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Aims & Scope

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Research Articles

Research article

Comparison of Platelet Count in Malaria Positive and Negative Subjects with Hematology Analyzer and Microscopic Examination

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Abstract
This study included 250 blood samples submitted for malaria investigation and were evaluated under microscope for malarial parasites and platelet count. All samples were additionally analyzed for platelet count with automated haematology analyzer. Thirty seven (37) samples were found to be malaria positive microscopically. Out of 37 cases with malaria positive microscopically, thrombocytopenia was observed in 24 (64%) cases of malaria. So there is association of thrombocytopenia with malaria.

Key words: Malaria, thrombocytopenia, P. falciparum

Introduction

Over 300 to 500 million people are infected by malaria yearly[1] with the mortality rate of 1%[2]. It is epidemic and endemic in Africa, Central and South America, the Middle East and parts of Asia; regions with hot, humid environment which is ideal for breading of anopheles mosquito and is transmitted by bite of infected female anopheles mosquito. It is also transmitted by blood transfusion, trans-placentally and between drug addicts by reusing syringes[3]. Four species of plasmodia i.e. P. vivax, P.ovale, P. falciparum and P. malariae can cause malaria which is distinguishable on peripheral blood smear[4]. Plasmodium sporozoites are injected by the bite of female mosquito, reach liver, multiply there and released after 1-2 weeks to infect red cells. Severe cases of malaria are seen in falciparum infection. The severity of malaria may be determined by the magnitude of parasitaemia. The malaria is usually presented with febrile paroxysms, malaise and anemia[2]. The main hematological findings in patient's blood are anemia, thrombocytopenia, variable (low, normal, high) white cell (WBC) count, bleeding and parasitaemia[2,5]. Malaria is still common in oasis and costal areas of the Saudi Arabia. Expatriate work force also imports malaria from their home countries especially endemic areas of malaria[6]. This study aimed to evaluate malaria and platelet count to assess correlation.

Methodology

Two hundred and fifty (n=250) adult subjects suspected of malaria were selected from Riyadh Medical

Complex. Thirty seven (37) were found positive for malarial infection microscopically by thick and thin smears. Platelet counts were performed manually[7] as well as with automated hematology analyzer (Cell Dyn 3700). All slides were stained by Giemsa method[8]. Identification and level of parasitemia was done for each positive case[9].

Results

In this study two types of plasmodium species, P. falciparum and P. vivax were found on thick and thin smear. There were 34(92%) cases of falciparum and 3(8%) cases of vivax. The comparison of positive and negative cases is given in Table 1. Five (20.1%) patients had platelet count less than 50×10^9/L, eleven (45.1%) had thrombocytopenia in the range of 50- 100×10^9/L and eight (34.8%) had thrombocytopenia in the range of 100 – 150 x 10^9/L. Out of these five patients, three had parasitemia in the range of 3-10%. Maximum parasitemia was found to be 10% while 0.1% was the lowest. There were 8 (21.6%) patients who have parasitemia of 1% or above. These patients had platelet count of 60×10^9/L or less and this high parasitemia was inversely related to platelet count. A low platelet count was associated with high parasitaemia ($p<0.05$).

Table - 1: Platelet count in malaria positive and negative cases with microscopy and hematology analyzer

Methods	Platelet count in malaria positive cases	Platelet count in malaria negative cases
Microscopy	117 ± 35.06	285.6 ± 41.01
Hematology analyzer	137 ± 48.13	292.3 ± 40.3

Discussion

Thrombocytopenia is a well-documented finding in falciparum malaria and in mixed falciparum/ vivax infection [10,11]. A platelet count less than 150×10^9/L was considered thrombocytopenia but is not associated with adverse outcome[12]. Thrombocytopenia is considered as an important indicator of malaria [13]. Maximum thrombocytopenia occurs on the fifth or sixth day of infection and gradually returns to normal within 5-7 days after parasitemia has ceased [14]. In the present study thrombocytopenia of less than 150×10^9/L was found in 24 (65%) of the malaria cases. Mean platelet count in P. falciparum infection was 141×10^9/L in hematology analyzer while on microscopy the mean platelet count was 120×10^9/L.

Thrombocytopenia has been observed in 60-80% of both P. falciparum and vivax infection[15]. The shortened life span of platelet is 2–3 days in comparison to 7- 10 days in normal controls [15,16]. The mechanism of thrombocytopenia in malaria is still unclear. Fajardo and Tallent [17] suggested a direct lytic effect of parasite on platelets. Both non-immunological destruction and immunological mechanism involving platelet specific antibodies have been demonstrated [18-19]. Mohanty et al suggested that thrombocytopenia in malaria is partly immune mediated[20]. During malarial infection, initial

hyperactivity results in aggregation and later hypoactivity of platelets causes intravascular lysis. There is peripheral destruction and consumption of platelet in infected persons. Srichaikul noted that despite thrombocytopenia, the number of megakarocytes in the bone marrow remained adequate or increased in malarial infection [18]. Ladhani et al found that a low platelet count is associated with parasite density but not with bleeding problem or mortality [13].

Thus screening complete blood count can be a rapid and inexpensive yet valuable component in the diagnostic investigation of any patient suspected of malaria, particularly the patient with pyrexia of unknown origin and thrombocytopenia.

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Case report

Unusual Presentation of Factor XII Deficiency with Bleeding: A Rare Case Report

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Abstract:
Factor XII (FXII) is a coagulation protein involved in the initiation of coagulation via contact activation system. Congenital FXII deficiency is a rare, asymptomatic disorder, associated with an isolated prolonged activated partial thromboplastin time (APTT). FXII deficiency is not commonly associated with any bleeding symptom except for a few cases presenting with occasional minor bleeds, which does not require treatment. Instead, a few literature reports suggest an increased incidence of various thromboembolic events in these patients. We report a rare occurrence of FXII deficiency presenting with severe bleeding symptoms.

Key words: Factor XII deficiency, contact activation system, thromboembolic events.

Introduction

Factor XII (FXII), also known as Hageman factor, is an 80 kDa plasma protein synthesized in liver. It is involved in coagulation of blood via contact activation as seen in the activated partial thromboplastin time (APTT). Activation of FXII occurs when the complex of FXII, factor XI (FXI), pre- kallikrein, and high molecular weight kininogen contact a negatively charged surface. FXII subsequently activates FXI and thus plays a role in fibrin clot formation. A rather more important role of FXII is the conversion of plasminogen to plasmin and initiation of fibrinolysis[1].

Congenital FXII deficiency is inherited as an autosomal recessive disorder with a very low incidence of approximately 1 in a million individuals.[2] Rare cases with autosomal dominant pattern have also been reported.[3] The gene for FXII is located on chromosome 5. FXII deficiency is usually asymptomatic and associated with prolongation of APTT.[4] The condition is usually diagnosed incidentally e.g. during pre-operative coagulation work up. FXII deficiency is normally asymptomatic, however, a few patients with occasional minor bleeds have been previously reported.[3] In contrast, this disorder has been associated with a significantly increased risk of thrombosis, due to impaired fibrinolytic system.[4]

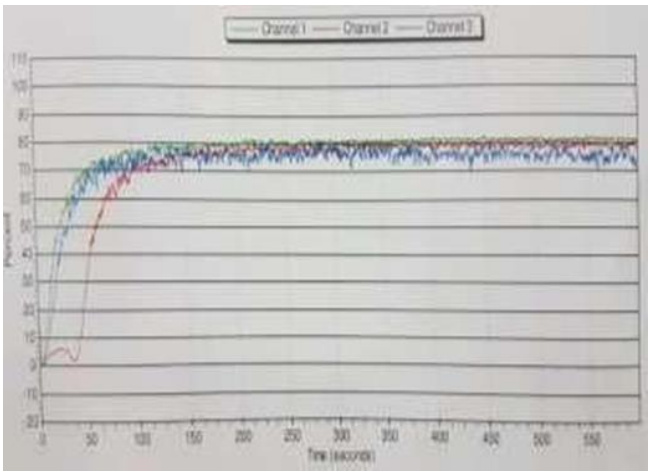
Case Report

A 17 years old female born to consanguineously married couple presented to us for diagnostic work up of a suspected bleeding disorder. She complained off and on right knee joint pain with associated swelling for the past two years. Her past history included several episodes of epistaxis, gum bleeding, per rectal bleeding, melena and spontaneous bruising since childhood. She was transfused multiple times with whole blood and plasma for the mentioned bleeding symptoms; the symptoms would resolve with the transfusions. Two years back, she underwent exploratory laparotomy and abdominal lavage due to primary peritonitis. In the process she was given multiple fresh frozen plasma (FFP) and whole blood transfusions. She also complained of menorrhagia since menarche. Her complete blood count showed haemoglobin: 12 g/dl, total leukocyte count: 5.8 x 109/l, platelet count: 196 x 109/l. Coagulation screen revealed a normal prothrombin time (PT) of 12 seconds, (reference range 10-13sec), bleeding time: 3 minutes (reference range 3-7 min) with a prolonged APTT: more than 120 sec (reference range 24-32sec). A 1:1 mixture of patient's plasma and pooled normal plasma revealed complete correction of patient's APTT, excluding the presence of any inhibitor. We further performed coagulation assays of factors VIII, IX, XI and XII, (performed on Sysmex CA 550) as these clotting factor deficiencies can cause a prolonged APTT. Our results revealed normal levels of factors VIII, IX and IX with absent FXII. Von Willebrand antigen (Sysmex CA 500) levels and ristocetin co-factor activity (Aggram Helena) were also within normal limits. As FXII deficiency is usually not associated with any bleeding symptoms we further performed fibrinogen assays (Sysmex CA 50), platelet aggregation studies (Aggram Helena) and factor XIII (FXIII) levels to rule out any concomitant pathology; all of these turned out to be normal. Work up for renal and autoimmune disorders was also done to exclude the possibility of acquired deficiency. Thus, she was diagnosed as a case of congenital FXII deficiency. Interestingly, no mutations in the factor XII gene were found on Snager's sequencing. She was put on oral hormonal contraceptive for her menorrrhagia. FFPs were given at 10ml/kg to treat right knee joint haemarthrosis after which her symptoms were resolved. She was offered physiotherapy for further management.

Discussion

FXII deficiency is a blood disorder with a very low incidence. It may be congenital, in which case it is usually diagnosed incidentally by an isolated prolonged APTT during routine coagulation screen. Other related disorders associated with a prolonged APTT include deficiency of factors VIII, IX, XI, contact factors, Von Willebrands disease as well as a few inhibitors of coagulation including lupus anticoagulant and acquired inhibitors against various coagulation factors e.g factor VIII. However, FXII deficiency is usually not associated with any bleeding manifestations, in contrast to other clotting factor deficiencies e.g factor VIII, IX etc. Another acquired form of factor XII deficiency may be caused by inhibitors against FXII. This has been reported in patients with nephrotic syndrome and leukemia.5 Conversely, these patients have an increased risk of thromboembolic phenomenon due to impaired fibrinolysis in FXII deficiency. Various researchers have reported association of FXII deficiency with myocardial infarction, pulmonary embolism and other life threatening thrombotic episodes.6

Parameter	Patient Value	Reference Range
Bleeding score	19*	-
First Line Coagulation Screening		
Bleeding time	3 min	3-7 min
PT	12 sec	10-13 sec
APTT	More than 120 sec	24-32 sec
Fibrinogen	213mg/dl	180-350 mg/dl
Intrinsic Pathway		
Factor VIII	141%	50-150%
Factor IX	91%	50-150%
Factor XI	81%	50-150%
Factor XII	0%	50-150%
Associated Factors		
Factor XIII	76%	50-150%
VWF Antigen	89%	50-200%
Ricof	94%	50-200%
Platelet disorders	Normal platelet aggregation	-
Autoimmune Work Up		
Anti ds- DNA	1.68 U/ml	<20 U/ml
ANA	<1:100 (negative)	<1:100
Renal Work Up		
Urea	20 mg/dl	10-50 mg/dl
Creatinine	1.1 mg/dl	0.7- 1.3 mg/dl
Genetic Work up		
Snager's sequencing	No mutation detected	



Platelet Aggregometry Results

Ristocetin: 80.9%

Collagen : 80.7%

ADP : 82%

Few reports have also emphasized that female patients with Hageman factor deficiency might also be at a higher risk for recurrent pregnancy losses.7, 8 Therefore, once diagnosed, these patients should be followed up closely for any thrombotic events. In a study carried out on all the available members of Swiss families affected by factor XII deficiency, it was found that patients with homozygous FXII deficiency are more likely to develop thromboembolic disease whereas partial FXII deficiency is not usually associated with thrombosis.9 However, different subsequent studies have shown that these patients developed thrombosis due to other risk factors rather than factor XII deficiency.10,11 Recently, researchers are working on newer anticoagulants targeting factor XII.11 There were a few limitations in this case report. Antigen level of FXII could not be performed because of non-availability of required resources and the diagnosis had to be made solely based on FXII activity. Similarly, we were unable to rule out any concomitant deficiency of other contact factors e.g pre-kallikrein, high molecular weight kininogen etc.

Conclusion

FXII deficiency is a rare genetic blood disorder; usually not associated with any bleeding tendencies. Moreover, these patients also do not bleed following invasive procedures such as surgery and dental extraction. Thus, these patients routinely do not need any treatment or prophylactic measures. However, as reported in our case a few patients with FXII deficiency may present with bleeding symptoms and

should be managed accordingly. A probable cause for these haemorrhagic incidents in our case might be the complete absence of FXII activity in the plasma. The purpose of this case report is to bring forth the rare occurrence of severe bleeding manifestations in patients with FXII deficiency.

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Research article

Patterns, Types and Causes of Mandibular Condyle Fractures, association with other Mandibular Fractures at LUMHS Hospital Hyderabad, Sindh

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Abstract:
The objective of current study was to assess the patterns, causes and types of mandible condylar fractures and relationship between mandible condylar fractures with associated mandible fractures. This prospective study was conducted at Oral & Maxillofacial surgery department LUMHS Jamshoro/ Hyderabad. History, clinical examination and radiographic analysis of 60 mandibular condylar fracture patients were performed and data were recorded. Data was analyzed on SPSS 16. Out of total 60 patients with mandible condylar fractures, n=51 (85%) were male and n= 09 (15%) were female. Age range of patients was 4 to 56 years) and mean age of 28 years and third decade was most common. The road traffic accident was common cause of mandibular condylar fractures n=29 (48.33%) followed by assault n=14 (23.33%) and fall n=12 (20%). Unilateral fractures were n=49 (82%) and n=11 (18%) were bilateral fractures. Mandible Subcondylar fractures were the most common, both in the unilateral and bilateral groups which accounted for 38 (63%), condylar neck were 16 (27%), condylar head were 06 (10%). 36 (60%) were isolated condylar fractures and 24 (40%) were associated with other mandible fractures. Road traffic accident was found to be common cause of condylar fracture. Young male adults were involved in most of the accidents, with the unilateral pattern and subcondylar fractures type were more common. It divulges inadequate road traffic logic in road users, lack of road protection methods and rule in our population.

Key words: Assault, Mandible condylar fractures, Road traffic accident and Subcondylar fracture.

Introduction

Mandible is the most commonly fractured bone in facial region. [1-3] An analysis of fractures frequency in different anatomic sites of the mandible shown that, condylar fractures are most common fractures in the mandible [2,4,5] . It is also frequently over-looked and least detected fracture site in the facial region.[6] Mandibular condyle is involved in up to one third of all mandible fractures, with a frequency that range

from 10% to 57%. [7-9] Several studies suggest that after the angle and body fracture, the second most mandible fractures are condyle region. [7,10] while few studies showed that condyle fractures are most common after symphysis and parasymphysis. [1, 9] Direct or indirect trauma can fracture condyle, usually result from force applied at the body or symphysis region which is transmitted to the condylar process. [11,12]

