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Heterotopic Ossification in Patients with Spinal Cord Injury

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Abstract

A retrospective study of 45 patients of Spinal Cord Injury (SCI) with Heterotopic ossification (HO) who were admitted to Spinal Injury Ward, Department of Rehabilitation, Safdarjang Hospital, New Delhi and managed conservatively was undertaken. Thorough clinical history with emphasis on massage, manipulation, pressure sores, urinary tract infection (UTI): roentegraphic studies done at frequent intervals, and routine conservative managements strategies were tools used. This study revealed male predominance of the condition particularly in adult age group, with lesion around dorsolumbar spine and prediliction for hip and knee joint involvement. It was found equally common in spastic and flaccid paraplegics. Infected pressure sores, massage and manipulation seems to have variable contribution in genesis of HO. This study emphasise significance of UTI in pathogenesis of HO. With conservative management, it was possible to retain functionally useful ROM in all cases of present series.

Introduction

Heterotopic ossification is one of the common complication encountered in spinal cord injury patients. Its incidence has been reported to be 16-53% in different studies. 48.7% Dejerine & Callier[2,3] (1919), 37.0% Soule [19](1945), 41.0% Abramson (1948), 16.0% Harday et at al (1963). Heterotopic ossification is formation of new bone in the connective tissue surrounding the major joints as a result of metaplastic transormation of mesenchymal cells to bone forming cells (Subbarao J.V. 1990). It has been described by various names in the literature like myositis ossificans (Dejerin et al)[2]. Osteosis neurotica paraarticularis or paraossitis (Geldmachar 1925)[5]. Pathological ossification (Hardy et al 1963)[7], neurogenic ossifying fibromyopathy (Soule 1945)[18] and paraosteoarthropathy (Rossier et al 1973)[16] but the term heterotopic ossification or ectopic bone formation seems the most appropriate because it cover the bone formed in muscle and other soft tissue whether of inflammatory or non inflammatory orgin.

Aetiology of heterotopic bone formation in paraplegics is not known but various theories like arteriovenous malformation, some unknown hormonal substance liberation and mesenchymal cells metaplastic transomation into bone forming cells are described in literature. Various contributing factors for new bone formation have been reported in literature like:

- Excessive manipulation/massage
- Spasticity
- Pressure sores
- Urinary tract infection and
- Septicemia.

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Objective

This is study was undertaken with aim to study pattern of HO in spinal cord injury cases, magnitude of ossification, predeliction for specific joints or sites, to identify and relate various causative and precepitating factors mentioned in literature, difference of incidence between spastic and flaccid lesion and to find out managment, strategies most suitable in our conditions.

Review of Literature

Modern concept of Heterotopic ossification complicating SCI and other neurological disorders derives from thesis on the subject by Dejerine & Ceilier (1918)[2,3] which remains single most important reference till date, although Reidel (1983) is usually credited with first description followed by Echhoust (1895), Konig (1906), Potzl (1908) and Kutiner (1908).

Dejerine and Ceilier postulated factors responsible for formation of HO, one essential local factor was lowering of resistance of connective tissues by persistent oedema. Functional immobilisation following neurological defict which caused altered vasomotor outflow[16] was thought to be important as it caused altered blood flow, venous stasis tissue hypoxia, altered pH, and oedema[13,16]. Other local factors i.e. muscle trauma[14] and chemical mediators were also implicated. Second factor was thought to be central neurogenic perhaps due to irritability of intermedio lateral column of grey matter causing changed autonomic activity.[2,3]

Leri (1919)[9,12,14] believed pressure and traction during massage, forceful manipuation resulted in formation of tissue bone which could sustain it, but various other authors [4,6,7,16, 21,22,23] discounted this observation. Lhermite (1919) added a belief in the necessity of super-imposed factor of local infection.[4,5,7]

Lerice & Pollcard[10] suggested that heterotopic ossification in general develops in an ossifiable connective tissue near a depot of calcareous salt based as it was on concept of non specificity of various type of connective tissue derived from mesenchyme.

Abramson[1] found no relationship between ossification and level or pattern paralysis. Which was supported by others.[4,7,22,27] for HO appears only with in area of neurologic deficit i.e. below the level of cord lesion [2,7,8] with predilication for hip and knee joint [1,4,7,12] either unilateral or bilateral. Involvement of upper extremity is infrequent and HO below elbow is rarely reported.[22]

Hardy & Dickson[7] apparently ruled out any co-relation between occurence of HO spastic or flaccid lession and were supported by Wharton et al [23]. Ceilier[2]. Damanski[4] etc. Damanski[4] (1961) in a clinical study stressed importance of skin ulceration, hypoproteinemia, severe UTI in pathogenesis of HO. But others believed [7,8,11,15] these factors to be associated with bone formation arising as a secondary process in necrotic area.

