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JOURNAL OF BANGLADESH COLLEGE OF PHYSICIANS AND SURGEONS

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EDITORIAL

Antiretroviral Therapy in Adolescents and Adults in Developing Countries

The acquired immunodeficiency syndrome, or AIDS, was first reported in 1981 as *"Pneumocystis* pneumonia - Los Angeles".[1] Since then, AIDS has become the most devastating disease.

From the time the epidemic began, more than 60 million people have been infected with the human immunodeficiency virus (HIV). At the end of 2002, an estimated 42 million people globally were living with HIV. In developing countries, the majority of new infections occur in young adults. HIV/AIDS was late in coming to Asia. Until 1999, only Cambodia, Myanmar and Thailand had documented nationwide epidemics. The situation is now rapidly changing. In 2002, almost one million adults and children were newly infected with HIV in the Asia and Pacific region bringing to an estimated total of 7.2 million people now living with the virus. By the end of 2001, an estimated 3.97 million Indians were living with HIV/AIDS.[2] In Bangladesh and in some other countries, currently infection rates are low, though risk behaviour is common.[3] Indonesia, the world's fourth most populous country is an example of how suddenly HIV/AIDS epidemic can emerge. It had low rates of HIV for more than a decade, but infection rates are now rapidly rising.[2]

Prevention programmes can keep infection rates lower in specific groups and reduce the risk of spread of HIV among the wider population. Excellent examples in South-East Asia are Thailand and Cambodia, where prevention efforts have probably averted millions of HIV infections.[4] Preventive strategies work and deserve greater support.

Antiretroviral therapy for HIV infected patients was first introduced in 1986. Zidovudine (ZDV), a nucleoside reverse transcriptase inhibitor (NsRTI), was the first antiretroviral drug and reduced deaths and accompanying opportunistic infections in patients with advanced HIV infection.[5] Subsequently, other drugs were introduced. However, the benefits of single drug therapy were short lived due to the emergence of resistance. It was later shown that combining 3 antiretroviral drugs produced more sustained benefit.[6,7,8] New classes of drugs, the protease inhibitors (PI) and non-nucleoside reverse transcriptase inhibitors (NNRTI) allowed the use of more potent antiretroviral regimens. These regimens, consisting of three or more drugs, have resulted in dramatic reduction in HIV levels in blood, markedly improved immune function and significantly reduced morbidity and mortality. Combination antiretroviral therapy has transformed the disease into a treatable and chronic condition. Patients receiving these regimens are less likely to develop opportunistic infections and require less admissions to hospital than patients with untreated disease.[9,10] Antiretroviral therapy also reduces the perinatal and heterosexual transmission.[11,12] Combination of three antiretroviral drugs is expensive, but studies indicate that it is a cost effective use of resources.[13,14]

Millions of people infected with HIV in developing countries face disease and early death unless they receive appropriate medical care. World Health Organisation (WHO) estimated that in 2002, some 6 million in developing countries were in need of antiretroviral therapy. Instead, only 230,000 had access to them, and half of these lived in Brazil. United Nations General Assembly

Special Session on HIV/AIDS (UNGASS) in 2001, urged the complementarity of HIV care and prevention including antiretroviral treatment for people living with HIV/AIDS.

The difficulties related to the use of antiretroviral drugs in developing countries have been medium/long term availability and affordability of drugs and the lack of health infrastructure necessary to use them. Furthermore, there are concerns that difficulties with adherence to complicated medication regimens would promote drug resistance.[15] However, the use of antiretroviral drugs in poorest countries is now an urgent priority and should go hand in hand with programmes to prevent HIV transmission. The reasons for combining AIDS prevention and therapy include a humanitarian rationale, to save children and the fabric of the society, for continuing economic development and to optimize preventive efforts. When treatment is unavailable, less incentive exist to undertake the test, since HIV positive status may mean a death sentence. When testing is linked to therapy, people have an incentive to be tested providing a rational response: primary prevention for HIV negative persons and therapy for HIV positive patients.[16] Brazil, Thailand and Haiti have shown that it is possible to deliver combination antiretroviral therapy in developing countries.[17] Pharmaceutical companies in developing countries like India and Thailand are now producing generic drugs at a much lower cost.

WHO, South East Regional Office (SEARO), has recently published a simplified approach for the use of antiretroviral drugs in HIV infected patients in developing countries.[18] The main indications for the use of antiretroviral drugs include treatment of HIV infected patients, prevention of mother to child transmission and post exposure prophylaxis following an accidental occupational and non occupational exposure to HIV.

The currently available antiretroviral drugs are unable to eradicate HIV infection and treatment is lifelong. In adolescents and adults, treatment is indicated in asymptomatic persons (WHO stage I) with CD4+ T lymphocyte count less than 200/ mm^3. Treatment is also indicated in symptomatic patients WHO Stage II and WHO Stage III with CD4 count less than 200/mm^3 and in WHO Stage IV irrespective of the CD4 cell count.[18,19,20] Twelve antiretroviral drugs are currently in the WHO Model List of Essential Medicines, year 2002. These include five NsRTIs, two NNRTIs, and five PIs. Current guidelines recommend treatment of HIV infection using regimens of 3 or more antiretroviral drugs. A high degree of adherence to medication is necessary for satisfactory response. In resource-constrained countries, monitoring of disease progression and response to therapy is based on clinical assessment and CD4 cell count. Viral load determination performed in developed countries, is often not possible in resource constrained countries due to the cost of the test, lack of laboratory facilities and trained personnel.[21]

Prior to implementing an antiretroviral therapy programme in a resource constrained country like Bangladesh, it is essential to do a situation assessment to estimate the burden of public health problem. Programme planning with clear objectives should be defined including long term sustainability. Adequate measures for implementation at the programme level and health facility level need to be taken to make an antiretroviral therapy programme a success.[18]

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(J Bangladesh Coll Phys Surg 2002; 20 :1-3)

References:

1. Centers for Disease Control and Prevention. *Pneumocystis* pneumonia - Los Angeles. Morb Mortal Wkly Rep MMWR 1981;30:250-2.
2. UNAIDS, WHO. AIDS epidemic update: December 2002. Geneva. Joint United Nations Programme on HIV/AIDS, 2002.
3. National AIDS/STD Program, MOHFW, Government of the People's Republic of Bangladesh. HIV in Bangladesh: where is it going? November 2001.
4. The Thai Working Group on HIV/AIDS Projection. Projections for HIV/AIDS in Thailand: 2000-2020. March 2001.
5. Fischl M, Richmann DD, Grieco MH, et al. The efficacy of azidothymidine (ZDV) in the treatment of patients with AIDS and AIDS-related complex. A double blind, placebo controlled trial. N Engl J Med 1987;317:192-7.
6. Hammer SM, Squires KE, Hughes MD, et al. A controlled trial of two nucleoside analogues plus indinavir in persons with human immunodeficiency virus infection and CD4 counts of 200 per cubic millimeter or less. N Engl J Med 1997;337:725-33.
7. Gulick RM, Mellors JW, Havlir D, et al. Treatment with indinavir, zidovudine, and lamivudine in adults with human immunodeficiency virus infection and prior antiretroviral therapy. N Engl J Med 1997;337:734-9.
8. Staszewski S, Morales-Ramirez J, Tashima KT, et al. Efavirenz plus zidovudine and lamivudine, efavirenz plus indinavir, and indinavir plus zidovudine and lamivudine in the treatment of HIV-1 infection in adults. N Engl J Med 1999;341:1865-73.
9. 2001 USPHS/IDSA. Guidelines for the prevention of opportunistic infections in persons infected with human immunodeficiency virus. http// :www.hivatis.org.
10. Gebo KA, Chaisson RE, Folkemer JG, Bartlett JG, Moore RD. Cost of HIV medical care in the era of highly active antiretroviral therapy. AIDS 1999;13:963-9.
11. Connor EM, Sperling RS, Gelber R, et al. Reduction of maternal-infant transmission of human immunodeficiency virus type 1 with zidovudine treatment. Pediatric AIDS Clinical Trial Group Protocol 076 Study Group. N Engl J Med 1994;331:1173-80.
12. Quinn TC, Wawer MJ, Sekankambo N, et al. Viral load and heterosexual transmission of human immunodeficiency virus type 1. N Engl J Med 2000;342:921-9.
13. Bozzette SA, Joyce G, McCaffrey DF, et al. Expenditures for the care of HIV infected patients in the era highly active antiretroviral therapy. N Engl J Med 2001;344:817-23.
14. Freedberg KA, Losina E, Weinstein MC, et al. The cost effectiveness of combination antiretroviral therapy for HIV disease. N Engl J Med 2001;344:824-31.
15. Farmer P, Lendre F, Mukherjee J, et al. Community based treatment of advanced HIV disease: introducing DOT-HAART (directly observed therapy with highly active antiretroviral therapy). Bulletin of World Health Organisation 2001;79:1145-51.
16. Individual members of the Faculty of Harvard University. Consensus statement on antiretroviral treatment for AIDS in poor countries. Topics in HIV Medicine 2001;9:14-26.
17. Farmer P, Lendre F, Mukherjee J, et al. Community-based approaches to HIV treatment in resource-poor settings. Lancet 2001;358:404-09.
18. World Health Organisation.The use of antiretroviral therapy: a simplified approach for resource-constrained countries.WHO/ICP/ HIV001/2002.
19. World Health Organisation. Scaling up antiretroviral therapy in resource-limited setting: Guidelines for a public health approach. Executive Summary. WHO/HIV/2002.01.
20. World Health Organization. Scaling up antiretroviral therapy in resource-limited setting: Guidelines for a public health approach. WHO/HIV/2002.02.
21. World Health Organisation and UNAIDS. Guidance modules on antiretroviral treatments. Module 5. Laboratory requirements for the safe and effective use of antiretrovirals. WHO/ASD/ 98.1. UNAIDS/98.7.

ORIGINAL ARTICLES

Prospective, Non Blind Trial of Sulphasalazine on Seronegative Spondyloarthropathy

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Summary :

This study was designed to see the efficacy of sulphasalazine in patients with seronegative spondyloarthropathies viz ankylosing spondylitis (AS), juvenile chronic arthritis (JCA) and Reiter's syndrome (RS) / Reactive arthritis (RA). Thirty patients were randomly selected and sulphasalazine was given orally to each patient. 11(36.6%) cases and 10(33.3%) cases showed complete and partial improvement respectively. Individual therapeutic response with sulphasalazine were 61% in ankylosing spondylitis and 77% in JCA respectively. The improvement was significant because statistical analysis showed P<.001.

(J Bangladesh Coll Phys Surg 2002; 20 : 5-12)

Introduction :

The spondyloarthropathies are a group of inflammatory arthritis affecting younger people characterized by seronegative, asymmetrical oligoarthritis with a predilection for axial involvement, enthesopathy, anterior uveitis, familial association and high prevalence of HLA B27 with other extra articular features. It encompasses several disorders such as ankylosing spondylitis, a subset of juvenile chronic arthritis, Reiter's syndrome, reactive arthritis, enteropathic arthritis and psoriatic arthropathy. Ankylosing spondylitis is the commonest disease among the seronegative arthropathies[1].

The prevalence of ankylosing spondylitis varied from virtual nil to 4.2% in several community studies. Calin reported that it was found 4 per 1000 among the males and 0.5 per 1000 among females. Another study showed in India, a prevalence of 4.2% in adult males[3]. The incidence of JCA was 12 per 100,000 and the prevalence varied from 56 to 100 per 100,000 population[4,5] in Western countries. Post dysentric Reiter's syndrome occurs in 1 to 2% of cases[6]. Prevalence varied from 1/1000 to 10% in Western population[7]. In our population, a study done by Alam et al reported incidence of AS, JCA, RS/Re A and psoriatic arthropathy were 3.13%, 2.92%, 0.89% and 0.15% respectively[8].

The natural course of the disease varies from mild symptoms to severe disability leading to death due to cardiac complication or amyloidosis[9,10,11].

Treatment of this group of arthropathies is not satisfactory. NSAIDs help to keep the patient symptoms free but they can not stop the disease process and progression. Sulphasalazine (SSZ) was shown to be effective in AS[12-15]. It is also effective in the treatment of JCA[16,18] and reactive[19,20] arthritis.

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The present study was undertaken with a view to find out the efficacy of sulphasalzine in the treatment of seronegative spondyloarthropathies.

Materials and Methods :

This prospective study was carried out in the department of Physical Medicine (Rheumatology Clinic) and Medicine department of IPGMR now BSMMU, Dhaka between the period from October 1995 to March, 1997 including monthly follow up for 6 months in each case.

Thirty patients of spondyloarthritis which included AS 18, JCA 9 and Reiter's syndrome/ reactive arthritis 3 cases respectively, were included of both sexes, age 10 to 50 years, duration of disease more than 3 months, fulfilment of diagnostic criteria, grade-II & III tenderness and regular intake of NSAD with adequate doses at least for 3 months. The patients who had haematological, hepatic and renal disorders; known G6PD deficiency, known sulphonamide and salicylate toxicity were excluded from the study.

Before starting therapy, total blood count, Haemoglobin percentage, Erythrocyte sedimentation rate, platelet count, RA test, Rose-Waaler test, X-ray of lumbosacral spine, sacro-iliac joints and other involved joints, serum bilirubin, SGPT, blood urea and serum creatinine were done in each patients.

Total dose of sulphasalazine was calculated as 40 mg/kg (max 2 gm), full dose was gradually reached over one month in two divided doses; starting dose was 500 mg daily orally. NSAID was also given in each patients at the same time.

Results :

Thirty patients including 28 male (93.33%) and 2 female (6.67%) were studied male to female ratio of 14:1. The mean (SD) age of the patients was 25.10(8.77) years with a range varying from 12 to 50 years.

The follow up of the patients was done monthly over a period of 6 months in all cases. The follow up parameters (Table-I) were number of involved joints, joints swelling index, joint tenderness index, visual analogue scale, functional index, 80 ft. walking time (secs), schober test (cm), finger to floor distance (cm), ESR (mm in 1st hour), NSAID score, physicians global assessment (PGA) total leucocyte count, haemoglobin percentage (gm%).

The therapeutic response of sulphasalazine on seronegative spondyloarthritis as a whole, showed significant improvement of all parameters except schober test, finger to floor distance, total leucocyte count and haemoglobin percentage (Table-II). In this series 70%(21 out 30) cases showed significant clinical improvement and rest 30%(9 out of 30) minor improvement.

In AS, 61% cases showed significant clinical improvement invloving both axial and peripheral joints (Table-III, IV) and the rest 38.8% showed minor improvement (Table-V).

Majority parameters showed significant improvement in JCA (Table-VII). 77.7% cases responded satisfactorily and in the rest results were not satisfactorily (Table-VI).

Almost 100% patients with RS/ReA showed significant clinical response (Table-VI) and almost all parameters improved significantly (Table-VII).

Adverse reactions due to sulphasalazine were found in 19(54%) cases of which gastrointestinal symptoms were 22.5%. Others included headache 14.5%, jaundice 5.7% and skin rash 5.7% cases respectively.

Table-I

Baseline characteristics of the patients included in the study treated with SSZ (n=30).

