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Cerebral amoebiasis

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CEREBRAL AMOEBIASIS

Pages with reference to book, From 78 To 80

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Amoebiasis caused by entamoeba histolytica mainly involves the gastrointestinal tract[1]. Its extraintestinal involvement is mainly seen in liver and lung[2,3]. Involvement of the brain is known[4,5] but extremely unusual[6,7]. Even rarer is involvement of the brain without any involvement of lung or liver[2]. We present cerebral computed tomographic findings of a case with cerebral amoebiasis without any involvement of lung or liver. Patient was managed by surgical decompression and amoebicidal drugs and he recovered to his normal neurological functions.

CASE REPORT

R.A.B., male, 57 years old, non-diabetic, non-hypertensive, smoker presented to the emergency room with a history of progressive weakness and numbness of left side of the body for three days. This had increased over the last. 12 hours with added complaints of confusion, headache and vomiting. There was no history of loss of consciousness or seizures. On examination patient had dysphasia; left sided hemiplegia, hyperreflexia, hypertonia; up going plantars and left hemi- anesthesia with left homonomynous hemianopia and right lateral strabismus. Fundi were normal with moderate neck rigidity. A routine hematological and biochemical screening only revealed a neutrophilia of 18,000/cu mm with rest of the profile normal. A pre and post contrast (80 ml urografin 76%) enhancement CT scan (G.E. 9800 Quick) of the head was done. This revealed a large area of mixed density occupying right parieto-occipital region without any significant contrast enhancement. There was very little midline shift but there was compression of posterior half of left lateral ventricle (Figure).

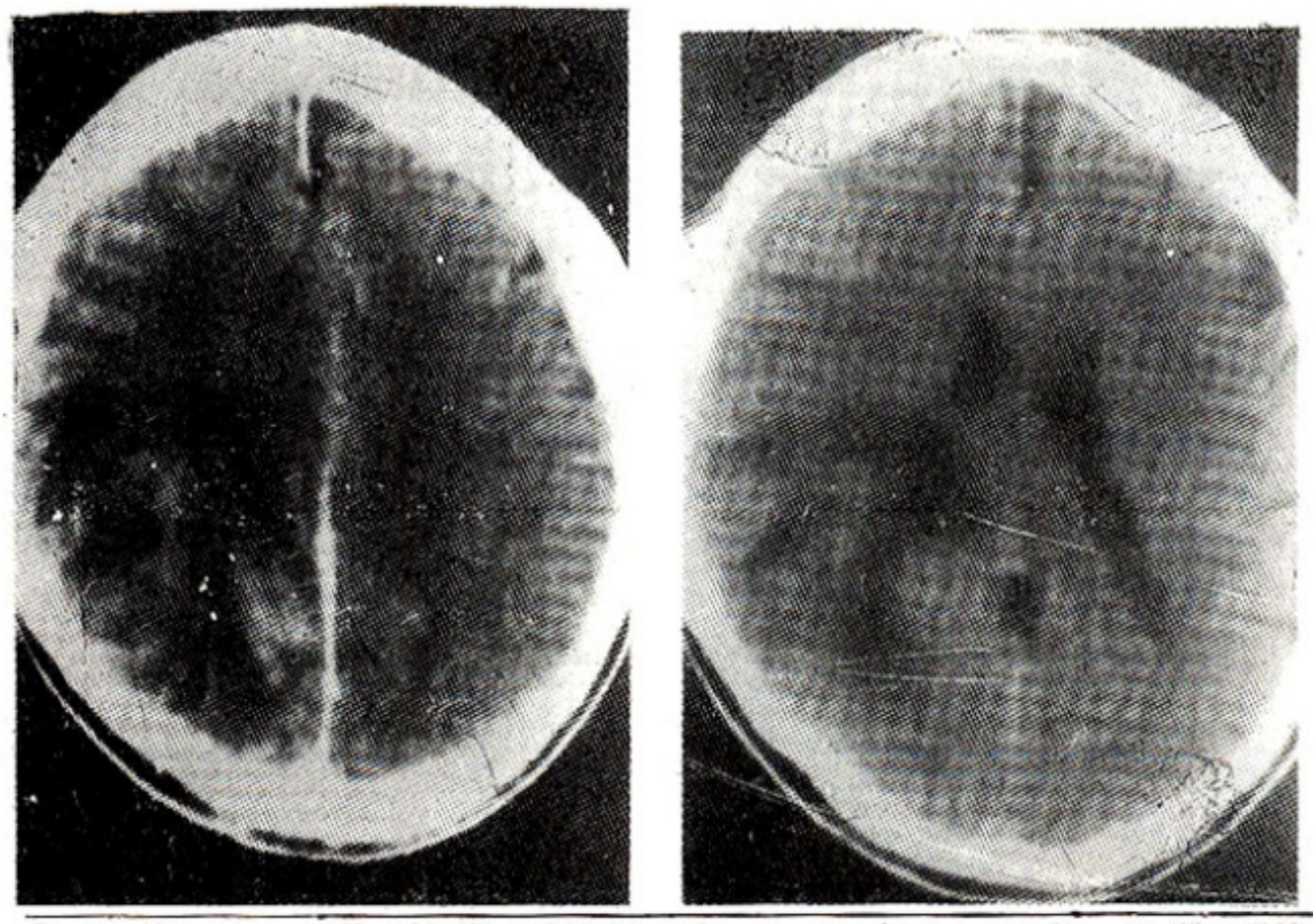

Figure. Non-contrast [L: + 30, W: + 100] (a) and contrast enhancement [L: + 28, W: + 100] (b) CT scan of head showing a large area of mixed density in right parieto-occipital area without any contrast enhancement. No significant midline shift is seen.

