscles of mastication, facial muscles, tongue and neck muscles.

**Figure 2A and B:** T2WI-coronal and sagittal sections of CE-MRI neck shows innumerable cystic lesions with hypointense eccentric focus in neck, shoulder and back muscles (snakeskin appearance).