Parameters	
Age (years)	25.47 ± 9.70
Duration of illness (year)	4.22 ± 3.07
No. of Jt. involvement	2.40 ± 2.51
Visual analogue scale	7.72 ± 1.39
Jt. swelling index	2.37 ± 3.09
Jt. tenderness index	5.70 ± 3.15
Functional index (0-3)	5.37 ± 1.61
50 ft. walking	24.90 ± 2.75
Schober test (cm.)	3.17 ± 1.25
Finger to floor distances (cm.)	11.53 ± 12.08
Physician's global assessment(0-5)	3.60 ± 0.59
Total leucocyte count	9530.00 ± 1884.26
Haemoglobin (gm%)	10.11 ± 1.49
ESR	38.17 ± 33.56
NSAID score	8.65 ± 4.04

Table-II

Treatment response of individual patients with sulphasalazine (n=30).

Parameters	Pre Treatment	Post Treatment	P
	Mean ± SD,	Mean ± SD	
No. of Jt. involvement	2.40 ± 2.51	0.77 ± 1.28	0.001
Visual analogue scale	7.72 ± 1.39	2.65 ± 1.64	0.001
Joint swelling index	2.37 ± 3.09	0.20 ± 0.66	0.001
Joint tenderness index	5.70 ± 3.15	1.12 ± 1.13	0.001
Functional index (0-3)	5.37 ± 1.61	1.17 ± 1.66	0.001
50 ft. walking (secs)	24.90 ± 2.75	14.17 ± 2.15	0.001
Schober test (cm.)	3.17 ±1.25	3.78 ± 1.30	0.06
Finger to floor distances (cm)	11.53 ± 12.08	10.10 ± 13.31	0.66
ESR (Erythrocyte sedimentation rate)	68.17 ± 33.59	21.90 ± 12.99	0.001
NSAID score	8.65 ± 4.04	2.82 ± 4.23	0.001
Physician's global assessment(0-10)	3.60 ± 0.59	1.70 ± 0.73	0.001
Total leucocyte count (cu mm)	9530.00 ± 1884.26	8600.00 ±1260.27	0.02
Haemoglobin (gm%)	10.11 ± 1.49	10.56 ± 1.50	0.24

Table - III

Treatment response of Ankylosing spondylitis (n=18).

Parameters	Pre Treatment Mean ± SD	Post Treatment Mean ± SD	P
No. of Jt. involvement	2.33 ± 2.76	0.67 ± 1.24	0.02
Visual analogue scale	8.03 + 1.36	2.89 ± 1.44	0.001
Joint swelling index	1.72 ± 2.89	0.00 ± 0.00	0.01
Joint tenderness index	6.58 ± 3.61	1.28 ± 1.31	0.001
Functional index (0-3)	5.94 ± 0.94	1.31 ± 1.81	0.001
50 ft. walking (secs)	24.67 ± 2.64	14.22 ± 2.67	0.001
Schober test (cm.)	2.97 ± 1.19	3.67 ± 1.47	0.13
Finger to floor distances (cm)	13.39 ± 11.46	11.89 ± 13.78	0.72
ESR(Erythrocyte sedimentation rate)	68.56 ± 32.94	23.00 ± 11.72	0.001
NSAID score	9.01 ± 4.07	3.68 ± 4.35	0.001
Physician's global assessment(0-10)	3.61 ± 0.63	1.83 ± 0.84	0.001
Total leucocyte count (cu mm)	9466.67 ± 2003.23	8722.22 ± 1382.05	0.20
Haemoglobin (gm%)	10.10 ± 1.52	10.54 ± 1.59	0.40

Table-IV

Response of sulphasalazine on AS patients with,axial and peripheral joint involvement (n=9).

Parameters	Axial joints (n=9)			Peripheral joints (n=9)		
	Pre Treatment Mean ± SD	Post Treatment Mean ± SD	P	Pre Treatment Mean ± SD	Post Treatment Mean ± SD	P
No. of Jt. involvement	0.00	0.00	-	4.44 ± 2.40	1.33 ±1.50	0.005
Visual analogue scale	7.75 ± 1.68	2.88 ±1.13	0.001	8.13 ± 0.95	2.89 ± 1.76	0.001
Joint swelling index	0.00	0.00	-	3.44 ± 3.30	1.00 ± 1.20	0.01
Joint tenderness index	9.77 ± 5.86	1.88 ± 2.02	0.00f	8.11 ± 5.57	1.67 ± 1.87	0.004
Functional index (0-3)	5.88 ± 0.92	0.89 ± 1.24	0.001	6.00 ± 1.00	1.72 ± 2.83	0.001
50 ft. walking (secs)	24.00 ± 2.17	13.66 ± 1.93	0.001	25.33 ± 3.00	14.77 ± 3.27	0.001
Schober test (cm.)	3.05 ± 1.21	3.77 ± 1.39	0.25	2.88 ± 1.24	3.55 ± 1.60	0.34
Finger to floor distances (cm)	12.33 ± 8.66	10.88 ± 9.25	0.73	14.44 ± 14.19	12.88 ± 17.77	0.84
ESR (Erythrocyte sedimentation rate)	61.22± 25.19	23.44±10.66	0.001	75.88 ± 39.37	22.55 ± 13.32	0.001
NSAID score	10.51+ 3.44	4.53+5.28	0.01	7.50 ± 4.26	2.83 ± 3.26	0.01
Physician's global assessment(0-10)	3.88 ± 0.65	2.11 ± 0.74	0.001	3.33±0.90	1.50 ± 0.88	0.001
Total leucocyte count (cu mm)	9755.55 ±1667.00	8722.00 ±1481.00	0.19	9177.77 ±2283.00	8722.00 ±1365.00	0.61
Haemoglobin (gm%)	10.36 ± 1.75	10.46 ± 1.81	0.90	9.83 ± 1.34	10.62 ± 1.44	0.24

Table - V

Improvement of individual subclasses of seronegative spondyloarthrypathy.

Category of Improvement	AS SSZ (%)	JCA SSZ (%)	ReA/Reiter SSZ
Complete	6(33.3)	3(33.3)	2(66.6)
Partial	5(27.7)	4(44.4)	1(33.4)
Clinically important improvement (Complete + partial)	11(61.1)	7(77.8)	3(1.00)
Minor improvement	7(38.8)	2(22.2)	0(0.0)
Total	18(100)	9(100)	3 (100)

Table- VI

Treatment response of JCA

Parameters	Pre Treatment Mean ± SD	Post Treatment Mean ± SD	P
No. of Jt. involvement	2.0.0 ± 2.29	1.00 ± 158	0.30
Visual analogue scale	7.32 ± 1.48	2.50 ± 2.12	0.001
Joint swelling index	2.89 ± 3.18	0.67 ± 1.12	0.06
Joint tenderness index	4.55 ± 1.84	1.03 ± 0.87	0.001
Functional index (0-3)	4.56 ± 2.01	1.28 ± 1.56	0.001
50 ft. walking (secs)	24.33 ± 2.35	13.89 ± 1.17	0.001
Schober test (cm.)	3.22 ± 1.46	3.83 ± 1.15	0.33
Finger to floor distances (cm)	11.67 ± 13.86	9.89 ± 13.91	0.79
ESR(Erythrocyte sedimentation rate)	69.22 ± 33.73	22.56 ±16.59	0.001
NSAID score	7.10 + 1.94	1.39 ± 4.17	0.001
Physician's global assessment(0-10)	3.61 ± 0.49	1.61 ± 0.49	0.001
Total leucocyte count (cu mm)	9833.33 ± 767.77	8666.67 ±118.03	0.11
Haemoglobin (gm%)	10.42 ± 1.59	10.23 ± 0.93	0.76

Table-VII

Treatment response of Reiter's / Reactive arthritis.

Parameters	Pre Treatment Mean ± SD	Post Treatment Mean ± SD	P
No. of Jt. involvement	4.00 ± 1.00	0.67 ± 0.58	0.001
Visual analogue scale	7.00 ± 1.00	1.67 ± 1.16	0.001
Joint swelling index	4.67 ± 3.79	0.00 ± 0.00	0.09
Joint tenderness index	3.83 ± 1.04	0.50 ± 0.50	0.001
Functional index (0-3)	4.33 ± 2.52	0.00 ± 0.00	0.04
50 ft. walking (secs)	28.00 ± 3.46	14.67 ± 0.58	0.001
Schober's test (cm.)	4.17 ± 0.29	4.33 ± 0.58	0.68
Finger to floor distances (cm)	0.00 ± 0.00	0.00 ± 0.00	-
ESR (Erythrocyte sedimentation rate)	62.67 + 50.01	13.33 ± 7.64	0.17
NSAID score	11.17 + 7.69	2.00 ± 3.46	0.13
Physician's global assessment(0-10)	3.50 ± 0.87	1.17 ± 0.29	0.01
Total leucocyte count (cu mm)	9000.00 ± 2000.00	7666.67 ± 577.35	0.33
Haemoglobin (gm%)	9.20 ± 0,72.	11.67 ± 2.31	0.15

Discussion :

The present study was done in the Rheumatology clinic and Medicine department in the

Institute of Postgraduate Medicine and Research now Bangabandhu Sheikh Mujib Medical University in a prospective manner. Period of study was between October, 1995 to March, 1997. Thirty patients of both male and female were included in the study where male (93.33%) to female (6.7%) ratio was 13.88:1.

Age ranged from 12 to 50 years, with a mean (SD) was 25.10(8.77) years. Age of the subclasses 28.40(7.88) in AS, 16.82(2.63) in JCA and 29.56(9.36) years in RS/ReA respectively.

In our series, the average duration of illness was 4.55(3.27) years with a range from 6 months to 15 years. This wide range was due to the included of subclasses of the disease. The lowest duration was 6 months.

Successful use of sulphasalazine in the treatment of AS, JCA and RS/ReA was shown by several authors such as Nissila 1988[21]; Yamane 1993[22]; Carlos 199023; Owen ET 1979[24].

Number of joints involved in the study was 2.40(2.51) which included only peripheral joints; axial joints had not been accounted because they were inaccessable and difficult to count.

After 6 months, treatment with sulphasalazine on seronegative spondyloarthritis on 30 patients changes of the all assessment parameters were noted significantly pre and post treatment (P<.001) except schober test, finger to floor distance, haemoglobin percentage and total leucocyte count (Table-II).

The follow up parameters used in this study for seronegative spondyloarthritis were age, duration of illness, number of joint involvement, visual analogue scale, joint swelling index, joint tenderness index, functional index, 50 ft. walking, schober test, finger to floor distances, physicians global assessment, total leucocyte count, haemoglobin percentage, erythrocyte sedimentation rate and NSAID score shown in Table-I.

Eighteen patients of AS was treated by sulphasalazine showed significant improvement as all the parameters decreased after treatment except schober's test, finger to floor distance, total leucocyte count and haemoglobin percentage (Table-III).

In this study, mean value of involved joint before and after treatment with sulphasalazine on AS patients was 2.33(2.76) and 0.67(2.24), P<0.02 which was significant. This was comparable with the findings of Nissila et all[21] who showed 1.2(2) and 1(1.3) pre and post treatment, P value was <0.05. Joint swelling index in AS patients on SSZ were 1.72(2.89) and 0.00(0.00) pre and post treatment respectively (P<.01).

Mean increase in the height of lumber spine (Schober test) in AS patients on SSZ was 2.97(1.19) and 3.67(1.4) before and after treatment (P<0.13) but this difference was significant in Nissila's series. Similarly in the study of Nissila (SSZ on AS) finger to floor distance were 18.7(15.1) cm and 15.7(7.9) cm pre and 6 months post treatment (P<.02) which was not consistent with our result 13.39(11.46) and 11.89(13.78) before and after treatment (P<0.72). The differences in total leucocyte count and haemoglobin percentage were not significant in AS, JCA and RS/ReA patients on SSZ, so these two parameters were not taken as valuable guide to follow up the therapeutic response with SSZ.

It is to be noted that SSZ cured significantly symptoms of 61% (11 out of 18) cases involving both axial and peripheral joint respectively in our study (Table-V) and the rest 39%(7 out of 18) showed minor improvement.

In our series, seven parameters showed significant improvement with SSZ for six

month trial (P<.001) but the remaining parameters did not show significant changes (Table-VI).

Joint swelling index in JCA with SSZ pre and post treatment values were 2.89(3.18) and 0.67(1.12) respectively (P<0.06) which was similar with corresponding findings of Vaidya et al (1996). But mean number involved joint both axial and peripheral did not consistent with Vaidya's findings as our result was 2.00(2.29) (p value <0.30) while his result was 5.4(3.65) and 1.51(2.88) before and after treatment (P<0.05) respectively.

It is to be mentioned that 77.7% cases showed significant improvement involving both axial and peripheral joints and rest 22.3% did not show significant change with SSZ on JCA patients in this study (Table-V).

In RS/ReA significant improvement was observed in respect of number of joint involved, visual analogue scale, joint tenderness index,functional index, 50 ft walking time and physicians global assessment. ESR and NSAID score also did not change significantly. This may be due to very small number of patients included, so conclusion can not be drawn.

Trnavsky et al[20] showed in their study that significant improved was observed in 15 cases out of 18 patients of reactive arthritis (P<.02).

References :

01. Dougados M, Patrick B, Amor B : Sulphasalazine in ankylosing spondylitis a double blind controlled study in 60 patients. BMJ 1986; 293: 911-914.

02. Calin A : Ankylosing spondylitis. In: Kelly WN, Haris ED Jr. Ruddy S et al, eds. Text book of rheumatology, 3rd ed vol 2 Philadelphia: Saunders 1989; 1021-37.

03. Ball V Gene: Ankylosing spondylitis, In McCarthy Daniel; Koopman J. William, eds. Arthritis and allied condition, Philadelphia, Lea & Febiger 1993; 1051-60.

04. Gare BA, Fasth A, Anderson J : Incidence and prevalence of JCA; a population survey. Ann Rheum Dis 1987; 46: 277-81.

05. Hoyeraal HM : Methodological problems: Juvenile chronic arthritis. Scand J Rheumatol 1987; 66: 67-74.

06. Arnett FC : Seronegative spondyloarthropathies Bull Rheum Dis 1987; 37: 1-12.

07. Calin A : Seronegative spondyloarthritidies. Medicine International 1994; 7: 145-51.

08. Alam MN, Haq SA, Moyenuzzaman M, Samad MA, Chowdhury Q, Das KK et al

Rheumatological disorder in IPGMR, Dhaka. Bangladesh J. Medicine 1996; 7: 1-7.

09. Jimenz Balderas FJ, Mintz G: Ankylosing spondylitis; Clinical course in women and men. J Rheumatol 1993; 20: 2069-72.

10. Keat A : ABC of Rheumatology, Spondyloarthropathis. BMJ 1995; 310: 1321-24.

11. Ford DK : Reiter's syndrome: Reactive arthritis. In McCarty Daneil; Koopman

J; Willium eds, Arthritis and Allied conditions 11th Edition Philadelphia, Lea & Febiger 1989; 944-952.

12. Kirwan J, Edwards E, Huitfeld B et al : The course of established ankylosing

spondylitis and the effects of sulphasalazine over 3 years. Br J Rheumatol 1993; 32: 729-733.