Etiology of the condyle fractures varies in accordance with both sociologic and age factors. Different studies have confirmed that motor vehicle accidents are the most common cause,[1,13] others proved that IPV are most common cause, [11,14,15] while falls are also predominant cause in few studies, especially in females and young children.[6,16] Condylar fractures may occur isolated or associated with other mandible fractures.[14] Extracapsular fractures are more predominant than intracapsular in all age, subcondylar fractures are more common in adults than children.[14,17] In particular, in children mandibular fractures differ significantly from adults because of growth and inadequate dentition of the mandible.[16] In children the condylar intracapsular fracture usually cause temporo-mandibular joint ankylosis and masticatory disability.[16] Treatment of mandible condylar fracture is still controversial in respect of conservatively or surgically. Principal factors that determine the treatment decision include level of fracture, site of fracture, and patient's age. [18, 19] Therefore it is important for both functional and cosmetic reasons that condylar fractures should be properly diagnosed and adequately treated. There have been many articles published about fractured mandible, but only few of them have focus on patterns of mandibular condylar fractures. This study will determine the patterns of mandibular condylar fractures, with all their relative characteristics, common age group and causes of condylar fracture in this part of the country. This will help in proper diagnosis and management of fractured mandibular condyle.

Patients and Methods

This descriptive study was conducted at LUMHS Hyderabad. A total of 60 patients of mandibular condylar fracture who attended the OPD or Emergency department from 1st July 2014 to 30th June 2015 were part of this study. An informed consent was taken from the all patients or their attendants. History of trauma was asked from the patient or attendant and recorded. Diagnosis of mandibular condylar fracture was made by history, clinical findings and appropriate radiographs. Conventional radiographs orthopentomogram (OPG) and postero-anterior view of face (P.A) X-rays were used for all the patients. All the significant information was recorded on pre-designed proforma, including patient's demographic data, etiology, patterns and relationship of fractures. We classified fractures as described by Lindahl classification of fracture mandible condyle; Condylar head fracture, Condylar neck fracture and Sub condylar fracture. Data was analyzed on (SPSS) version 16.0. Categorical variables (ie sex, etiologies, and pattern) was analyzed by frequency and percentages and for continuous variables, (like age) mean was computed. No inferential test was used because of descriptive statistics.

Results

Out of 60 patients, n=51 (85%) were males and n=9 (15%) were females, with male/female ratio of 5.7:1. Males were predominant in all age groups of patients. Road traffic accident (RTA) was the common cause

of mandibular condylar fractures n=29 (48.33%) followed by inter personal violence (IPV), n=14 (23.33%). Male patients were predominated involved in RTA and IPV, which accounted 27 (45%) and 14 (23.33%) respectively. While fall was more prevalent in females. Among unilateral fractures, right side was 27 (55%) slightly more than left side which accounted 22 (45%). Bilateral condylar fractures were mostly occurred by considerable force through (RTA and fall) as compared to lesser force through (Assault). Of the total samples, n=36 (60%) were isolated condylar fractures and n=24 (40%) were associated with other mandibular fractures.

Table 1: Age distribution with Gender of condylar mandibular fractures.

Age (yrs)	Male	Female	Total	Percentage
01-10	06	02	08	13%
11-20	08	02	10	17%
21-30	18	02	20	33%
31-40	12	00	12	20%
41-50	04	02	06	10%
51-60	03	01	04	07%
Total	**51**	**09**	**60**	**100%**

Table 2: Causes of the mandibular condylar fractures with Gender

Etiology	Male %	Female %	Total %
RTA	27 (45%)	02(3.33%)	29 (48.33%)
IPV	14 (23.33%)	_	14 (23.33%)
Fall	05 (8.33%)	07(11.67%)	12 (20%)
Sport	03 (5%)	_	03 (5%)
Others	02 (3.33%)	_	02 (3.33%)
Total	51 (85%)	09 (15%)	60 (100%)

Table 3: Pattern of Mandibular Condylar fracture.

Pattern	Male	Female	Total	Percentage
Bilateral fracture	09	02	11	**18%**
Unilateral fracture	42	07	49	**82%**
Total	**51**	**09**	**60**	**100%**

Table 4: Types of Mandibular Condylar fractures.

Etiology	Condylar Head	Condylar Neck	Sub Condylar	Total
RTA	O2	07	20	29
IPV	--	02	12	14
Fall	04	05	03	12
Sports	--	01	02	03
Others	--	01	01	02
Total %	**06(10%)**	**16(27%)**	**38(63%)**	**60(100)**

Table 5: Isolated and Combine fractures of mandible condyle region

Pattern	Bilateral	Unilateral	Total	Percentage
Combine fracture	02	22	24	40%
Isolated fracture	09	27	36	60%
Total	**11**	**49**	**60**	**100%**

Discussion

Among Gender 85% (n=51) of patients presenting with mandible condyle fractures were males, and only 15 % (n=9) were females, this male dominance in this study could be because of such fractures result from road traffic accident, assault, fall, sports injury etc, where men are dynamically involved. This ratio is comparable to those reported by Kim YK at al [19] Bormann K H et al, [11] and Silvennoinen U et al. [14]

However it is greater than that reported by Amaratunga N.A.de S, [20] Thoren H et al. [21] Marker P et al. [15] Increase in female ratio in their studies indicates that women in this part of world are more vulnerable to facial injuries, because of their participation in outdoor activities. The low ratio of female in our study was because most female are house bound and their outdoor activities are limited.

Male in the third decade constituted the major group in this study, similar to the data reported by Sawazaki R et al, [13] Silvennoinen U et al, [14] and Marker P et al.[15] During this age of life, the young adults are dynamically involved in outdoor activities, which makes them more prone to facial injuries in this age. The causes of facial injuries have changed by decade to decade. Data reported in our study showed, road trafficaccident was the common cause of mandible condyle fracture 29 (48.33%). This result was different from the results of Kim YK et al,[19] Silvennoinen U et al,[14] Rikhotso E et al,[23] and Adam CD et al [24] from Australia, this show that the causes of maxillofacial trauma varies because of social, cultural and geographic setup. The result of this study is comparable with those reported by Klenk G et al, [10] and Abbas I et al. [25] In our country, road traffic related maxillofacial injuries occurred because of not properly followed traffic regulations , poor condition of vehicles and inappropriate size of roads. As compared to developed countries there is, well-trained drivers, broader roads, and traffic regulation is also strictly followed which make different in accident.

Subcondylar fractures were most common in present study that was 63%, and Condylar neck fractures 27%, which is comparable with study of Silvennoinen U et al,[14] and zachariades N et al, [19] but different from Sawazare R et al, [13] and Marker P et al. [15] studies. Present study observed condylar head fractures 10% and mostly occurred in below age 6 years, similar to the data reported by Choi J et al, [6] and Thoren H et al. [22] Type of condylar fractures seems to be influenced directly by its cause. Subcondylar fractures are tension failures in response to bending of the mandibular neck, because the mandible distributes the force of impact, frequently fractures occur only in subcondylar region. [26] In present study unilateral condylar fractures were reported 49 (82%), similar to the data observed by previous studies in Finland 1992, [14] and South Africa 2008 [23] and Denmark 2000, [15] Among unilateral condylar fractures right side was encounter more as compare to left side in our study.

In our study 36 (60%) of condylar fractures were isolated and 24 (40%) were combine with other mandible fractures. Those results are comparable with previous study by Silvennoinen u et al, [14] and Newman A. [17] It seems that condylar fractures consequences from an indirect force applied to the mandible [26] This submits that condyle fractures may be the result of the exertion of force which is not fully absorbed in the majority of cases in the area of its primary application, i.e the mental region.

Conclusion

This study showed that RTA were the main cause of condylar fractures, Subcondylar fracture and unilateral mandibular condylar fracture is more common. Diagnosis of patterns and types of condylar fracture is essential for proper treatment and management.

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Research article

Determine the Influence of Prenatal Stress on Fetal Development

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Abstract:
Prenatal stress is an epigenetic factor which can produce long-lasting alterations in brain structures and body functions. The objective of this was to examine the ratio of prenatal stress among pregnant women. It is necessary to assess stress separately at each trimester of pregnancy to differentiate chronic from acute stress in order to evaluate the effects on child development. Prenatal stress can alter neurotropic growth factors, synapse development, neurotransmitter levels and also adult neuron development. Prenatal stresses significantly influence the development of the brain and the organisation of behaviour. Prenatal stress can also be the main cause of hypertension, type 2 diabetes, schizophrenia and cardiac disorders in adulthood. But the impact of stress is not very clear. The survey based on a cross-sectional study was conducted in Karachi. The sample size calculated for the survey is 45. The pregnant women were asked to fill the questionnaire of 23 questions.

Keywords: Prenatal stress, maternal stress, antenatal stress, fetal development & depression.

Introduction

Prenatal stress (or antenatal maternal stress) is exposure of the associated expectant mother to worry, which might be caused by disagreeable life events or by environmental hardships [1]. The ensuing changes in the mother's secretion and the system might hurt the fetuses (and when birth, the infant's) immune perform and brain development [2]

Prenatal stress is shown to possess many effects in vertebrate brain development. Within the hippocampus of male rats, antenatal stress has shown to decrease the speed of proliferation and necrobiosis within the hypothalamus-pituitary axis[3,4]. Antenatal stressed animals have prolonged glucocorticoid response. Removing the adrenal glands of the mother eliminates the impact of the pup's glucocorticoid response. Supplementing the adrenalectomized mother with glucocorticoid, saved the

hypothalamic-pituitary-axis response to maternal stress for prenatally stressed offspring[5]. Antenatal stress caused high glucocorticoids that successively affect the hypothalamic- pituitary-axis feedback [6]. A study by García-Cáceres et al. showed that antenatal stress decreases cell turnover and proliferation within the neural structure of adult rats that reduces structural physical property and reduces the response to worry in adulthood [7]. This study conjointly showed that once prenatally stressed rats were stressed in adulthood the females showed a rise in corticotrophin-releasing internal secretion suggesting it to be associate up-regulation within the hypothalamic-pituitary adrenal axis8. Males showed no elevation of glucocorticoid levels. Increase in adrenocorticotropic internal secretion adrenocorticotrophic hormone ACTH hormone, endocrine internal secretion} with no impact of adult stress and a decrease within the corticotrophin-releasing hormone ribonucleic acid within the neural structure showed a down-regulation. The author concludes that this makes prenatally stressed females less reactive to later life stressors than males. 9-11