The mass of new bone were either single or multiple and showed variation in size and extent. When large, mass of bone was preceded by palpable soft tissue swelling, sign of inflammation[1,2,3,6-8] in varying number of cases and limitation of passive ROM of involved joint. In most patients bone appears early rapidly increased in size then remains unchanged. Rising serum alkaline phosphatase level in blood receded roentagraphic appearance of ectopic bone and is a useful indicator of osteoblastic activity[16]. Usually appears by 3-4 months and time taken for maturation is 18-24 months[1,2,7,16,23].

Wharton[22] reported that 3-8 percent who develop HO progress towards complete ankylosis although 20% of patient with HO suffer severe functional limitation causing difficulty in self care, transfer, ability to sit in wheelchair.

Other neurological disorders in which HO is known to occur include myelitis, encephalomyelitis, CVA, cerebral tumour, various menningitis Head injury, MS, spinal cord tumour etc. some of which were discussed in papers by Lorber (1946), Mcneur (1954), Irvin etal (1954), Storey[20] and Splele (1959).

There are number of reports in literature regarding management of this condition although no effective treatment exist till date. Good prophylactic care including maintenance of good general condition, avoidence of pressure sore, constant change of postition, early passive and active mobilisation, avoidence of UTI reduced risk of HO substantially[4]. Early diagnosis, timely intervention with passive ranging of joints with or without use of drugs i.e. etidronate sodium, indomethacin are likely to minimise functional loss[19,21].

Surgical intervention after maturation of ectopic bone in ankylosed or restricted joint may be helpful[7,22,23].

Material & Method

We have reviewed 45 patients of HO who were admitted to SCI ward of Rehabiliation Department over a period of 10 years. These patients developed signs/symptoms suggestive of HO during their rehabilitation programme and were studied in detail. History of excessive unsupervised exercises and massage was taken, restriction of passive ROM noted and patients screened with plain X-rays. Other associated factors reported in literature to be contributing to pathogenesis of HO noted. On diagnosis these patients were managed conservatively.

Observations/Results

HO was found to be more common in adult males who were in 3rd decade of life with male female ration of 5.5:1. It was more commonly seen in patients with injury around dorsolumbar region with no patient having injury below L_2 vertebra.

HO was more common in paraplegics (86.7%) who had complete injury (80%) with 55% flaccid and 44% spastic cases (Table-I). The duration between SCI and development, of HO between 9 to 36 weeks with average of 16 weeks. Majority of patients 72% developed HO between 13-20 weeks of post injury period (Table I & Table-II).

Neurological status of patients

Status		No. of Patients	% Age
I.	Quardriplegia	6	13.33
	Paraplegia	39	86.67
II.	Complete	36	80.00
	Incomplete	9	20.00
III.	Spastic	20	44.44
	Flaccid	25	55.56

Table I

Duration between spinal cord injury and Development of heterotopic ossification

Duration in weeks	No. of cases	% Age
Less than 8 weeks	-	-
9 - 12 weeks	3	6.66
13 - 16 weeks	20	44.44
17 - 20 weeks	12	26.66
21 - 24 weeks	4	8.88
25 - 28 weeks	4	8.88
More than 28 weeks	2	4.48
Total	45	100.00

Table II

In our series only 20 patients (44.44%) had definite history of strenous manipulation or exercises and massage (Table-III). Majority of patients had sign of acute inflammation like rise in local temperature 62%, swelling of joints in 32 patients with restriction of passive ROM in 39 (86.66%). In 13 Patients (29%) sign of local inflammation were absent.

As far as radiological observation are concerned, all patients were screened with plain X-ray. We have quantified HO in our observation of these X-ray into three groups. Mild-ectopic bone in form of

History and clinical observatrion

History and Clincial observation	No. of Cases			Total
	Definite	Doubtful	Absent	
Forcible Manipulation or. Ecessive Exercise .	20	14	11	45
Massage	20	14	11	45
Local Rise Temperature	28	-	17	45
Swelling	32	-	13	45
Restriction of Passive ROM	39	-	6	45

Table III

thin needles, flakes or thin plate. Moderate-thick plate of ectopic bone localised to one particular region and not crossing joints; Severe - In the form of sheets or large masses of bone crossing the joints or not localised to one region alone (Table-IV).