13. Fraser SM, Sturrock RD : Evaluation of sulphasalazine in ankylosing spondylitis an interventional study. Br J Rheumatol 1990; 29: 37-39.

14. Davis MJ, Dawes PT, Beswick E et al : Sulphasalazine therapy in ankylosing spondylitis, its effect on disease activity, immunoglobulin A and complex immunoglobin A a AT. Br J Rheumatol 1989; 28: 410-13.

15. Taylor HG, Beswick EJ : Sulphasalazine in ankylosing spondylitis: A radiological,

clinical and laboratory assessment. Clinical rheumatology 1991; 10: 43-48.

16. Joo SR, Veys EM, Meilants H, VanWerveke S, Goemaere S : Sulphasalazinetreatment in juvenile chronic arthritis: An open study (asbstract). J Rheumatol 1991; 18: 880-4.

17. Settas LP, Alexious P, Dimitriadis G, Mata-tsi E, Souliou E, Papapanagiotou J Effects of sulphasalazine in patients with JCA. Acta Universitatis Carolinae Medica 1991; 3 7: 76-79.

18. Rose CD, Singsen BH, Elchenfield AH, Goldsmith DP : Athereya BH. Safety and efficacyof MTX therapy on JRA. The journal of Paediatrics 1990; 117: 653-59.

19. Meilants H, Veys EM and Joos R : Sulphasalazine in the treatment of enterogenic reactive synovitis and ankylosing spondylitis with peripheral arthritis. Clin Rheumatol 1986; 5: 80-83.

20. Trnavasky K, Peliskova Z and Vacha J : Sulphasalazine in the treatment of reactive arthritis. Scand J Rheumatol 1988; 67: 76-79.

21. Nissila M, Lehtinen K, Leirisalo Repo M, Luukkainen R, Mutru O, Yli-Kerttuta U Sulphasalazine in the treatment of ankylosing spondylitis. A twenty six week placebo controlled clinical trial. Arthritis and Rheumatism 1988; 31: 1111-16.

22. Yamane K, Saito C, Natsuda H, Yuhara T, Akama T, Suzuki H et al : Ankylosing spondylitis successfully treated with methotrexate Internal Medicine 1993; 32: 53-6.

23. Owen ET and Cohen ML: Methotrexate in Reiter's disease. Ann Rheum Dis 1979; 38: 48-50.

24. Vaidya HR : Efficacy of sulphasalazine on JCA. Thesis submitted for fulfilment of requirement of Doctor of Medicine (MD) degree from Dhaka University, 1996.

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Two Methods for Hysterectomy : A Randomized Prospective Study

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Summary :

Objective: To detect the difference in preoperative and short term postoperative outcomes between vaginal and abdominal hysterectomy.

Study Design: Randomized controlled trial.

Setting: A private clinic.

Sample size: Two hundred and ten women scheduled for hysterectomy for benign conditions of uterus where selected.

Methods: Randomization into two treatment arms, vaginal hysterectomy (n=120) and total abdominal hysterectomy (n=90).

Main outcome measures: Mean size of uterus, type of operation, peroperative course, postoperative complication, time needed for operation and duration of hospital stay.

Results: The mean age and parity were 41.82±6.39 vs 40.17±4.75 yrs and 4.12±1.60 versus 3.28±1.75 in the two groups respectively. The indications for hysterectomy were fibroid uterus, DUB, PID, Adnexal mass and pelvic endometrioses. The size of uterus was 8 wks. in most of the cases, in cases of larger sized uteri, more hysterectomies were done abdominally, but in some cases during operation by vaginal route, the size of uterus was reduced by enucleation of the fibroid and then the operation was completed. Hysterectomy was done in 95% and 47.7% in two groups, whereas hysterectomy with unilateral salpingo-oophorectomy was done in 0.8% and 30% and hysterectomy with bilateral salpingo-oophorectomy was done in 4.2% and 23.30% in Group-1 and Group-II respectively.

Dissection was difficult in 18.33% and 4.44% respectively in Group-I and Group-II. Time needed for operation was 37.38±12.83 and 41.13±8.51 in Group-I and Group-II respectively and the length of hospital stay was 5.32±0.8 and 6.40±0.2 in the two groups respectively.

Conclusion: Vaginal hysterectomy should be a primary method for uterine removal. It is a safe, feasible and patient friendly method. It is a less invasive technique with benefits, which include shorter hospital stay and faster convalescence. It is the surgical method of choice for benign conditions other than utero vaginal prolapse.

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Introduction :

Hysterectomy is one of the most common major operation with 72,362 procedures performed per year in the National Health Services in England from 1993 to 1994 and 592,000 procedures performed in the United States in 1990[1]. The chance of having a hysterectomy by the age of 55 for women in the United Kingdom has been estimated as 1 in 5[2]. All large scale surveys of hysterectomy practice have shown that 70-80% of hysterectomies are performed by the abdominal approach. In the management of utero-vaginal prolapse, the vaginal route is nopmally used, but this indication accounts for only approximately 10% of cases[3].

Traditionally, the vaginal route is preferred for prolapse uterus in our country, but this route can also be used for cases of benign conditions of the uterus with non-descent.

Vaginal hysterectomy has been found to be associated with less febrile morbidity, less bleeding necessciating transfusion, shorter hospitalization and, faster convalescence than abdominal hysterectomy[3]. There is evidence for lower morbidity and a quick

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recovery in patients undergoing vaginal compared with abdominal hysterectomy[3]. The question then arises as to why is it that relatively few hysterectomies are performed vaginally. Training and experience in vaginal surgery appear to be the major determinants for the type of hysterectomy women are offered. An abundance of well documented articles show that the abdominal approach is associated with a higher incidence of complications[4]. Also the vaginal technique is regarded by many gynaecologists as the most cost effective[5]. There are few randomized controlled trials to compare results of different surgical technique for hysterectomy and to date only uncontrolled or case controlled studies have compared all the three methods[6].

The aim of the study is to detect the differences in peroperative and short term postoperative outcomes between vaginal and abdominal hysterectomy.

Materials and Methods :

Two hundred and ten women scheduled for hysterectomy for benign conditions of uterus were selected randomly using the random selection table.

Inclusion criteria

- Menorrhagia, leiomyoma
- Size of uterus not more than 12 weeks
- Benign disease of uterus
- No previous history of abdominal operation
- No descent of uterus

Exclusion criteria

- Size of uterus more than 12 weeks
- Malignant condition of uterus and cervix
- Previously known dense adhesion
- Utero vaginal prolapse

A protocol was made for data collection. Detailed history and thorough clinical examination was done in each case. Particular attention was given to operative time, per and post operative complications, amount of blood loss and hospital stay. Out of 210 patients, 120 underwent operation by the vaginal route and 90 underwent operation by the abdominal route.

Operative Technique :

During abdominal hysterectomy the abdomen was opened and closed according to the surgeons preference but in most of the cases, Pfannenstiel incision was made. The uterus was removed by extrafacial technique and the vagina closed. During vaginal hysterectomy the peritoneal folds were opened and ligaments and uterine vessels were divided and ligated. The uterus was removed, the peritoneum closed, followed by suturing of vaginal vault.

Patients were followed from time of admission to discharge. Type of anaesthesia was chosen by the anaesthesiologist. All of the operations were performed under spinal anaesthesia. The time of operation was calculated from the beginning of operation to end. During operation the following parameters were recorded.

- Peroperative blood loss
- Number of suture materials needed
- Peroperative and postoperative complications

All patients had at least one dose or prophylactic antibiotic peroperative i.e. Inj. Metronidazole 1 gm I.V stat and Inj. Cephradine 1 gm I.V stat.

During post operative period, fever, pervaginal bleeding, urinary tract infection (UTI), haematuria, wound infection, wound dehiscence and length of hospital stay were ascertained. Statistical analysis was done.

Results :

The mean age was similar in Group-I and Group-II. The mean parity was 4.12±1:60 in Group-I and 3.28±1.75 in Group-II. This difference is incidental.

Surgical indications are listed in table-II. More cases were done by abdominal route for fibroid uterus and adnexal mass and pelvic

endometriosis. Similarly, where there was definite adnexal mass (clinically evident and suggested by ultrasonography) it was done per abdomen. In cases which were suspected to have pelvic endometriosis, vaginal route was avoided for risk of encountering adhesions. Considering the size of the uterus before surgery although larger size uteri were more done abdominaly, in some cases during operation by vaginal route, the size of the uterus was reduced by enucleation of the fibroid and then the operation completed.

While selecting patients for vaginal route, more free uterus were chosen so that dissection and delivery of uterus would be easier. The condition of the cervix was an incidental finding.

The reason is that during the operation through the vaginal route, if the adnexal were apparently healthy, they were not removed, but during abdominal hysterectomy if there was the slightest doubt regarding the condition of the ovaries, the ovaries were removed also. It may be mentioned here that as this was only the beginning of attempting hysterectomy by the vaginal route so -more advanced techniques could not be applied. As more and more cases will be done, we will also get acquainted with the techniques of removing the ovaries and so ovaries can be removed as frequently during vaginal operation as done during the operation through the abdominal route.

The dissection was difficult in 22 (18.33%) cases through the vaginal route as compared to the abdominal route. The difficulties were in some cases due to narrow approach and sometimes due to high up uterus. Bringing out uterus was also difficult in 19 (15.83%) cases in the same route. Dissection was difficult in 4 cases (4.44%) in abdominal route due to dense adhesions. These were cases of PID with severe adhesion. The complication during operation was negligible. There was haemorrhage which was controlled. Slippage of ligature occurred in 2 cases of vaginal hysterectomy, which was again secured.

The time needed for operation was shorter in Group-I than in Group-II i.e 37.38±12.83 minutes in Group-I and 41.13±8.51 minutes in Group-II, the time being less needed in the vaginal route and the difference was statistically significant ($P < 0.05$).

In few cases there was some complication during operation shown in Table-VIII.

The requirement of postoperative analgesic was analysed for 1st and 2nd postoperative day. Patients with abdominal hysterectomy required different types of analgesic in increasing doses.

The experience of pain in postoperative period was found to be more in patients undergoing abdominal hysterectomy than in those for vaginal hysterectomy. This is, because there is no abdominal scar, so less pain in the patients undergoing operation by vaginal route. The mean hospital stay was 5.32±0.81 days in vaginal cases and 6.40±0.21 days in abdominal cases and the difference was statistically significant.

Unusual thing about this study is that all the operations have been performed by the same surgeon. This has an advantage that there will be uniformity of findings and the time needed for surgery will be correctly ascertained.

Table-I

Patient characteristic

	Group-I (n=120) Mean ± SD	Group-II (n=90) Mean ± SD	P value[a]
Age (years)	41.82±6.39	40.17±4.75	
Parity	4.12 ± 1.60	3.28 ± 1.75	

[a]Unpaired student's 't' test

Table-II

Indication for hysterectomy

	Group-I (n=120) no. (%)	Group-II (n=90) no. (%)	Total No. (%)
Fibroid uterus	40 (33.33)	48 (53.33)	88 (43.33)
DUB	48 (40.83)	16 (17.77)	65 (29.3)
PID	28 (23.33)	20 (22.22)	48 (22.77)
Adnexal mass	3 (2.5)	6 (6.67)	9 (4.58)
Pelvic endimetriosis	0 (00)	5 (5.55)	5 (5.55)

Table-III

Size of uterus condition of Fornics

Size of uterus	Group-1 (n=120) no. (%)	Group-11 (n=90) no. (%)	P value
6 wks.	34 (28.33)	11 (12.22)	
8 wks.	70 (58.33)	55 (61.11)	0.019
10 wks.	5 (4.16)	9 (10.0)	
12 wks.	6 (5.0)	11 (12.22)	
14wks.	5 (4.16)	4 (4.44)	

x2 =11.749, df—4, P=0.019 (<0.05) significant

Table-IV

Condition of fornics

Free	109 (90.83)	50 (55.55)	
Thickened	4 (3.33)	25 (27.77)	0.001
Fixed/adnexal mass		1 (0.8)	10 (11.11)

x2 = 40.783, df—3, P=0.000 (<0.001) highly significant

Table-IV

Condition of cervix

	Group-I (n=120) no. (%)	Group-II (n=90) no. (%)	P value
Healthy	50 (41.66)	66 (73.33)	
Congested	58 (48.33)	20 (22.22)	0.000
Hypertrophied	12 (10.0)	4 (4.44)	

x2 =11.576, df—2, P=0.000 (<0.001) highly significant

Table-V

Type of operation

Type of operation	Group-I (n=120) no. (%)	Group-II (n=90) no. (%)	Total
Hysterectomy	114 (95.0)	42 (46.7)	156 (74.3)
Hysterectomy with unilateral salpingo-	1 (0.8)	27 (30.0)	28 013.3)
Hysterectomy with bilateral salpingo-oophorectomy	5 (4.2)	21 (23.30)	26 (12.4)

X2 = 64.245, df=2, P=0.000 (<0.001) highly significant

Table-VI

Per operative course

Dissection	Group-I (n=120) no. (%)	Group-II (n=90) no. (%)	P value
Easy	98 ()	89 (98.88)	0.028
Difficult	22 (18.33)	4 (4.44)	
x2 = 4.810, df=1, P=0.028 (<0.05) significant			
Bringing out uterus			
Easy	98 (81.66)	89 (98.88)	0.015
Difficult	19 (15.83)	1 (1.1 l)	
Peroperative Complication			
Haemorrhage	6 (5)	12 (13.33)	
Slippage of ligature	2 (1.66)		
x2 = 5.901, df=I, P=0.015 (<0.05) significant			
Time needed for operation Mean±SD	Mean±SD	P value	
Time (min)	37.38±12.83	41.13±8.51	10.0450

[c] *Unpaired student's 't' test significant at P<0.05*

Table-VII

Postoperative complications

Complication	Group-I (n=9) no. (%)	Group-II (n=34) no. (%)	Total
Fever	8 (88.88%)	12 (35.29%)	20 (62.08)
UTI	-	6 (17.65%)	6 (17.65)
Haemorrhage	I (11.11)	-	1 (1 I.1 I)
Pervaginal bleeding		4 (11.76%)	4(11.76)
Wound infection		12 (35.29%)	12 (35.29)

Table-VIII

Type and number of postoperative analgesics used

Vaginal / Abdominal	No. Doses 1st Gr-I / Gr-II	2nd Gr-I / Gr-II	3rd
First 24 hours			
• Inj. Pethedine	106/84	0/4	
• Inj. Diclofenac	106/81	1/1	
• Diclofcnac supp	19/11		
• Inj. Toradol	19/11		
Second 24 hours			
• Inj. Pethedine	1/2		
• Inj. Diclofenac	2/2	1/1	
• Diclofenac suppository	86/4	20/68	1/13
• Inj. Toradol	4/0		

Table-IX

Hospital stay

Table	Group-I	Group-II	P value[a]
	n=120	n=90	
	Mean ±SD	Mean ±SD	
Length of hospital stay	5.32±0.81	6.40±0.21	0.039

[a] unpaired students 't' test; significant at P<0.05.

Discussion :

The aim of this study was to compare two surgical methods for hysterectomy. The route of operation was determined prior to surgery. The unfavourable characteristic for vaginal surgery was lack of utero vaginal prolapse sufficient to require vaginal repair, enlarged uterus upto the size of 12 weeks gestation and a need for oophorectomy.

Dorsey JW et al[9] reported that no patient with uterus >12 wks. underwent vaginal hysterectomy but 30.6% with uterus >12 wks. were operated by laparoscopic assisted vaginal hysterectomy (LAVH). The indications for hysterectomy in this study were fibroid uterus, DUB, PID and adnexal mass, endometrium. Similar indication for hysterectomy has been reported by others[7,8].

In the present series, uteri with >12 wks. were not operated vaginally, but in some cases, even uteri of 14 weeks gestation size was done through this route but prior reduction of size was done by enucleating of the fibroid. As larger size of the uterus has been formed to be a major hindeerance to the approach through the vaginal route[7,8,9,10], different authors have described various techniques to reduce the size of the uterus, prior to removed through the vagina. Adam Magos et al[11] selected women with fibroid uterus between 14-20 weeks of gestation and described bisection, myomectomy,

morcellation and coring to reduce the uterine size. Vaginal uterine morcellation is the key to a successful operation and obviates the need for either abdominal or laparoscopically assisted hysterectomy solely to deal with moderate uterine enlargement[12,13].

A comparative study performing vaginal hysterectomy with or without morcellation provide that morcellation is safe and facilitates the vaginal removal of moderately enlarged uterus without increasing perioperative morbidity[14]. It is now proved that LAVH is preferred to abdominal hysterectomy, but involves knowledge of laparoscopic surgery as well as vaginal hysterectomy. There is sufficient evidence to suggest that if vaginal hysterectomy can be done it is preferred to LAVH [15].

Removal of the ovaries was initially thought to be a contraindication to the surgery through the vaginal route[7,8] but, the need to perform oophorectomy should not any more be considered a contraindication to vaginal hysterectomy. Romawa PB[16] has described that among procedures done in conjunction with vaginal hysterectomy, in 21% cases unilateral or bilateral adnexectomy was done. In 97.5% of cases both the ovaries were removed vaginally during vaginal hysterectomy salpingo-oophorectomy, oophorectomy, and transvaginal endoscopic oophorectomy utilizing endoloop sutures or bipolarelectric surgery[17]. The technique of vaginal oophorectomy at vaginal hysterectomy is a safe procedure for the experienced surgeons, the favourable factors being slim patient adequate vaginal space, pliable soft tissue multiparity, uterine descent and accessory ovaries free from pathology[18]. The visibility and accessibility provided by vaginal hysterectomy make it possible to grade the position of the ovaries accurately and to determine whether this can be removed transvaginally. Good surgical practice dictates that visibility and accessibility be the primary criteria for selecting the route of oophorectomy. It has been demonstrate that the ovaries are visible and accessible to transvaginal removal in most patient[19].

The requirement of post operative analgesic was less in the operation performed by the vaginal route as compared to the abdominal route. The pain experienced by the patients were also less in Group-I. Similar results have been reported by others[7,8,18].

The hospital stay has been found to be 5.32±0.8 in Group-I and 6.40±0.2 in Group-II. The difference is statistically significant ($P<0.05$). As the patient is more comfortable after operation through the vaginal route it was possible to discharge them from the hospital earlier as compared to the patients with a wound in the abdomen in Group-II who required a longer hospital stay. Christian Ottoson's study[8] showed that abdominal hysterectomy required on average a longer hospital stay of one day and one additional week of convalescence compared with traditional vaginal hysterectomy. The length of hospital stay reported by Dorsey JH et al[9] was 3.5 and 4.4 days for total vaginal hysterectomy and total abdominal hysterectomy respectively. P Bratila[16] reported that post-operative stay was limited to one day, only in 37 patients among 1130 vaginal hysterectomies performed (3.27%). A shorter hospital stay after vaginal hysterectomy has also been reported by others [7,13,14,18].

The cost of the operation was significantly lower in Group-I as compared to Group-II. Reduction in postoperative analgesic required, shorter hospital stay are the factors for the reduced cost. A cost of analysis for abdominal hysterectomy, LAVH and vaginal hysterectomy by Ransom, SB[20] has revealed that vaginal hysterectomy was significantly more cost effective for permanent management of primary menorrhagia than LAVH and total abdominal hysterectomy. Cost effectiveness of the vaginal route has also been described by Anthony Davies et al[7].

Conclusion :

Vaginal hysterectomy should be a primary method for uterine removal. It seems to be a safe, feasible and patient friendly method. It is a less invasive technique with benefits, which include shorter hospital stay and faster convalescence. It is the surgical method of choice for benign conditions other than utero vaginal prolapse.

As there are obvious advantages of hysterectomy performed vaginally, there would be a major impact on the vaginal hysterectomy rate if gynaecologists were trained to carry out vaginal surgery when there is no significant uterine prolapse; when uterus is enlarged or when oophorectomy is indicated.

References :

1. Wilcox LS, Koonin LM, Pokras R, Strauss LT, Xia Z, Peterson HB. Hysterectomy in the United States, 1988-1990. Obstet Gynaecol 1994; 83: 549-55.
2. Vessey MP, Villard-Mackintosh I, McPherson K, Coulter A, Yeates D. The epidemiology of hysterectomy; findings in a large cohort study. Br. J Obstet Gynaecol 1992; 99: 402-7.
3. Dicker RC, Greenspan JR, Strauss LT, Cowart MR, Scally MJ, Pterson HB, et al. Complications of abdominal and vaginal hysterectomy among women of reproductive age in the United States. The Collaborative Review of Sterilization. Am J Obstet Gynaecol 1982; 144: 841-8.
4. Kovac SR, Cruikshank SH, Retto HE Laparoscopy-assisted vaginal hysterectomy. J Gynaecol Surg 1990; 6: 185-93.
5. Meeks GR. Harris RL. Surgical approach to hysterectomy: abdominal, laparoscopy-assisted or vaginal. Clin Obstet Gynaecol 1997; 40: 886-894.
6. Olsson JH, Ellstrom M, Hahlin M. A randomized prospective trial comparing laparoscopic and abdominal hysterectomy, Br. J Obstet Gynaecol 1995; 103: 345-350.
7. Davis, A, Vizza Bournas N, O'Connor H and Magos A. How to increase the proportion of hysterectomies performed vaginally. Am J Obstet Gynaecology 1998; 179(4): 1008-12.
8. Ottosen C, Lingman G, Ottosen L. Three methods for hysterectomy; a randomized, prospective study of short term outcome. Br. J of Obstet Gynaecol 2000; 107: 1380-1385.
9. Dorsey JH, Steinberg EP, Holtz PM. Clinical indications for hysterectomy route: patient characteristics or physician preference. Am J Obstet Gynaecol 1995; 173(5): 1452-60.
10. Dancoisne P, Abossolo T, Orvain E, Tauaillon J, Sommer JC, Riviere JP, Amat D. Criteria for choice of vaginal hysterectomy for lesions other than invasive cancer. Rev- Fr-Gynaecol-Obstet, 1994; 89(1): 11-4.
11. Magos A, Bournas N, Sinha R, Richardson RE and O'Connor H. Vaginal hysterectomy for the large uterus. British J of Obstet and Gynaecol, 1996; 103: 246-251.
12. Unger JB. Vaginal hysterectomy for the woman with a moderately enlarged uterus weighing 200 to 700 grams. Am J Obstet Gynaecol, 1999; 180(6) 1: 1337-44.
13. Hoffman MS, DeCesare S and Kalter C. Abdominal hysterectomy versus transvaginal morcellation for the removal of enlarged uteri. Am J Obstet Gynaecol. 1994; 171(2): 309-15.
14. Doak DK and Mao J. Vaginal hysterectomy with and without morcellation: The university of New Mexico Hospital's Experience Obstet Gynaecol, 1996, 88(4): 560-563.
15. Sheth SS. Vaginal Hysterectomy. FIGO World Congress. Washington DC, USA, September 3-8, 2000.
16. Romania PB. Vaginal hysterectomy on non-prolapsed uteri-our clinical experience FIGO. Washington DC, USA, 2000; 3-8.
17. Dvaies A, O'Connor H, Magos AL. A prospective study to evaluate oophorectomy at the time of vaginal hysterectomy. Br. J Obstet Gynaecol. 1996; 103(9): 915-20.
18. Sheth SS. The place of oophorectomy at vaginal hysterectomy. Br. J of Obstet, Gynaecol, 1991; 98: 662-666.
19. Kovac SR Cruikshank SH. Guidelines to determine the route of oophorectomy with hysterectomy. Am J Obstet Gynaecol, 1996; 1483-88.
20. Ransom SB, McNeeley SG, White C, Diamond MP. A cost analysis of endometrial ablation abdominal hysterectomy, vaginal hysterectomy, and laparoscopic-assisted vaginal hysterectomy in the treatment of primary menorrhagia. J Am Assoc Gynaecol Laparosc. 1996 Nov; 4(l): 29-32.

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Depression in the Chief Stroke Caregivers - Analysis of 297 Caregivers

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Summary :

We compared the incidence and severity of depression among the chief caregivers (spouses, 63%; children, 37%) of stroke patients at 3 and 12 months after stroke. Two stroke registers were used and factors influencing depression were analyzed. At 3 months, 42% caregivers and 17% of controls were depressed (Odds Ratio, 3.47; 95% Confidence Interval, 1.48 to 4.14; Chi-square value 23.72 with P <.001) and the difference was maintained at 12 months 41.5% versus 18%(Odds Ratio, 3.26; 95% Confidence Interval, 1.47 to 4.16; Chi-square value 20.96 with P <.001). Severe SSS long-term score (< 42 points) at 3 months among the patients were associated with the depression of the caregivers at 3 months in the univariate analysis. Other univariate risk factors for depression of caregivers at 3 months were female sex and family history of stroke.

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Introduction:

As the third leading cause of death and the most common disabling disease, stroke has an enormous emotional impact on both patients and their family members[1]. This is especially true for the western societies with aging populations. Average life span of Bangladeshi people has been increased. So, the incidence of stroke is also increasing. Depression is an important consequence of stroke and it influences stroke recovery[2,3,4,5,6,7,8]. In spite of this, there is no population-based study in now country. Even in western countries there are only few population-based studies of the incidence and severity of depression after stroke in patients and among their caregivers [9,10,11,12]. The aim of the study was to determine the occurrence and severity of depression at 3 and 12 months after stroke among the chief caregivers and the analysis of factors influencing the outcome of depression and its severity.

Materials and Method :

Stroke registers were maintained - one in Barisal town and another in Mitford Hospital under Neurology Department. The registers contain the details of patients and their major caregivers. In Barisal, the register was kept from January, 1998 and in Mitford Hospital it was kept from June, 2000.Total period of study was from January, 1998 to December, 2000. The study population covered the whole Barisal division and part of Dhaka district.

Both health centers had been headed by a chief physician and a variable numbers of doctors including a psychiatrist were included in the team. Stroke patients and their family members who would take care of the patients were actively encouraged to participate in care giving of the patients. The patients and caregivers were also provided adequate knowledge they needed for adapting to life after stroke. A total of 297 patients with first-ever stroke and 297 caregivers aged 15 and older were registered.

The evaluation of the patients and caregivers took place upon their attendance to the

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center after the onset and at 3-and 12-months follow-ups. The trained study coordinators in each study center interviewed the patients and their chief caregivers and their controls. Then Scandinavian stroke scale (SSS) were applied at onset and at 3-and 12-months; the Beck's Depression Inventory (BDI) at 3-and 12-months; and quality of life assessment at the onset and at 12 months [13,14,15,16,17,18,19,20,21].

Three months after the stroke, 212 patients were still alive and BDI was performed for 161 caregivers (of whom 152 lived with stroke victims and others did not). At 12 months, BDI was repeated for 156 caregivers (of whom 149 lived with the stroke victims and others did not) of 195 stroke survivors. Filled questionnaires were rejected if more than one third of 21 items were found blank in the BDI instrument; or the time limit of evaluation exceeded (6 weeks at 3 months and 2 months at 1 year). Very few caregivers refused to participate in the evaluation.

Out of 297 caregivers of stroke survivors, 161 BID datas were available. Caregivers with a BID assessment included 81 males and 80 females. Age and sex matched another 161(81 males and 80 females) were also included in this study. The mean age of the caregivers were 54.9 ± 10.5 years for females and 51.7 ± 13 years for males.

The categorical variables were compared with use of the X^2 test and Odds Ratios were calculated for risk factors of depression [22,23]. A cutoff point of >60 years was used for age. For analysis the continuous variables were dichotomized as follows. SSS (maximum of 22) was dichotomized into patients with severe symptoms (<14 points) and those with mild to moderate symptoms (>14 points) were used.

We used the original version of the BDI [14], which has been validated in stroke patients [24]. The cutoff point for depression was >10 points, which has been used in the evaluation of patients with somatic illness[25,26]. The depression was categorized as follows: 0 to 9 points, null to minimal; 10 to 18 mild to moderate; 19 to 29, moderate to severe; and 30 to 63, severe[17,25,26]. The latter three categories (from 10 through 63 points) were used in assessing the severity of depression.

Results :

At 3-months, 42% caregivers were depressed which is higher than that of controls(17%) (Odds Ratio 3.47; 95%'confidence Interval 1.48 to 4.14; Chi-square value 23.72 with P <.001). This difference was maintained at 12-months (41.5% versus 18%) with Odds Ratio 3.26; 95% Confidence Interval 1.47 to 4.16; Chi-square value 20.96 with P <.001 (Table - I, II & III). Factors at stroke onset associated with depression of stroke patients at 3 months are shown in Table-IV. Of these Odds ratio and Chi-square for female sex, living alone, age more than 70 years, family history of stroke and those with a severe prognostic score on the SSS were significant. Other tested variables (diagnosis, site of hemiparesis, heart disease, smoking, diabetes, hypertension, increased body mass index and hyperlipidemia did not reach at significant level (Table - IV). The depression among the chief caregivers were not properly diagnosed and treated. Only 35% of them were undergoing antidepressant drug treatment at 12-months after onset (Table - V). Patients characteristics at 3-months associated with depression of the caregivers at the same point are shown in Table-VI. Depression was more common among the caregivers of patients at 3-months with BDI score 10-63(Odds Ratio 34.44; 95% Confidence Interval 0.7 to 8.74; Chi-square value with Yates correction 49.18 with P<.001) but that did not reach the significant level among the caregivers of the patients with BDI score 0-9 (Table-VII & VIII). Table-IX & X show the distribution of depression among the caregivers of the patients with BDI score 10-18 and 19-63. Both show that depression was significantly more common

among them (P <.001 each). Univariate analysis of the patients with SSS score <14 and the caregivers of them showed that the depression was significantly more common. among the caregivers (Odds Ratio 6.76; 95% Confidence Interval 1.23 to 4.98; Chi-square value 31.06 with P <.001) but that did not reach the significant level among the caregivers of the patients with SSS score >14 (Table-XI & XII).Statistical analysis of stroke patients with family history of stroke and their caregivers at 3-months showed that the depression was significantly more common among the caregivers (Odds Ratio 10.70; 95% Confidence Interval 1.70 to 7.94; Chi-square value 19.4 with P <001) (Table-XIII).

Table -I shows the distribution of depression among the caregivers and controls at 3months. Odds Ratio and Chi-square show depression is significantly more common among the caregivers.

Table-II shows the distribution of depression among the caregivers and controls at 12months. Odds Ratio and Chi-square show depression is significantly more common among the caregivers.

Table -III shows the frequency and severity of depression of caregivers. At 3-month 32.9% had mild depression and 8.9% had moderate to severe depression. Whereas at 12- months the frequency and severity of depression of caregivers are slightly higher than that of at 3-months.

Table-IV shows that female sex, SSS prognostic score at the onset of stroke <14 points, age >70 years, family history of stroke and living alone after stroke caries the statistical significance.

Table -V shows the number of caregivers getting antidepressant therapy at 12 months. Only 35% are getting antidepressant therapy.

Table - VI shows the distribution of depression among the caregivers of stroke survivors at 3-months. Odds Ratio and Chi-square show that depression is significantly higher among caregivers.

Table-VII shows the distribution of depression among caregivers of patients with BDI scores 10 to 63. Odds Ratio and Chi-square show depression is significantly more common among them.

Table - VIII shows the distribution of depression among caregivers of the patients with BDI score 0 to 9 at 3-months. Odds Ratio and Chi-square show that depression is not significantly common among them.

Table -IX shows the distribution of depression among the caregivers of the patients with BDI score 10 to 18 at 3-months. Odds Ratio and Chi-square test show that depression is significantly more common among them.

Table -X shows the distribution of depression among the caregivers of the patients with BDI score 19 to 63 at 3-months. Odds Ratio and Chi-square test show that depression is significantly common among them.

Table -XI shows the distribution of depression among the caregivers of the patients with SSS score <14 at 3-months. Odds Ratio and Chi-square test show that depression is signifcantly common among them.

Table -XII shows the distribution of depression among the caregivers of the patients with SSS score >14 at 3-months after stroke. Odds, Ratio and Chi-square test show that depression is not significantly common among them.

Table – XIII shows the distribution of depression among the caregivers of stroke patients with family history of stroke-3 months after the stroke. Odds Ration and Chi-square tests show the depression is significantly common among them.

Table - I

Depression among the caregivers at 3-months.

	Depression	No Depression	Odds Ratio OR	95% CI	X^2	P
Caregivers	68(42%)	93(58%)				
			3.47	1.48 to	23.72	<.001
Controls	28(17%)	133(83%)		4.14		

Table - II

Depression among the caregivers at 12-months.

	Depression	No Depression	Odds Ratio	95% CI	X	P
Caregivers.	65(41.5%)	91(58.5%)				
			3.26	1.47 to	20.96	<.001
Controls.	28(18%)	128(82%)		4.16		

Table -III

Frequency and severity of depression of caregivers.

	At 3 months		At 12 months	
Severity of depression (BDI Points)	Caregivers No	Controls No	Patients No	Controls No
0 -9	93 (58.2%)	133 (83.0%)	91 (58.5%)	128(82"/0)
10 - 18	54 (32.9%)	22 (13.28%)	48 (29.81 %)	20 (12.88%)
19 - 29	14 (8.9%)	5 (3.10%)	12 (8.45%)	6 (3.84%)
30 - 63	0	1 (0.62%)	5 (3.24%)	2 (1.28%)
Total	161 (100%)	161 (100%)	156 (100%)	156 (100%)

Table -IV

Univariate Odds Ratios, 95% Confidence intervals, Chi-square and P values of variables associated with patients' depression after 3 months of stroke.

Variables		Odds Ratios	95% C 1	X^2	P
1.	Sex, Females -	2.62	1.29 to 4.74	8.68	<.O1
2.	SSS prognostic score at the onset of stroke <14 points -	2.46	1.30 to 4.72	7.61	<.01
3.	Age >70 years -	2.54	1.12 to 5.47	8.51	<.01
4.	Family history of stroke -	4.94	1.15 to 5.30	18.48	<.001
5.	Side of hemiparesis...Left -	1.05	1.31 to 4.66	.02	>.50
6.	Living alone after stroke -	4.77	1.05 to 5.83	4.77	<.001
7.	Hypertension -	1.02	1.21 to 5.07	.0059	>.5
8.	Diabetes -	1.04	1.24 to 4.92	.029	>.5
9.	Hyperlipidemia -	0.94	1.15 to 5.32	.0177	>.5
10.	Heart disease -	0.89	1.23 to X4.98	.0917	>.5
11.	Smoking =	0.99	1.29 to 4.73	.000047	>.5
12.	Increased body mass indices -	1.00	1.14 to 5.35	.00052	>.5
13.	Diagnosis.....Infarct -	1.13	1.04 to 5.86	.081	>.5

Table -V

Caregivers getting antidepressant therapy at 12 months.

	No	%
Caregivers getting anti- depressant therapy.	10	35%
Caregivers getting no anti-depressant therapy.	18	65%

Table -VI

Univariate Odds Ratio and 95% confidence Interval of patients' variable associated with depression of caregivers 3 months after stroke onset.

	Odds Ratio	95% CI	X 2	P
BDI score of caregivers at 3-months with 10-63 points.	34.44	0.7 to 8.74	49.18	<.001
SSS scores of the caregivers at 3-months <14 points.	6.76	1.23 to 4.98	31.06	<.001

Table - VII

Univariate analysis of patients with BDI 10-63 points associated with depression of the caregivers 3-months after stroke.

	Depression	No Depression	Odds Ratio	95% CI	X^2	P
Caregivers of patients with BDI 10-63.	63	03	34.44	0.7 to 8.74	49.18 with Yates correction	<.001
Controls.	25	41				

Table - VIII

Univariate analysis of patients with BDI score 0 to 9 associated with depression of the caregivers 3-months after stroke.

	Depression	No depression	Odds Ratio	95% CI	X^2	P
Caregivers of the patients with BDI Score 0 to 9.	05	90	1.70	.57 to 10.67	.7093 with Yates correction	>.5
Controls.	03	92				

Table - IX

Univariate analysis of the patients with BDI score 10 to 18 associated with depression of the caregivers 3-months after stroke.

	Depression	No depression	Odds Ratio	95% CI	X^2	P
Caregivers of the patients with BDI score 10 to 18.	42	02	25.2 to	0.53 with 11.53 correction	27.27 <.001 Yates	
Controls.	20	24				

Table -X

Univariate analysis of patients with BDI score 19 to 63 associated with depression of the caregivers 3-month after the stroke.

	Depression	No depression	Odds Ration	95% CI	X^2	P
Caregivers of the patients with BDI score 19 to 63.	21	01	71.4	0.26 to	24.7 with Yates.	<.001
Controls	05	17	23.29		correction	

Table XI

Univariate analysis of the patients with SSS score <14 associated with depression of the caregivers 3-months after the stroke.

	Depression	No depression	Odds Ration	95% CI	X^2	P
Caregivers of Pts. With SSS <14.	55	24	6.76	1.23 to 4.98	31.06	<.001
Controls.	20	59				

Table -XII

Univariate analysis of patients with SSS score >14 associated with depression of the caregivers 3-months after the stroke

	Depression	No depression	Odds Ration	95% CI	X^2	P
Caregivers of Pts with SSS > 14.	13	69	1.76	0.96 to 6.34	1.51 with Yates corr.	>.5
Controls.	08	75				

Table-XIII

Univariate analysis of stroke patients with family history of stroke associated with depression of the caregivers 3-months after the stroke.

	Depression	No depression	Odds Ration	95% CI	X^2	P
Caregivers of Pts with F/H of stroke.	29	13	10.70	1.70 to 7.94	19.4 with	>.001
Controls.		05	24			

Discussion:

The population in this study is the largest unselected stroke population in which the occurrence of depression has symmetrically been examined. The first major result was the high rate of depression among the stroke caregivers and its persistence. About 42% of the stroke caregivers were depressed at 3-months after the stroke; although this number had not decreased at 12-months. Depression was diagnosed and treated in only a minority (35%).

In an earlier Finnish population based stroke register,[27] the frequency of depression among stroke caregivers at 3-months from stroke onset was almost the same as in the present study but decreased from 44% to 41..5% in 1 year. Both studies used the BDI to measure depression, but in the earlier study the cutoff point for depression was higher (>14 points). The reduction of depression at 1 year - which is contrary to our result; is probably the stroke care givers were, on average, younger and also had access to a more extensive systematic rehabilitation program than was possible in the present study. In our country the rehabilitation program is very poor. Our findings of 42% depression at 3months after the stroke onset and 41.5% at 12-months after the stroke onset are similar to the finding of a study in Finland in 1989[28]. In a community-based study by Wade et al, [29], 41% of stroke survivors were classified as depressed during the 1-year follow up which is similar to our study result.

The proportion of depressive caregivers did not differ from other studies. Carnwath and Johnson[9] studied the occurrence of depression among spouses of stroke patients 3 years after the event. Similar to our observations, they found that 39% of the spouses in the stroke group were depressed compared with 12% in the control group consisting of patients with disease other than stroke. They also reported that depression increased with time during the 3 years. In a recent Australian study[12] in which emotional distress was evaluated among the chief caregivers of 84 stroke patients who had survived I year with residual handicaps, half of the caregivers showed the evidence of emotional distress.

In the present study, poor BID score and severe SSS score at 3-months from the onset of stroke were associated with depression of the caregivers at 3-months by univariate analysis. Anderson et al[12] did not find a relation between the degree of physical disabilities and emotional stress among caregivers, a discrepancy that is hard to understand. In our study, the more severe depression was found in the caregivers at the same point.

Conclusion :

A large number of stroke caregivers suffer from depression, which could be prevented by good counseling. And at the same time early administration of antidepressant to those who has been suffering from depression might eliminate the subsequent severe

depression. Thereby we may achieve better adjustment of the patients and caregivers. Evans et al[30] have reported reduced depression in patients and caregivers after counseling of stroke caregivers.

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References :

1. Weinfeld DF. The National Survey of Stroke. Stroke 1981; 12 (suppl 1): 1-91.
2. Robinson RG, Bolduc PL, Price TR. Two-year longitudinal study of post-stroke mood disorders: diagnosis and outcome at one and two years. Stroke 1987; 18: 827- 34.
3. Parikh RJ, Lipsey JR, Robinson RG, Price TR. Two-year longitudinal study of post-stroke mood disorders: dynamic changes in correlates of depression at one and two years. Stroke 1987; 18:579 - 584.
4. Starkstein SE, Robinson RG. Affective disorders and cerebral vascular disease. Br J Psychiatry 1989; 154: 170 - 182.
5. Robinson RG, Starkstein SE. Current research in affective disorders following stroke. JNeuropsychiatry Clin Neurosci 1990; 2: 1- 14.
6. Robinson RG, Morris PL, Fedoroff JP. Depression and cerebrovascular disease. J Clin Psychiatry 1990; 51: 26 - 31.
7. Astrom M, Adolfsson R, Asplund K. Major depression in stroke patients: a 3-year longitudinal study. Stroke 1993; 24: 976 - 982.
8. Sharpe M, Hawton K, Seagroatt V, Bamford J, House A, Moltneux A, Sandercock P, Warlow C. Depressive disorders in long-term survivors stroke: association with demographic and social factors, functional status and brain lesion volume. Br J Psychiatry 1994; 164:380 - 386.
9. Carnwath TCM, Johnson DAW. Psychiatry morbidity among spouses of patients with stroke. Br J Med 1987; 294: 409 - 411.
10. Evans RL, Bishop DS, Haselkorn JK. Factors predicting satisfactory home care after stroke. Arch Phys Med Rehabil 1991; 72: 144 - 147.
11. Grevenson GC, Gray CS, French JM, James OFW. Long-term outcome for patients and carers following hospital admission for stroke. Age Aging 1991; 20: 337 - 344.
12. Anderson CS, Unto J, Stewart-Wynne EG. A population-based assessment of the impact and burden of caregiving for long-term stroke survivors. Stroke 1995; 26: 843 -849.
13. Rankin J. Cerebral vascular accidents in patients over age 60 and their prognosis. Scott Med J 1957; 2: 200 - 215.
14. Beck AT, Ward CH, Meldenson M, Mock J, Erbaugh J. An inventory for measuring depression. Arch Gen Psycliialry 1961; 4: 53-63.
15. Mahoney FL, Barthel DW. Functional evaluation: the Barthel Index Md Med J 1965; 14:61-65.
16. Albert M. A simple test of visual neglect. Neurology 1973; 23: 658 - 664.
17. Folstein MF, Folstein SE, McHugh PR. Mini-Mental State: a practical method for grading the cognitive state of patients for the clinician. JPsychiatr Res 1975; 12: 189 - 198.
18. Scandinavian Stroke Study Group. Multicenter trial of hemodilution in ischemic stroke: background and study protocol. Stroke 1985; 16: 885-90.
19. Cote R, Hachinski VC, Shurvell BL, Norris JW, Wolfson C. The Canadian Neurological Scale: a preliminary study in acute stroke. Stroke 1986; 17:731-737.
20. Wade DT, Hewer RL. Functional abilities after stroke: measurement, natural history and prognosis. J Neurol Neurosurg Psychiatry 1987; 50: 177 - 182.
21. Sintonen H, Pekurinen M. A fifteen-dimensional measure of health-related quality of life (15 D) and its applications. In: Walker SR, Rosser RM. Eds. Quality oflife Assesment: Key Issues in the 1990s. Dordrecht Netherlands: Kluver Academic Publishers: 1993; 185-195.
22. Abacus Concepts. Statview. Berkeley, Calif 1987.
23. Dixon WJ, Brown MB, Engelman L,Jennrich RI, eds. BMDP Statistical Softwear. Los Angeles, Calif. University of California Press; 1990.
24. House A, Dennis M, Hawton K, Warlow C. Methods of identifying mood disorders in stroke patients: experience in the Oxfordshire Community Stroke Project. Age Ageing 1989; 18: 371-379.

25. Kendall PC, Hollon SD, Beck AT, Hammen CL, Ingram RE. Issues and recommendations regarding use of the Beck Depression Inventory. Cognitive Ther Res 1987; 11: 289-299.

26. Beck AT, Steer RA, Garbin MG. Psychometric properties of the Beck Depression Inventory: twenty-five years of evaluation. Clinl Psychol Rev 1988; 8: 77-100.

27. House A, Dennis M, Warlow C, Howton K, Molyneux A. Mood disorders after stroke and their relation to lesion location. Brain 1990; 113: 1113-1129.

28. Mervi Kotila,.Heikki Numminen, Olli Waltimo, Markku Kaste. Depression after stroke -results of the FINNS STROKE study. Stroke 1998; 29: 368-372.

29. Wade DT, Leigh-Smith J, Hewer RA. Depressed mood after stroke: a community study of its frequency. Br J Psychiatry 1987; 151: 200-205.

30. Evans RL, Matlock Al, Bishop DS, Stranahan S, Pederson C. Family intervention after stroke: Does counseling or educaiton help? Stroke 1988; 19: 1243-1249.