On CT appearance a slow growing astrocytic neoplasm was suspected. Another consideration due to rapidity of symptoms was an inflammatory process most probably of fungal aetiology. A craniotomy revealed intracranial abscess. When dura was cut yellowish colour necrotic brain tissue came out which was removed as much as possible. Histopathology of this tissue revealed large extensive areas of necrosis having acute inflammatory and fibrinous exudate with occasional areas of chronic inflammation. Isolated and grouped trophozoite forms of PAS positive entamoeba histolytica were seen. An IHA (indirect hemagglutination test) specific for amoebiasis (cellognost-Amoebiasis Behringer AG, Mar-burg, Germany) was done which gave a titre positivity of 1:512 as compared to normal maximum limit of 1:32. Patient responded with complete neurological and functional recovery over the next 25 days. A search was i.hen undertaken for the primary site considering the brain lesion as a secondary amoebic involvement. No focus was seen on repeated chest radiograph. An ultrasound of abdomen revealed multicystic lesion in the right upper quadrant not separable from either the liver or the right kidney. This mass was found to be in the upper pole of right kidney on CT scan of abdomen. No liver abscess was seen. The mass was later found to be clear cell carcinoma of the kidney. No evidence of hepatic involvement seen operatively.

DISCUSSION

Since 1875 when Losch found trophozoites in the stools of a poor Russian farmer in St. Petersburg[8],

amoebiasis due to infection with entamoeba histolvtica is common and considered prevalent worldwide[1]. Approximately 10% of the world's population is considered to be affected[1]. The incidence in the developed countries is considered to be declining. But this is still a disease to be considered due to infrequent travellers, in recent immigrants, in patients with AH)S and in male homosexuals[1]. In developing especially tropical countries this remains a prevalent disease. In Mexico about 50% of the population is considered to be harboring the disease at one time or other[1]. In India the incidence has been quoted up to 17.4% of all the infectious diseases[9]. Amoehiasis is generally considered as the infection of gastrointestinal tract. Hematogenous extra abdominal spread especially to brain is considcrccl as extremely exceptional and rare[3,6,7]. Secondary cerebral amoebiasis, as it is called[3], is an unusual cause of brain abscess with incidence varying from roughly I % to 8.1%[6] of patients with amoebiasis according to the prevalence of disease. The exact number of cases reported so far is difficult to assess. Hughes[10] calculated these to be 97. To this we can add 2 cases by Becker[11] and one each by Schmutzhard[12] and Tikly[13] to make it a total of 101 cases so far. The cerebral involvement in amoebiasis is considered to be generally fatal[3] with rapid deterioration occurs soon after onset[7]. Becker[11] after reviewing the literature came out with six cases who had survived and added one of his own. To this we add another patient reported by Schmutzhard[12] and one by Tickly[13]. This case is the tenth case reported so far of patients surviving cerebral amoebiasis. Our case is to our best of knowledge second case from this part of the world after one reported by Reddy et al from India[14]. Cerebral amoebiasis is thought to occur in patients with associated infections in other parts of the body. The liver is always involved with occasional involvement of lungs[2,3,10,12,13]. Primary cerebral involvement with free living amoeba has been described but this is a distinctly different aetiological agent[15]. In our case although an ultrasound as well as a CT scan of liver and kidneys were done, no evidence of liver involvement was found. No focus was seen on repeated chest radiographs. The CT appearances of amoebic brain abscess has been described in three cases[11-13]. In one case[13] there was a large ring enhancing lesion in the left cerebral hemisphere. In another case[12] there were 41 small ring enhancing lesions in both cerebral hemispheres, while in the case described by Becker[11] there was cerebritis initially and 5 days later it formed an abscess like ring en hancement. In our case there was no walled off abscess or ring enhancement on CT. Instead there was a non-enhancing mass like lesion without any significant midline shift. There are no pathognomic features of amoebic brain abscess on CT scan. An irregular lesion without a defined area of reactivity or wall is both compatible with the gross appearance of the lesion and is suggestive[11].

CONCLUSION

Amoebic involvement of brain is a rare complication of amoebiasis. It is life threatening but with the advent of newer antibiotics it can be treated if diagnosed early. Although there are no pathognomic features on CT, this disease should be suspected in acutely ill patients especially in areas where the disease is prevalent. It is also important for the surgeon as well as the pathologist to keep this disease entity in mind when looking at purulent material from brain abscesses. Special care has to be taken in preparing slides and staining them.

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