HO was most common around hip joint 28 cases (20 unilateral & 8 bilateral). Most common site in incidence & severity of HO were lesser trochanter (28 cases) Greater trochanter (26 cases). 17 cases had HO around knee joint, 13 unilateral and 4 bilateral. No HO was observed in upper extremities even in case of quardriplegia. UTI was present in 66% of cases. Infection with E.coli was seen in 48.88% of cases either singly or in combination. Most common combination was E. coli+Pseudomonas which was isolated in 26.6% cases (Table-V). 77.77% of patient had pressure sores; 32 patients had pressure sores in sacral region out of which 14 had large pressure sores (Grade III & IV), 10 patients had multiple sores and 10 patients did not have any pressure sores (Table-VI)

All these cases were treated on line of acute traumatic myositis ossificances in early stages with rest, NSAID, antibiotics to contain infection and supportive specific therapy wherever need for UTI, pressure sore or spasticity etc. was given. Once sign of acute inflammation subsided, gentle passive ROM exercises were given.

We observed that following this treatment regime, we were able to give all patients a 90-90 position needed for wheel chair ambulation.

Radiological Observation

Site of Heterotopic Ossification	Extent of Heterotopic - Ossification			Total Cases
	Mild	Moderate	Severe	
Around HIP Joint (28)				
- Near Greater Trochanter	8	8	10	26
- Near Lesser Trochanter	8	10	10	28
- Ischial	1	-	-	1
Around Knee Joint (17)				
- Medial Fermoral Condyle	6	3	8	17
- Lateral Femoral Condyle	3	3	3	9
- Supra Patellar	-	2	4	6

Table IV

Associated Urinary Tract Infection

Name of Bacteria	No. of Patients	%Age
No Infection	6	13.37
E. Coli	8	17.77
Pseudomonas	8	17.77
Klebsella	5	11.11
Staph Pyogenes	2	4.44
E.Coli+Pseudomonas	12	26.66
E. Coli+Klebsella	2	4.44
Mixed or more than 2 bacteria	2	4.44
Total	45	100.00

Table V

Pressure Sores

Site of Pressure	Size of Pressure Sore No. of Patients			Total
	Small	Medium	Large	
No Pressure Sore	-	-	-	10
Sacral	10	8	14	32
Trochanteric	4	2	3	9
Mallelar	2	2	-	4
Calcaneal	6	2	-	8

Table VI

Discussion

Our study revealed HO to be more common in adult male in the age group of 20-30 as reported by earlier authors[2,3,4] also. In this series all the changes were found below the level of cord lesion. HO was seen more in the patients with lesion around dorsolumbar spine. In confirmity with the finding of Damanski[4], we did not find HO in patient with injury below L_2. There was in general little difference in the incidence of HO between those with spastic and those with flaccid paralysis[2,3,4,7].

In our series most commonly involved joints were hip joint and knee joint. No HO was observed in upper extremity even in cases of quardriplegia. These finding confirm earlier views of Hardy & Dickson[7], Damanski[4], Ceilier[2], Wharton[22] et al. The average duration between SCI and development of HO was sixteen weeks. Massage and manipulation did seem to have a influence though inconclusive in our series of patients with 44.4% accounting for a definite and 29% doubtful history of manipulation. But majority of cases were unilateral (76%) although paralysis was symmetrical and physiotherapy was given to both legs.

Contrary to the views of Damanski[4], we do not believe in pressure sores contribution to the pathogenesis of HO as 24% of patient in our series never had any pressure sore and 41% had grade-I pressure sore. In 14% of cases, there was no corelation between site of pressure sore and site of HO.

34% of patient never had significant UTI but majority of the patients with severe UTI had severe HO. This could be explained on the basis of impaired renal function thereby imparied ability to exerete calcium and phosphate whose levels in blood are higher because of osteoporotic changes occuring in the bone following functional immobilisation[4,7,16].

Hardy et al[7], Wharton[23] and others have underlined importance of surgical intervention in patients with complete ankylosis of major joints (hip & knee) to gain functional independence, However none of 45 patients in our series required surgical intervention and maintain useful ROM with conservative management.

Conclusions

HO is common in adult age group male patients with injury around dorsolumbar spine with complete lesion and is equally common in spastic and flaccid paraplegics. Hip and knee joints are commonly involved and involvement of upper extremity joints is infrequent even in case of quardriplegia. Massage and manipulation seems to have a variable contribution in genesis of HO. HO may or many not be associated with typical sign of inflammation like local rise of temperature and swelling so progressive restriction of ROM in paraplegics should arouse suspicion of HO. Urinary tract infection seems to play a significant role role whereas pressure sores do not seem to cause genesis of HO at local sites. Treatment of HO in acute stage on the lines of acute myositis ossificans followed by passive ROM exercises retain atleast functionally useful ROM even in severely affected joints.

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