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Placenta Previa - its Association with History of Previous Caesarean Section and Abortion

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Summary :

The purpose of this study is to determine the incidence of placenta previa and to quantify the risk of placenta previa based on the presence and number of caesarean deliveries and a history of spontaneous or induced abortion. The study was done in Sir Salimullah Medical College and Mitford Hospital from January 2000 to December 2000. Total 126 cases of placenta previa were admitted and studied during this period. Here the incidence was found 2.25%. Among 126 cases, 68 (54%) had previous history of either abortion (spontaneous/induced) or caesarean section or both. Only 3(2.38%) had history of both abortion and caesarean section and 58(46.03%) had no such history. Among 68 cases 48(38.09%) had previous history of one or more abortion and 20(15.87%) had history of previous caesarean delivery. Here advanced maternal age and high parity was also found to be associated with increased frequency of placenta, previa. A more significant trend was found with increase number of previous caesarean delivery and abortion.

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Introduction :

In placenta previa, the placenta is implanted in the lower uterine segment within the zone of effacement and dilatation of the cervix thus constituting an obstruction to descent of the presenting part. Placenta previa is encountered in approximately one in 200 birth, but only 20% are total (placenta over the entire cervix)[1]. Although placenta previa is relatively uncommon, it is regarded as one of the leading cause of uterine bleeding during the latter stage of gestation and has been recognised as an important determinant of maternal morbidity and adverse perinatal outcome[2]. Risk factors associated with placenta previa include advanced maternal age, multiparity, cigarette smoking and cocaine use, history of placenta previa, caesarean delivery, spontaneous and induced abortion and prior gynaecological surgeries. Nonetheless, the etiology of placenta previa largely remain obscure and speculative. In spite of the advent of ultrasonography to diagnose this disorder and the ability to assess fetal lung maturity to appropriately time delivery, efforts to improve perinatal out come incase of placenta previa continue to pose a challenge. It appears that the rate of caesarean delivery has been increasing steadily over the past two decades. Some studies have observed an increased frequency of placenta previa among women with a prior caesarean delivery or abortions, suggesting an association with surgical procedures that disrupt the uterine cavity. Nonetheless, the extent to which a history of prior caesarean delivery or spontaneous and induced abortion predisposes women to the development of placenta previa is unclear[3].

Materials and Method :

This was a one year prospective study carried out in Sir Salimullah medical college and Mitford hospital during the period of January 2000 to December 2000. During this study period total 5579 obstetric patients were admitted. Among them 126 cases of placenta previa were admitted with or without clinical symptoms. Data of those pregnant women and deliveries were collected by a preformed questionnaire and direct examination of patients and different parameters were

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recorded. The study group includes patient having typical symptoms of placenta previaed, diagnosed by detail, history. clinical examination, during caesarean section by ultrasonography (diagnostic incidental). Other factors also evaluated such as age, gravidity, socioeconomic condition, previous obstetric and surgical history e.g. MR, D & C and the nature of P/V bleeding and its relation with activity, number of bleeding episodes, association with pain or not and detailed history regarding any antenatal care. All patient were clinically examined for anaemia, pulse, BP, and oedema. Per abdominal examination also were done to exclude accidental haemorrhage. Necessary investigation also was done e.g. Hb%, blood grouping, Rh. typing & crossmatching of blood and USG. Active management was done in 108 cases irrespective of gestational age by caesarean section due to persistent bleeding or when pregnancy had reached 38 weeks of gestation with moderate or no bleeding. Conservative management were possible for remaining 18 patients. Postnatally they were followed up for postpartum hemorrhage (PPH), infection, rate of sub-involution of uterus. There were some limitations in this study e.g. many of the patient did not know their exact date of last menstrual period (LMP), most of the patient had no antenatal checkup, many information were recorded from the attendants. As because it is a referral hospital most of the patient were admitted in odd time, so time and scope for investigation, especially USG was very limited.

Result :

During this one year study period total admitted obstetric patient was 5579 and total delivery occurred in 5078 patient and remaining patient were discharged undelivered with conservative management. Among 5579 patient, total 126 patient were diagnosed as placenta previa considering the various parameters with different gestational age ranging from 30 week to term. So, the incidence of placenta previa was found 2.25% shown in table- I

Among 126 cases of Placenta Previa it was found that 68 (54%) had previous history of either abortion (Spontaneous or induced or both), caesarian deliveries or both and 58 (46.03%) had no such history. From these 68 patient only abortion was found in 48(38.09%) cases of which 33 (26.19%) had only spontaneous abortion and induce abortion including MR was found in 15(11.90%) cases. Among spontaneous abortion 22(17.72%) had one, 10(7.94%) cases had 2 (two) and 1 (.79%) had 3 (three) spontaneous abortion and all are treated by dilatation and curettege (D & C). In the group of induced abortion including MR, 10(7.94%) had one and 5 (3.97) had 2 (two) induced abortion followed by D & C. Shown in table-II.

Total 678 cases of pregnancy with HO abortion were admitted during this study period and among them 48 patient had placenta previa. So placenta previa complicated 7.07% of cases with history of previous abortion shown in table -III.

Placenta previa with previous caesarean section was found in 20 (15.87%) cases of which 12(9.52%) had one and 8 (6.35%) had 2 (two) prior caesarean section. During this study period, total patient with history of previous Caesarean Section was 599, of which 20 patient had placenta previa. So, placenta previa complicated 3.33% of cases with history of previous Caesarean Section. Shown in table IV & V.

In this study we found that total 1277 patient had either history of caesarean section, abortion or both and among them placenta previa was found in 68 cases. So placenta previa complicated 5.32% of cases with scarred uterus, shown in table V I.

Remaining 3801 cases from total delivery had no prior history of Caesarean Section or abortion and placenta previa was found in only

58 cases. So, placenta previa complicated 1.52% of cases without prior history of Caesarean Section or abortion shown in Table- VII.

The age ranged from < 20 years to > 35 years and peak incidence was 30-35 years found in 40 (31.74%) cases, 30(23.80%) were 20-30 years, 30 (23.80%) were >35 years and 26 (20.63%) were < 20 years of age group, shown in Table-VIII.

Regarding gravity >G_3 was found 43 (34.12%) cases, G_3 was 39 (30.95%) cases G_2 was 23 (18.25%) cases and 21 (16.66%) cases was found in primigravida shown in Table- IX

Table-I

Incidence of Placenta Previa

Total admitted patient	Total Placenta Previa	Percentage
5579	126	2.25%

Table-II

Relation of placenta Previa with abortion (Spontaneous and induced)

Total placent previa case	Placenta previa with History of abortion	Percentage	Spontaneous abortion	Percentage	Induced abortion	Percentage
126	48	(38.09%)	33	(26.19%)	15	(11.90%)

Table-III

Total admitted Pregnancy with H/0 abortion cases	Total admitted placenta previa	Placenta previa with H/O abortion	Risk
678	126	48	7.07%

Table-IV

Relation of placenta previa with previous caesarean section

Total placenta previa admitted	Placenta previa with H/O prior caesarean section	H/O Previous 1(one) caesarean section	H/O previous 2 (two) caesarean section
126	20 (15.87%)	12 (9.52%)	08 (6.35%)

Table-V

Total admited Patient with H/O previous c/s	Total placenta previa	Placenta previa with H/O prior c/s	Risk
599	126	20	3.33%

Table V I

Relation of placenta previa with scarred uterus.

Total patient with H/O- c/s, abortion or both	Total placenta previa	Placenta previa H/O-. c/s, abortion or both	Risk
1277	126	68	5.32%

Table-VII

Total patient without H/O of C/S or abortion	Total placenta previa admitted abortion	Placenta previa without prior C/S or	Risk
3801	126	58	1.52%

Table-VIII

Age group (year)	Number of patient	Percentage
<20 years	26	20.63%
20-30 years	3,0	23.80%
30-35 years	40	31.74%
>35 years	30	23.80%

Table-IX

Relation of placenta previa with gravidity.

Gravida	No. of case	Percentage
Primi gravida	21	16.66%
Second gravida (G_2)	23	18.25%
Third gravida (G_3)	39	30.95%
Fourth or more (>G_3)	43	34.12%

From present study it is concluded that placenta previa complicated 5.32% of cases with scarred uterus compared with 1.52% of cases with non scarred uterus a 3.5 fold increase risk of development of placenta previa in subsequent pregnancy with scarred uterus.

Discussion :

Placenta previa is one of the important obstetric hazard contributing significantly to maternal death and perinatal loss in developing countries. Although the management of a woman with placenta previa has become standarized, with expectant management favoured untill fetal lung maturity is established, followed by aggressively moving to delivery[4]. It has been reported to occur approximately 0.28% to 1.96% of pregnancies shown in different studies[3,4,5]. In present study the incidence of placenta previa is much more higher 2.25% which is very similar to other studies [6,7] e.g. one in every 44 cases. This increase incidence may be the result of increase caesarean section, MR, abortion rates and also may be due to various methods of

diagnosis e.g, wide spread use of USG for detection of placenta previa, definition used, and diverse nature of population studied. Almost four decades ago Bender[8] first observed an increased frequency of placenta previa among women with uterine scarring (because of caesarean delivery and abortion) in prior pregnancy. An association between placenta previa and prior caesarean delivery is biologically plausible. Damage to the endometrial, and myometrial uterine lining can predispose to a low implantation of the placenta in the uterus. Like wise, curettage of the uterus during spontaneous or induced abortion may significantly damage the endometrium and uterine cavity so as to increase risk for placenta previa[3]. Clark et al proposed that the higher incidence of clinically observed placenta previa at term is caused by the failure of differential growth of a scarred lower segment, so that the originaly low lying placenta would be less likely to migrate upwards[9]. In present study 15.87% of patient having placenta previa had prior caesaren delivery which is similar to other studies[5,9]. So, this study shows placenta previa complicate 3.33% of cases with history of previous caesarean delivery. On the other hand, we found 38.09% cases of placenta previa had previous history of abortion (Spontaneous or induced), e.g. placenta previa complicated 7.07% of cases with previous history of abortion which is similar to study done by Barrett et all[10]. From present study we found that placenta previa complicated 5.32% of cases with scarred uterus and 1.52% of cases with non scarred uterus. So a 3.5 fold increase risk of development of placenta previa in subsequent pregnancy with scarred uterus than non scarred uterus. This result is much more close to other studies[11,12,13].

Regarding age and parity the risk of placenta previa increases both because of ageing effect of the uterus and repeated pregnancies[14].Physiologically, as age of women increases collagen progressively replace normal muscle in the walls of myometrial arteries[15]. Naeye et al found that the percentage of intra myometrial arteries with sclerotic lesion increased from 11% at age 17 to 19 years to 37 % at 20 to 29 years and 61% at 30 to 39 years and 83 % after 39 years. These vascular changes with age were not altered by adjustment for parity. These atrophic change in older women may also result defective vascularization of the decidua and consequently restrict the blood flow to the placenta. Both underperfusion and under vascularization have been postulated to play important role in the development of placenta previa[16]. In each pregnancy whether ending with early pregnancy termination or term birth, may damage the endometrium underlying the implantation site. These region may be no longer suitable for implantation. which predisposes subsequent implantation towards the lower uterine segment[17]. Goplerud et al also postulated that vessel at the previous placental sites undergo changes that may decrease the blood supply to those areas of the endometrium. In subsequent pregnancy, more surface area may be required, for placental attachment to provide adequate maternal blood flow to the intervillous space, increasing the risk of placenta previa[18]. In present study shows about 31.74% cases of placenta previa had age group was 30-35 years and 34.12% was found in grand multipara which is similar to studies[6,7,19]. From present study, it is clearly demonstrates that there is a strong association between having a previous spontaneous or induced abortion, caesarean delivery and subsequent development of placenta previa. Moreover, this risk increase dramatically with increasing number of prior caesarean delivery or abortion. These finding are consistent with the hypothesis that endometrial / myometrial damage is a significant aetiological factors in low placental implantation.

Conclusion :

This study suggest that a reduction in uterine instrumentation rate for the management

of both spontaneous and induced abortion could further reduce the risk of placenta previa and also provide yet another reason for reducing the primary caesarean delivery and for advocating vaginal birth for women with prior caesarean delivery. Pregnant women with a history of caesarean delivery or abortion must be regarded as being at increase risk for the subsequent development of placenta previa and must be monitored carefully.

References :

1. Marlin L. Pernoll, Md. third trimester haemorrhage, current obstetrics and G ynaecologic dignosis and treatment Eight edition, P- 404-5.
2. Cunningham GF, Mac Donald PC, Gant NF, Levens KJ, Gilstrap LC, editors. williams obstetrics. 19th ed. Norwalk (CT): Appleton & Lange; 1993, P. 819-51.
3. Cande V. Ananth, Ph D, MPH a, C John C. Smulian MD, MP Hat and Anthony M. Vintzileos, MDh "The Association of placenta previa with history of caesarean delivery and abortion: A metanalysis. Am J. obstet Gynecol 1997, 177, 5: 1071-8.
4. Silver R, Deep R, sabbagha RE, Dooley SL, Socol ML, Tamura RK, Placenta Previa aggressive expectant management. Am J. obstet Gyncol, 1984; 150:15-22.
5. Hershk owitz R; Fraser-D; Manzor M; Leiberman JR, "one or multiple previous caesarean section are associated with similar increased frequency of placenta previa, Eur, J Obstet Gynecol Reprod-Biol. 1995 Oct; 62(2): 185-8.
6. Mahmuda B. Relationship between the clinical presentation and maternal & fetal outcome in placenta previa at DMCH: Personal study of 100 cases, Dissertation, BOPS. Dhaka, 1999.
7. Ashrat F. Study of pregnancy outcome in placenta previa: Personal study of 50 cases. Dissertation, Dhaka, BCPS 1996.
8. Bender S. Placenta previa and lower segment caesarean section. Surg. Gynecol obstet 1954: 98:625-7.
9. Clark -SL; koonings -PP; phelan JP "Placenta previa/ acreta and prior caesarean section. obstet- Gynecol 1985; 66(1): 89-92.
10. Barrett JM; Boehm-FH; killam- AP. induced abortion; a risk factor for placenta previa. Am-.J-obstet- Gynecol. 1981 Dec 1; 141 (7): 769-7.2.
11. Chattapadhyay SK, Kharif H, Sherbeeni MM. Placenta previa and acreta after caesarean section. Eur-J-obstet - Gynecol-Reprod-biol. 1993; 30; 52 (3): 151-6.
12. Niclsen-TF; Hag berg-H; Ljung blad -U "placenta previa and antepartum haemorrhage after previous caesarean section- Gynecol Obstet. Invest. 1989; 27 (2): 8890.
13. Taylor VM, kramer MD, Vaugham TL, Peacock S. Placenta previa and prior caesarean delivery: How strong is the association? obstet-Gynecol. 1994; 84 (1) : 55-7.
14. Meyer MB, Jones BS, Tonacia JA. Perinatal events associated with maternal smoking during pregnancy. Am J. E pidemiol 1976; 130: 464-76.
15. Naeye RL. Maternal age, obstetric complication and the out come of pregnancy. obstet gynecol 1983; 61: 210-6.
16. Naeye RL. The duration of maternal Cigarette smoking fetal and placental disorder. Early Hum Dev. 1979; 3: 229-37.
17. Green JR. Placente previa and abruptio plcenta. In creasy RK, Resnik R, eds. maternal -fetal medicine; principles and practice. Philadelphia WB Saun ders, 1989: 592600.
18. Goplerud CP. Bleeding in late pregnancy. In Danforth DN, Scott JR, eds. obstetrics and Gyneacology. philadelphia: JB lippencott, 1986: 433-9.
19. Parazzini F, Dindelli M, Luchini L, La Rosa M; Potenza MT; Frigerio L, Ferrari A, Risk faclors for placenta previa placenta 1994 Aft 15 (3) 321=6. (16) Naeye RL. The duration of maternal Cigarette smoking fetal and placental disorder. Early Hum Dev. 1979; 3: 229-37.
20. Green JR. Placente previa and abruptio plcenta. In creasy RK, Resnik R, eds. maternal -fetal medicine; principles and practice. Philadelphia WB Saun ders, 1989: 592600.
21. Goplerud CP. Bleeding in late pregnancy. In Danforth DN, Scott JR, eds. obstetrics and Gyneacology. philadelphia: JB lippencott, 1986: 433-9.
22. Parazzini F, Dindelli M, Luchini L, La-Rosa M, Potenza MT, Frigerio L, Ferrari A, Risk faclors for placenta previa placenta 1994 Aft 15 (3) 321-6.

