Introduction:
The incidence of the bilateral facial nerve palsy is very rare ranging from 0.3% to 2.0% of all facial palsy cases. There are many causes of bilateral facial nerve palsy enlisted in the literature of which Bell’s palsy accounts only 23%. Many a time it is a sign of an underlying serious condition and some of the causes need early diagnosis and prompt treatment. Hence such cases require extensive investigations to come to a diagnosis. Here we report a case of bilateral facial nerve palsy where after extensive investigations we could not get a definite cause and treated as Bell’s palsy.

Case Report:
A 24-year-old unmarried female diagnosed as two weeks’ old left sided infranuclear facial palsy was referred to us for electrotherapy. She was taking oral methylprednisolone and antiviral drugs. On examination she had mild fever and occipital headache for which she was taking acetaminophen 500 mg three times daily and cefuroxime 1g/day orally in divided doses. We advised for electrical stimulation of the facial muscle and active and passive orofacial muscle exercises. Within 6 days of treatment the right side of the face also got paralysed. The priority was to rule out life threatening diseases like leukaemia, Guillain-Barre syndrome (GBS). On taking history she was a beautician and denied of taking any abusive drugs or alcohol. On examination she had House-Brackmann grade V bilateral facial nerve palsy (Fig 1a,b,c) Bell’s phenomenon could be elicited on both sides. Taste sensation was present but reduced on both side and there was no hyperacusis. Muscle bulk on both sides of the face was reduced. Eye opening was normal and other cranial nerves (V, VI, IX and X) were intact. Skin sensation was preserved. Endoscopic examination of nose and paranasal sinuses revealed only septal spur on left side.

Routine haematological reports were normal except reduced total lymphocyte count (TLC) of 970/cumm. Cerebrospinal fluid (CSF) analysis shows cell count of 5/µl, predominantly lymphocyte, sugar 40mg% and protein 50mg/dl. Gram-stain was negative. HIV antibody detection test was negative. Impedance audiometry was bilateral ‘A’ type. Chest x-ray was within normal study. Computed tomography (CT) of the chest showed multiple calcified lymph nodes in aortopulmonary window and paratracheal region and hyperattenuating foci in right middle lobe suggestive of old healed granuloma. Magnetic resonance imaging (MRI) and contrast enhance CT of the brain showed normal study.

Abstract
We report a case of 24-year-old female with facial paralysis involving both sides in a sequential manner. This could not come to a definite aetiology with series of investigations and hence treated as bilateral Bell’s palsy. Bilateral facial nerve palsy is a rare condition and requires prompt action to trace to a definite cause so as to rule out life threatening causes and plan specific management in the right’s hand.

Key words: Bilateral facial nerve palsy, neurosarcoidosis, Bell’s palsy.

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We could not correlate the bilateral facial palsy to a definite cause hence treated as Bell’s palsy. Oral methylprednisolone was continued for a month and stopped by tapering the dose. Electrotherapy was stopped after one month stimulation and continued with orofacial muscle exercises as home based therapy. After one year follow-up we found that the palsy reduced to House-Brackmann grade II (Fig 2a, b). The frontalis muscle palsy and intermittent occipital headache still persist.

Discussion:
Bilateral facial nerve palsy is defined as the involvement of the opposite side within thirty days of the onset of the first side. It is a rare finding to have bilateral facial nerve palsy and is seldom due to Bell’s palsy. The true incidence of bilateral facial palsy may be under estimated due to uneven manifestation of the two sides making it very difficult to identify a partial paralysis on one side. There are many causes of bilateral facial palsy listed in literature. Infective conditions like Lyme disease, HIV, GB syndrome were ruled out by haematological profile and CSF analysis, though treponemal antibody test was not done. The patient was in antibiotic (cefuroxime) and if due to Lyme disease, must have responded. Moreover Lyme disease is not common in this part of the country. Inflammatory demyelinating polyneuropathy (GBS) typically presents as progressive ascending palsy of voluntary muscles of limbs and trunk. Cranial nerves (CN) commonly involved are IX, X and VII. Here in our case there is no limb involvement and other CN are intact.

Neoplastic conditions like acute leukaemia was ruled out from the blood picture (TLC 970/cumm). Tumours that can cause facial palsy like cerebellopontine angle (acoustic neuroma) tumour could not be revealed from CT and MRI of the brain. Impedence audiometry shows normal pattern (A-type) which conclude that there is no pathology in the middle ear cavity. We came across four cases reported in the literature where bilateral facial palsy is the only presentation of sarcoidosis (neurosarcoidosis) but in our case we could not get any remarkable findings in chest x-ray and chest CT. CT showed only old healed granuloma in right middle lobe and multiple calcified lymph nodes.

Fig 1- (a) Unable to close the eyes on both sides (Bell’s phenomenon); (b) Trying to show the teeth; (c) Trying to make furrows on forehead

Fig 2- After one year: (a) Able to show the teeth; (b) Still not able to make furrows
in aortopulmonary window and paratracheal region which could be due to earlier tuberculosis infection and could not correlate with the present condition. Moreover, there was no sign of hilar lymphadenopathy. CSF findings were normal (though oligoclonal IgG and angiotensin-converting enzyme assays were not included).

On not being able to find a definite cause of the bilateral facial palsy we treated the case as Bell’s palsy and in one year follow-up the palsy reduced to HB grade II. Unfortunately, the frontalis muscle palsy and intermittent occipital headache still persist.

References: