Lipscomb (1955)[5] studied one hundred and eight cases of supracondylar fracture of humerus in ten years period, out of these, two patients had rupture of brachial artery. He also treated the ruputured artey by ligating its two ends with normal restoration of circulation.

Fig. 3.

Patient 8 months after injury. No Vascular deficit. Scar mark visible on cubital fossa.

Our results of ligation of severed ends of ruptured brachial artery are similar to that of Spear (1951)[6] and Lipscomb (1955)[5]. This is because of rich arterial anastamosis around the elbow joint. However, inorder to preserve collateral circulation, it is essential to spare as much of the brachial artery as possible (Spear 1951)[6].

Smith (1956)[7] described another case of complete rupture of brachial artery. As in our study Fowles (1978)[8], reported a case of complete reputure of brachial artery alongwith displaced fracture of both bones of ipsilateral forearm.

It is thus evident that rapture of brachial artery is a rare entity, which occurs as a result of severe trauma and that simple ligation of the vessel is as efficacious as grafting or end to end arterial anstamosis, provided the ligature is below the level of ulnar collateral artery (Louis, 1974)[9]. In order to prevent reflex bleeding, ligation of distal end of the artery is also mandatory (Kilburn, 1962)[4].

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RUPTURE OF BRACHIAL ARTERY AS A RESULT OF SUPRACONDYLAR FRACTURE OF HUMERUS

A CASE REPORT

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Abstract

Rupture of brachial artery in association with supracondylar fracture of humerus is rare as compaired to contusion and arterial spasm (Gregon, 1951). However, it is nearly always seen as a result of severe injury (Griffth, 1948)[1]. Rupture of brachial artery should be suspected, whenever there is open fracture and extensive wound involving the antebrachial fossa.

Key Words: Fracture, Supracondylar, Brachial Artery Rupture.

Case

During the year 1988 to 1992, we studied 62 cases of supracondylar fracture of humerus in children. There was one case of ruptured brachial artery (incidence of 1.8%). It was a case of ten years old boy, who sustained open supracondylar fracture as a result of road traffic accident. There was profused bleeding from the wound, in the cubital fossa. The brachial artery was completely severed. On X-Ray there was totally displaced supracondylar fracture of humerus (Tybe IV Holmberg, 1945) (Fig-1). Ends were retracted, we tried to approximate the ends, but the ends could not be brought together, moreover facilities for microvascular repair were not available we finally ligated the ends and reduce the fracture, through same wound and fixed it with two Kirschner wires (Fig. 2). Radial & Ulnar pulses returned after forty eight hours (Fig. 3).

Fig. 1.

Displaced Supracondylar Fracture.

Fig. 2.

Fracture after fixation with Kirschner wire.

Discussion

Methods used in dealing with ruptured brachial artery vary widely. Marnhom (1939) simply ligated the divided ends of the artery, as did Roberston (1952)[2]. After ligation the radial & ulnar pulses returned with in forty eight hours. Six months later there was no evidence of ischemic muscle contracture or nerve lesion Mathewson in 1940 successed in performing end to end anastomosis. JACKSON (1940)[3] in the same year applied a vascular graft Kilburn (1962)[4] identified and tied only the proximal end of the arter, in one of these cases, without any evidence of vascular deficit. Common to both, grafting and end to end arterial suture is requirement of highly technical skill as well as more time consuming.

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Discussion

The hydatid disease is common in liver (75%) and lungs (20%). The bony involvement is rare and accounts for less than 1% of the total lesions (Gide 1992)[1]. Bony lesions are common in the vertebral column, plevis, long bones and skull. Spine is involved in about 50% of the cases (Charles 1988)[2]. The osseous lesions are primary and not extensions from the soft tissue. The joint involvement without a bony focus is exceedingly rare.

Intraosseous foci of echinococcus predominate in spongiosa and consist of minute, thin wall cysts. As cysts grow and expand, they produce cortical thinning, pathological fracture, and breaking of cortices results in soft tissue extensions. There is little periosteal reaction. The bone cysts lack the host adventitia layer as seen in other organs. The symptoms result from pressure exerted on adjacent structures by enlarging cyst. The patient complains of pain and swelling or may present with pathological fracture and spinal cord compression.

Radiologically there are radiolucent areas with expansion of bone, thinning of cortices and minimal periosteal reaction. These signs and soft tissue calcification appears to be highly suggestive of hydatid disease (Booz 1993)[3]. The normal bone architecture may disappear and calcification may be visible in the wall of the lesion (Ectacyst) indicating death of the parasite. The computed tomography (CT) demonstrates local extension of lesion in both bone and soft tissue very well (Torricelli et al 1990)[4]. The magnetic resonance imaging (MRI) also shows exact extent of the disease.

Apart from the imaging studies the diagnosis may be aided by the laboratory tests, namely; Eosinophilic count, Casoni test, Complement fixation test and Precipitation test.

Differential diagnosis includes fibrous dysplasia, giant cell tumor, chondrosarcoma, plasmacytoma and chondromyxoid fibroma. Complications include pathological fractures, secondary infection, intraspinal rupture producing paraplegia and transarticular extension with osseous collapse and deformity.

The recommended treatment is combination of surgery and chemotherapy (Agarwal 1992)[5]. At surgery complete excision of intact cyst is not possible due to lack of adventitious layer, therefore thorough curretage and lavage with 33% saline is recommended. 1% formaline is also to be used. Bone grafting is performed to prevent pathological fractures. There is no published record of the use of acrylic bone cement as performed by us in case 1 on repeat surgery. The bone cement is used to fill the cavity after curretage for giant cell tumor of the bone. In addition to filling the gap, heat generated by the bone cement may be helpful in destroying larvae. Initially Mebandazol was used but due to its complication especially nephrotoxicity, Albendazol is preferred. Albendazol is used in doses of 10-20mg/kg BW/day for several weeks (Gideon 1992, Canerini 1993)[1,6].

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