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SPECIAL ARTICLE

Competence Based Education and Assessment

TAHMINA BEGUM FCPS

Introduction:

The aim of medical education is to produce doctors with a wide range of skills as well as with a fund of relevant knowledge and appropriate attitudes to meet the needs and demands of the society. We live in a changing society. Today's competencies are not tomorrow's. In medical education, new knowledge is constantly being discovered and old knowledge is being proved wrong. A competent professional will be able not simply to cope with changes but actively to shape changes. Society has become increasingly concerned with the sibling issues of competence and accountability in the profession. The assessment system needs significant reformation and all the competencies required by the doctor need to be assessed both for the purpose of documenting achievement and to convey a message about the qualities that are valued by the profession.

The aim of this review is to focus on few important aspects of competency based education, training and assessment of competencies in medical practice.

Conception of competence and performance: A competence- based approach to education, training and assessment is recognized as a key educational policy[1]. Competence is conceptualized in terms of possession of a series of desirable attributes including knowledge, skills and abilities such as problem solving, analysis, communication, pattern recognition and attitudes[2]. Obviously it incorporates -the ideas of professional judgement[1]. Clinical competence needed by the medical professionals, as used in this paper, refers to a complex set of skills that include the abilities to interview, perform a physical examination, make diagnostic and treatment decision and communicate with patients and their family while demonstrating good interpersonal skills[3].

Competence and performance can be defined in a several ways. Senior and Loyd distinguished between the meaning of competence and the performance of a physician (cited by Rethans et al)[4]. Competence is the ability to carry out a task and performance is actually doing what is required. In other words, competence can be defined in terms of what the students or doctors should be able to do at an expected level of achievement and performance can be defined as what a student or doctor actually does in real clinical practice[5].

There can be little doubt that competence and performance involves more than the possession of knowledge: they demand the exercise of technical skills, and are influenced by the nature of attitudes and values that have been acquired in the course of training. To say of an individual that he is competent is to assert that his actions are coming up to a standard. The higher the level of competence (level of competence- in the terminology of' National Council for Vocational Qualification- NCVQ) the more demanding the standard being expected.

Importance of defining competence: Competence in medicine is one of the important priorities in medical practice[6]. A doctor is a doctor but his professional activities are almost certainly contained within a discipline of medicine[7]. A doctor

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today is expected to be competent in everything that is involved in the practice of medicine. So it is important to decide the competencies which the professionals must have in order to do their job. At the same time, the school must develop curricular strategies for ensuring a defined level of practical competence amongst new graduates. Such a definition will definitely help prevent a mismatch between the programme goals of medical education and the rapidly changing demands of medical practice, of the community and the nation as a whole.

Competence based education: It is a form of education that derives a curriculum from an analysis of a prospective or actual professional role and that attempts to certify student's progress on the basis of demonstrated performance in some aspect of that role[8]. Competence based education and training is used as models in higher education[9]. These models include the precise specification of competencies or behaviors to be learned. The National Consortium of Competence Based Education Centre, USA established a set of criteria for describing and assessing competence based programme[9].

Competence specification:

The aim of the competence based study in medical science should be to provide the basis for a programme of courses, which will be relevant to the practice of medicine, and which, also, will make continuing education more appealing for the practicing doctors by concentrating on their individual needs[6]. A competence-based approach offers all professions, and thus all medical specialties, the chance to build their continuing education programme. CBE strives to predict that the student will succeed in specific roles after their educational experience but there are some difficulties in developing competence- based tools both in teaching and particularly in assessment.

Competence based assessment:

Competence based assessment (CBA) is the most important task facing medical teachers[5]. As clinical competence is regarded as the mastery of the body of relevant knowledge and the acquisition of a range of relevant skills, attitudes and problem solving abilities[4], the assessment of competence therefore requires several measurement instruments, each representing different aspect of competencies[6]. Under the competence based assessment system, assessor make judgements based on evidence gathered from performance about whether an individual meets criteria specified in a profession's competence[1]. It has been pointed out by Wolf[10] and Gonczi[1] that competence cannot be observed directly. It can only be inferred from holistic performance. This means assessment of competence will inevitably based on inference from a sample of performance[11]. Thus we need to think about the sort of performance, which will enable us to gather evidence of sufficient quality and quantity to make sound judgements about an individual's competence.

Traditionally, medical students have always faced clinical examination, usually one long case and several short cases. But this kind of assessment has been attacked for being too narrow and for being insufficiently comprehensive to test all the competencies needed by the doctors[6].

Competence can not be validly assessed solely by written examination[12]. Knowledge can be determined by conventional testing procedures but the ability to use that knowledge requires altogether different assessment methods[13]. In addition, the classic examinations with theoretical questions or multiple-choice questions do not assess clinical skills as we[14].

Key difference between traditional and CBA system is given below:

	Traditional	Competence based
Concept	Assessment of learning ability or achievement	Assessment of actual performance in a work role
Foundation	Curricula, defined centrally by teaching staff	Explicit standards of required performance defined by research
Assessment Requirements	Assessment is an integral part of learning programme	Assessment is independent of any learning programme
Evidence	1. Assessment evidence drawn from course assignment	Evidence collected from actual performance
	2. Assessment is norm-referenced	Assessment is criterion referenced

If the question arise " Is there any alternative to the traditional clinical examination?" the answer is probably "yes". Certainly, a number of approaches to the assessment of clinical competence have been developed which are worthy of consideration. In selecting a method few points should be taken into account.

- Methods should be selected that are most direct and relevant to what is being assessed[15].
- The choice of assessment method should be based on research on the reliability and validity of the available methods[16].
- An assessment method should be used that is most capable of assessing competence in an integrated manner. This approach seeks to combine knowledge, understanding, problem solving, technical skills, attitudes and ethics in assessment.

The Objective Structured Clinical Examination (OSCE) fulfils the most of the criteria of an ideal assessment system[17]. It is a method of assessing a student's clinical competence which is objective rather than subjective, and in which the areas tested are carefully planned by the examiners[18]. It provides a more valid examination than the traditional approach. The overwhelming advantage of the OSCE is that it enables the examiners to test the student's competence and performance in a wide range of contexts and situations and it is entirely practice oriented[6].

Case studies are example of an integrated assessment method e.g. simulated interview (an interview between doctor and patient) can assess number different types of competence at the same time[1]. Using checklist or logbook can assess skills that have to be certified as having been mastered. Stillman et al[19] considered standardized patients (SP) are more useful in assessing clinical skills. Performance based assessment of clinical skills using SPs and /or teaching associates provide a useful modality to evaluate clinical skills not adequately measured by cognitive examinations and not frequently analyzed by direct observation of trainees[20]. For performance based assessment, in addition to SP, three methods can also be used. These are:- written clinical simulation, more commonly termed as Patient Management Problems (PMPs), computer based clinical examinations and oral examinations[21].

Communication skills are also an essential component of competence. These skills can he included, as part of the OSCE and the assessment is more valid and reliable[17]. A more recent development in assessment of communication skills is the objective

structured video examination which examine the ability to recognize types of communication being utilized in a particular situation[17]. One of the principal recommendations of Tomorrow's Doctors is to include attitude and behavior in the competence. It is possible to assess attitudes by written examination, OSCEs and simulation[17].

Conclusion:

A competence based approach to assessment of professionals is potentially more valid than a traditional approach. It enables us to come closer than we have in the past in assessing what we want to assess- the capacity of the professional to integrate knowledge, values, attitudes and skills in the world of practice.

References :

1. Gonczi A. Competence based assessment in the profession in Australia. Assessment in Education 1994; 1(1): 27-44.
2. Hager P, Gonczi A. What is competence? Medical Teacher 1996; 18(1): 15-18.
3. Viet N U, Barrows H S, Marcy M L. Six years of comprehensive, clinical performance based assessment using SP at the southern Illinois University School of Medicine. Academic Medicine. 1992; 67(1): 64-69.
4. Rethans J J, Stermans F, Drop R, Vlenten C V R, Holens P. Does-competence of general practitioners predict their performance? Comparison between examination setting and actual practice. BMJ 1991; 303: 1377-81.
5. Newble D 1. Assessing clinical competence at undergraduate level. Medical Education Booklet, no 25, 1992.
6. Dunn W R, Hamilton D D. Techniques of identifying competencies needed by doctors. Medical Teacher 1985; 7(1): 15-25.
7. General Medical Council (GMC), Tomorrows Doctors: Recommendations on undergraduate medical education. 1993; London.
8. Lane D S, Ross V. The importance of defining physicians' competencies: Lessons from preventive medicine. Academic Medicine 1994; 69(12): 973-75.
9. Fletcher S. Competence based assessment technique, 2'a ed. Kogan page limited, London.
10. Wolf A. Perceived mistreatment and attitude change by graduating medical students: A retrospective study. Medical Education 1989; 25: 182-90.
11. Neufeld V R, Norman G R. Assessing clinical competence. Journal of Medical Education. 1989; 65: 902-7.
12. Mehrans W A. Using performance assessment for accountability purpose. Educational measurement issues and practice. 1992; 1(1): 3-9.
13. Editorial, Assessment in Education. 1998; 5(1); p-1.
14. Nuutinen M, Vainionpan L, Kokkomen J, Rantala H, Tapanainen P, Mottonen M et al. Practice oriented evaluation of medical students during a paediatric course. Medical Teacher 1997; 19(3): 12-15.
15. Baily M. Theoretical models and the assessment of competence. National Assessment Research Forum 1993; Sydney, TAFE Commission.
16. Maguire P. Assessing clinical competence. BMJ 1999; 398: 4-5.
17. Fowell S L, Bligh J G. Recent developments in assessing medical students. Post graduate Med J 1998; 74; 1 R-24.
18. Harden R M, Gleeson F A. Assessment of medical competence using a Objective Structured Clinical Examination. ASME Medical Education booklet, no 8, 1979.
19. Stillman P L, Swanson D B. Ensuring the clinical competence of medical graduate through standardized patient. Journal of Medical Education 1997; 147: 1049-52.
20. Hawkins R, Gross R, Beutel S. Use of SP for teaching and evaluating the skills of Internal Medicine Residents. Teaching and Learning in Medicine 1998; 10(2): 65-68.
21. Swanson D B, Norman G R, Linn R L. Performance based assessment, lessons from the health professional. Educational Research 1995; 19(1): 5-13.

CASE REPORTS

Twin Pregnancy with Hydatidiform Mole - A Case Report

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Summary :

Hydatidiform mole coexisting with a normal viable foetus in a multiple pregnancy is a rare phenomenon. Our patient had twin pregnancy with a viable foetus and normal placenta along with a Hydatidiform mole. The case was first diagnosed by ultrasonogram at 12 weeks. The pregnancy continued upto 26 weeks when she delivered prematurely. The foetus died immediately after birth. The serum β-HCG which was 252 5ml/Ulml came down to 02 mIU/ml at 8 weeks following delivery.

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Case Report :

A 22 yr. old young primigravid patient presented with 12 weeks amenorrhoea and PV bleeding. She was having hyperemesis. On examination the uterus was about 18 weeks in size.

USG of the uterus revealed twin pregnancy with one viable foetus of 12 weeks with a normal placenta near the fundus and a second sac containing numerous hypoechogenic vescicular shadows resembling the typical snow storm appearance of molar pregnancy.

The patient had conceived after treatment for primary infertility following laparoscopy and Dye test along with D & C. She conceived with Clomiphene Citrate and Injection pregnyl (HCG). This was a very much expected pregnancy, so the patient was explained about the fate. She was treated conservatively till 26 weeks of gestation. Throughout this period she remained in absolute bed rest with regular follow up by clinical examination and USG evaluation. The uterine height reached 30 weeks size at 26 weeks gestation while USG showed a very active baby near the fundus and molar tissue occupying the lower part of the uterus during this period she expelled one or two vescicles irregularly along with mild P/V bleeding. All the time she was having hyperemesis. At 26 weeks gestation with a uterine height of 30 weeks, she expelled the uterine contents the vescicles and the foetus spontaneously. The foetus died immediately after birth. The retained products were evacuated and sent for histopathology, which confirmed Complete

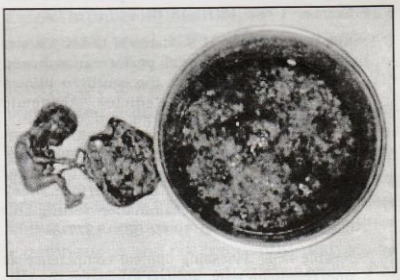

***Fig.-1** : Photograph showing foetus along with its cord and placenta. The molar tissue is shown in the bowel*

Hydatidiform mole. One week after evacuation the serum βHCG was 2525 mIU/ml. Following evacuation the patient continued to bleed irregularly. Two weeks later the βHCG came down to 250 mIU/ml. The patient had undergone one more D & C at the 10th day for P. V bleeding. Histopathology showed hydatidiform mole. The uterine involution was slow and took 8 weeks to revert

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back to normal size. The serum βHCG level become 02 mIU/ml at 8 weeks after delivery.

The patient is under regular follow up for the last 12 Months. She is asymptomatic with normal findings at follow up clinically, biochemically and with ultrasonogram evaluation.

Discussion :

Hydatidiform mole is the benign form of the trophoblastic disease spectrum. It is not an uncommon disease, but coexistence of a normal foetus with a molar pregnancy is a rarity. In our case the 22 yr. old mother had undergone treatment for infertility and had conceived 5 yrs. after marriage by ovulation induction with clomephene citrate and injection pregnyl. She was very anxious for this pregnancy and had reported with hyperemesis and threatened abortion at 12 weeks. On admission the fundal height was 18 weeks. By ultrasonogram a 12 weeks viable foetus with coexisting hydatidifoim mole was diagnosed. There are a number of case reports of complete Hydatidiform mole and multiple gestations. There are two case reports from Japan[1]. One is a twin pregnancy with a complete hydatidiform mole and the other one is a triplet pregnancy with two viable foetuses and one molar pregnancy. First case occurred after gamete intra fallopian transfer (GIFT) and the second one after In vitro fertilization and embryo transfer (IVF ET)[1]. There is another case report from Belgium of a complete Hydatidiform mole coexisting with a normal foetus and placenta[2].

The role of ultrasonogram in these cases is immense. Ultrasonogram plays a vital role in diagnosis and follow up for these patients. The typical snow storm echogenicity is diagnostic for hydatidiform mole and at the same time the foetal well being can also be assessed.

The dilema in our patient was the management of the case. It was a very much expected pregnancy for the patient. The case was discussed with the patient and her husband. They opted to continue the pregnancy. There is a case report of term twin pregnancy with a complete Hydatidiform mole and a normal live fetus in India[3]. There is an other report from Taiwan where a live anaemic female baby was delivered at 32 weeks gestation by a 30 year old lady. It was first diagnosed at 18 weeks by USG and at 32 weeks she delivered prematurely[4].

Our patient continued her pregnancy till 26 weeks of gestation. She was very sick with hyperemesis & mild pre eclampsia with a uterine height of 30 weeks. She expelled the uterine contents spontaneously. The baby died immediately after birth. Presence of a gestational sac with a foetus and a coexisting hydatidiform mole separately is a very rare finding. But in our case during follow up by ultrasonography the viable and active foetus was seen within a gestational sac and the vescicles of the hydatidiform mole separately. After delivery the very premature baby of 26 weeks gestational age and having birth weight of 600 gm died immediately. The baby had a separate placenta and membrane. The moler tissue were separate from the normal foetus, placenta and its membranes. There is a case report from USA where in a quadruplet pregnancy with 3 normal fetuses & one partial mole the patients had severe hyperemesis & preeclampsia at 16 weeks, and with the help of antibiotic, tocolysis and bed rest, the pregnancy continued upto 32 weeks[5]. The hyperemesis and the features of preeclampsia in our patient subsided after delivery. The serum βHCG which was 2525 mIU/ml after delivery reverted back to 02 mIU/ml after 8 weeks.

Conclusion :

In multiple pregnancy one of the conceptus may become a hydatidiform mole. Ultrasonogram plays an important role in these cases for monitoring of the foetal wellbeing and the progress of the disease

process. With symptomatic treatment and close supervision, the pregnancy may be continued even upto an optimum maturity till the patient goes into spontaneous labour. It is not essential to terminate the pregnancy as soon as it is diagnosed.

References :

1. Show-M; Akimoto-k; Kasai-T; Move-M; Michinura-Y; Hydatidiform Mole associated with multiple gestations after assisted reproduction; diagnosis by analysis of DNA finger print. Mol- 1998 rep; 4(9): 877 - 80.
2. Van deginste - S; Vergoto - 1B; Hanssens - M; Moeiman - P; page - G; Van - Den - Berghe -K; Van-Assche - A. Malignant trophoblastic disease following a twin pregnancy consisting of a complete hydalidiform mole and a normal fetus and placenta. A case report. Eur - Gynecol - Oncol. 1999; 20 (2):105 - 7.
3. Abbi - M; Kriplani - A; Uppal - R; Takkar - D; Term twin pregnancy with Hydalidifonn mole and a normal fetus. Arch -Gynecol - Obat. 1992; 262(3-4): 189-91.
4. Hsieh -CC; Hsieh - TT; Shueh -C; Kuo - DM; Lo - LM; Hung - TH; Delivery of a severely anaemil fetus after partial molar pregnancy. Clinical and ultrasonographic findings. Hum - Reprod. 1999 Apr, 14(4): 1122 - 6.
5. Marinoff - DN; Spitzbergh - EH; Chuch - JT; Goldman - JM; Downs -T. Delayed - interval delivery in quadruplet pregnancy after intrauterine death of partial molar pregnancy and preterm delivery. A case report. J-Reprod - Med. 1998 Dec; 43(12): 1051 - 4.

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Torsion of the Gravid Uterus

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Summary :

A multigravida presented with sudden severe pain in the abdomen and shock at 28 weeks of gestation. After laparotomy she was diagnosed to be a case of torsion of the gravid uterus -which was unicornuate. Its diagonostic difficulties management and treatment outcome are discussed.

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Introduction :

Torsion of an organ is described as a twist of the organ in its longitudinal axis around a narrow pedicle. Torsion of the gravid uterus is a very uncommon cause of obstetric emergency in pregnancy and requires emergency surgical intervention of the abdomen. A preoperative diagnosis is very difficult to establish even with the help of traditional investigations and it is usually misdiagnosed as Abruptio placentae, Abdominal pregnancy or any cause of intraperitoneal bleeding[1].

In this article, a patient with torsion of an unicornuate gravid uterus is reported.

Case Report :

A 30 year old woman, 3rd gravida, residing in Lalbag Dhaka was admitted in the department of Obstetrics and Gynaecology of Dhaka Medical College Hospital on 16.09.2000 at 3:00 a.m. with history of amenorrhoea for 6+ months and severe pain in the lower abdomen for 2 hours. She could not specify the date of her last menstruation but detailed history revealed that she was running about 26-28 weeks of pregnancy.

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She experienced an acute attack of pain in the midnight at about 1:00 a.m. The pain was sudden in onset, severe in intensity and was colicky in nature at the beginning. As described by her attendants, she was gradually deteriorating, developed rapidly increasing pallor and had a syncopal attack when she was admitted in Dhaka Medical College Hospital through emergency.

In this pregnancy she attended the out patient department of Dhaka Medical College Hospital for her antenatal checkup and was immunized with two doses of tetanus toxoid. She could not relate any complications detected during her antenatal checkup.

She was married for 10 years and had her last childbirth 7 years back. The delivery was conducted by local dai at home at term and was uneventful. There was history of one MR. 3 years back at 6-7 weeks of gestation.

On examination, she was pale, ill-looking but conscious. Clinically she was severely anaemic, pulse was 136/min but very feeble and Blood Pressure was not recordable_ Respiratory rate was 22/min, Tempeaature 98°F. Examination of Heart and Lungs revealed no abnormality.

Whole abdomen was tense and tender and the examination was not satisfactory. The height of the uterus seemed to be of about 30 week size, but the outline was difficult to delineate. There was no contraction but the uterus was tense on palpation. The foetal heart sound could no be detected. Bowel sounds were sluggish. There was no pervaginal bleeding and the cervix was very

difficult to reach (it was above the normal level of palpation and seemed to be pulled upwards), the cervix was soft, central and closed.

A provisional diagnosis of Abdominal pregnancy was made with a differential diagnosis of Abruptio placentae in mind.

The patient was resuscitated with intravenous fluids, injection Hydrocortisone 4 vials, injection Diclofenac 1 ampoule and two bags of compatible whole human blood. She improved gradually and was found haemodynamically stable at 1:00 a.m. with intravenous fluid.

In the morning, an ultrasonography was done. An intrauterine pregnancy of about 28 weeks size without evidence of any cardiac pulsation was found and a sonographic diagnosis of intrauterine death (I.U.D.) was made.

She was not relieved of pain but later it was dull aching. At about 11:00 a. m. abdomen was found to be more distended and rigid. Finally decision for Laparotomy was taken.

After taking written informed consent, abdomen was opened by right lower paramedian incision under general anaesthesia. A lump (just like an ovarian tumour -at a glance) with smooth congested surfaces (dark blue with patchy black areas) and occupying almost the whole of the abdomen was found. After careful identification of the structures in relation to the round ligament, it was found to be the uterus which was twisted anticlockwise about more than 180 degrees including the tube and ovary of the left side. After untwisting, the uterus was detected to be unicornuate. A transverse incision was given in the lower part of the uterus. A dead male foetus was delivered out and the placenta and the membranes were removed manually. It is important here to mention that a large amount of liquor stained with altered blood was sucked out.

After delivery, uterus was found flabby and there was no sign of uterine contaction. 2 ampoule of injection ergometrine was given intravenously and 4 ampoule of oxytocin was given in 1000 ml of Hartman's solution with a drop rate of 30-40 per minute. I ampoule of injection oxytocin was also given intravenously directly but without any improvement. Moreover, the uterus and left sided tube and ovary seemed devitalized (almost black in colour) and ultimately decision for removal was taken. Right ovary was present in the right lateral wall of the pelvis.

Subtotal hysterectomy with left sided salpingo-oophorectomy was done. Two bags of compatible blood was transfused per-operatively. Abdomen was then closed in layers as usual. The postoperative period was uneventful and she was discharged on the 8th postoperative day.

Discussion:

A minor degree of rotation of the uterus around its long axis is common and insignificant. It is seen specially in pregnancy when the twist is nearly always to the right that is clockwise as seen from above[1]. Torsion is much more common when the uterus is asymmetrical because of a tumour or mullerian duct fusion deformity, or when it is twisted by an adjacent lesion. [1]The torsion is usually at the level of the supravaginal cervix so the uterine vessels are obstructed; the uterus then becomes engorged, infiltrated with blood and ultimately gangrenous[1].

The patient presents with sudden and severe abdominal pain followed by shock. Vomiting and slight uterine bleeding are usual[1]. Occurrence of abnormal lie during labour in absence of detectable uterine pathology may be present in case of partial torsion[2]. The symptoms 'may occur while the patient is sleeping[3]. On examination muscle guarding and tenderness on palpation are present.

Torsion of the gravid uterus is a rare obstetric condition in which the diagnosis is usually made only on laparotomy[5]. Changing position of a fibroid in ultrasonography can give a clue to the preoperative prediction of uterine rotation[5].

This condition has to be distinguished from : Retroplacental haemorrhage (Abruptio Placentae), Abdominal pregnancy, all causes of intraperitoneal haemorrhage, torsion of other organs and ruptured cyst.

When torsion is suspected; immediate lapartotomy is required[1]; the surgical procedure then depends on the exact nature of findings. After laparotomy, the foetus may have to be delivered by posterior wall incision with irreduciable torsion[5,6]. If the torsion is incomplete or recent, the uterus may still be viable; it is then possible to conserve it and to stablize it by suture[1]. Prophylactic shortening of the round ligaments is suggested to prevent recurrent torsion in the immediate a puerperium[6]. When the torsion is sufficient to obstruct the blood supply and the tissues are gangrenous, they have to be removed[1]. Torsion of the gravid uterus may be a life threatening condition which can have a successful outcome if complications encountered in the pre- and post operative periods are treated quickly and effectively[7]. post operative recovery is usually [6,8]. uneventful.

There is an isolated case report where there was asymptomatic axial torsion of the pregnant uterus during mid-trimester because of an ovarian cyst. That case presented as a failure of induction of abortion by extra-amniotic isotonic saline instillation and oxytocin infusion[4].

Conclusion :

Extreme uterine torsion at term is a rare obstetric event and raises critical management consideration. The nonspecific clinical course and rarity of pathological torsion of the gravid uterus makes the preoperative diagnosis difficult. Diagnosis is usually made on laparotomy. The plan of surgery depends on peroperative findings. Prophylactic plication of the round ligaments may prevent torsion in the puerperium. When the tissues are gangrenous or beyond recovery, they are to be removed

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References :

1. Bhatla N. Torsion of the pelvic organs. in: Jeffcoate's Principles of Gynaecology. International Edition, Chapter -16, London / New Delhi, Arnold, 2001: 294-297.
2. Steigrad S. J. Torsion of the gravid uterus. Aust N Z J Obstet Gynaecol, 1987; 27(1) : 66-8.
3. I Lic V. Torsion of the pregnant uterus. Jugosol Ginekol Opstet, 1976; 16(5-6) 379-81.
4. Gupta I., Handa P. R, Khera K. R, Mahajan U. Asymptematic axial torsion of the pregnant uterus: a cause of the failure of induction of mid-trimaster abortion, Asia Oceania J obstet Gynaecol,1991; 17 (3) : 237-9.
5. Bolaji 11., Rafla NM, Mylotte MJ. Classical caesarean section through the posterior uterine wall. Ir J Med Sci 1992; 161(2) : 46-7.
6. Pelosi Ma 3'd, Pelosi MA. Managing extreme uterine torsion at tern. A case report. J Reprod Med 1998; 43(2) :153-7.
7. Ritt MG, Fossum TW. Successful treatment of uterine torsion and foetal retention in a post parturient Great Pyrences bitch with septic peritonitis and protrombotic complications. J Am Anim Hosp Assoc, 1997; 33(6) : 537-9.
8. Biswas MK, Summers P, Schultis SA, Hen= EH, Pernol ML. Torsion of the gravid uterus. A report of two cases. J Report Med 1990; 35(2) : 194-7.
9. Visser A. A., Giesteira M.V., Heyns A., Marais C., Torsion of the gravid uterus. Case reports. Br J. Ostet Gynaecol, 1983; 90(1) : 87-9.

COLLEGE NEWS

Communications

Dear Colleagues,
The Editorial Board of the Journal has opted in introduce this new column where we shall encourage free communications among the readers and authors of the published articles. Opinions and constructive criticisms have always been the tool of development and we expect to make use of it. Your opinions are welcome and we shall make every effort to publish them in subsequent issues.

We shall also undertake to publish in this column personal communications whereby ideas and innovations of researchers may be communicated to the readers if they so desire. This is likely to be in the form of short communications and not necessarily have to follow definite research protocols.

We hope that this shall help infuse dynamism in the journal and shall also improve communications and provide for better understanding among the readers.

We look forward to your continued interest in the journal.

Editor-in Chief

Intercollegiate Scientific Conference 2002 :

An intercollegiate Scientific Conference with

- Bangladesh College of Physicians and Surgeons
- Royal College of Physicians and Surgeons, Glasgow
- Institute of Medicine TUTH, Nepal
- College of Physicians and Surgeons, Pakistan

as "Partners in Progress" is scheduled to be held in BCPS, Dhaka from 28-31st January 2002 . All fellows and members are requested to complete registration formalities and join the conference. Communications may be made through **bcps@bdonline.com.** Further information is available on **www.bcpsbd.org**

Annual General Meeting

The Annual General Meeting is scheduled to be held on 1st March, 2002 (Friday) at the BCPS premises from 8.00 am

Fellowship and Membership Examinations :

The FCPS Part-I and II and MCPS examinations of the college for January 2003 has commenced on schedule from the 1st January 2002. As in previous years a number of senior and reputed academicians of UK, India, Pakistan, Nepal, Singapore, Srilanka have been invited as examiners of the above examinations along with their Bangladesh counterparts.

CORRIGENDUM :

We apologise for the unintentional misprint of the name of one of our valued authors in the **May 2001 issue (Vol-19 No. 2 PP. 71-74).**

The author's name " Dr. M. HOSSAIN, MBBS[b]. Should read as, "Dr. AKMM ISLAM, MBBS[b] and b. Dr. Monwar Hossain, MBBS, Medical Officer, BIRDEM, Dhaka should read as "b. Dr. AKM Monowarul Islam, MBBS. Medical Officer, BIRDEM, Dhaka".