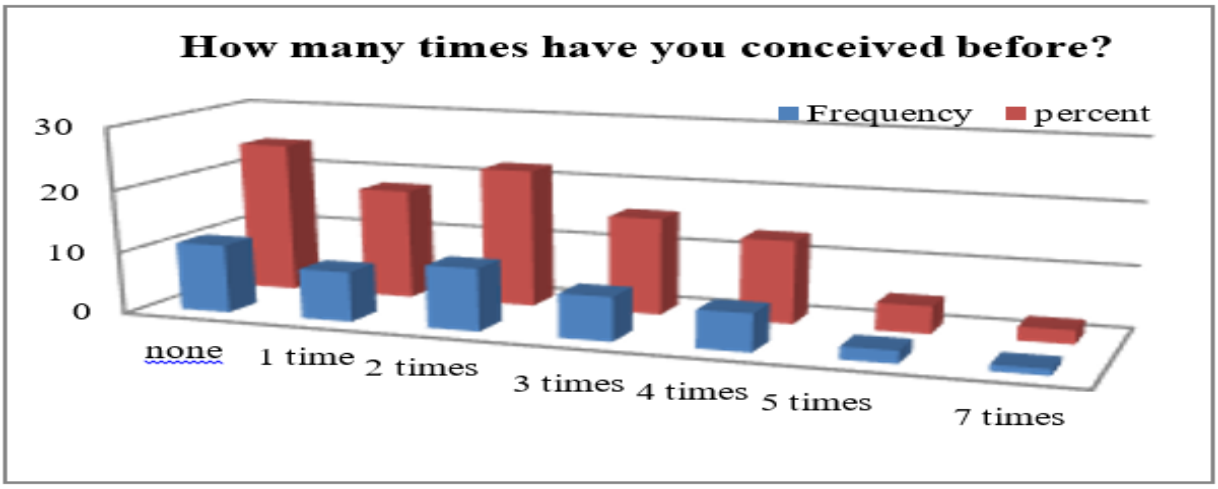
Methodology

The cross-sectional study was conducted in Karachi in November 2016, the surveillance study was conducted including pregnant women in the city. The sample size calculated for the survey was 45. A survey questionnaire was developed to address the study questions and was disseminated among the local population. The respondents were asked to answer 23 open- ended questions.

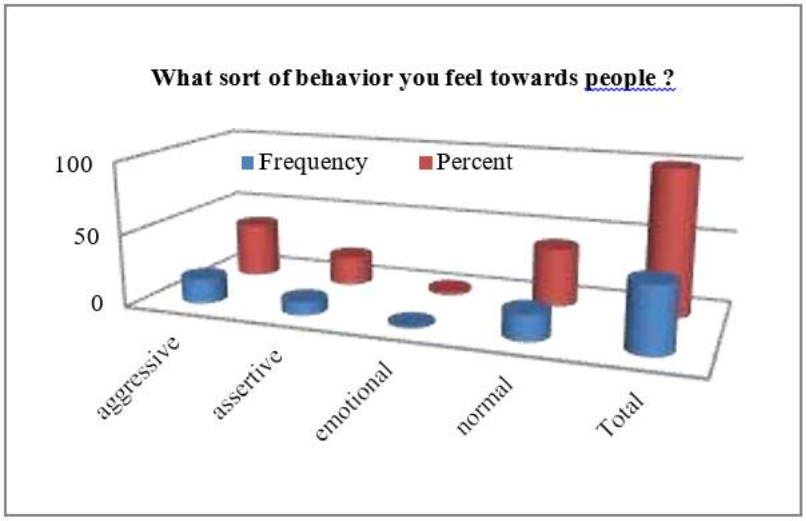
According to our estimation, we analyzed that majority of pregnant women are the prey of prenatal stress. Most of them didn't know about prenatal stress as well as its adverse effects that come in the form of fetal abnormalities.

Result

According to our report, 40% women feel changes in their behaviour due to prenatal stress and 60% have normal behaviour. Only 40% women during pregnancy feel changes in appetite, 24.4% don't feel changes in appetite. In our sample size 11.1% suffers environmental stress, 22.2% feels stress due to personal issues and 17.8 % suffers stress due to financial problem. In their relations with family and spouse, only 4.4% have a harsh relation, 11.1% have a good relationship with their family and spouse while others have a normal relation. The stress due to baby's gender, 51.1% pregnant women have stress about baby's gender from their family. There is a good ratio that 57.8 pregnant women are aware of abnormality of the fetus due to stress.



Questions	Frequency %	Significance Value
Do you feel craving towards anything?	53.3	0.467
Do you feel that your sense of smell is increased during pregnancy?	46.7	0.199
Do you feel of being strictly spic and span?	40	0.511
Do you feel of being more forgetful?	42.2	0.557
Do you feel any kind of breathing problems?	46.7	0.15
Do you feel any sort of problem in sleeping?	44.4	0.91
Do you feel of being chronic towards any disease?	22.2	0.596
Do you feel pain in any part of your body?	71.1	0.437
Any sort of pressure you feel from your family?	60	0.258
Do you have pressure about baby's gender from your in-laws?	51.1	0.776
Do you know that stress during pregnancy can cause abnormality in your child?	57.8	0.792



Discussion

According to CharilA et'al [12] reports that maternal mood is affected by stress during pregnancy and the physiology of fetus. As per our reports, we concluded that 40% maternal mood is affected by prenatal stress. In April 1991, a longitudinal study was conducted by T Deaveet'al [13]. The report suggests that prenatal stress is the cause of child development abnormality. As per our report, we concluded that 57.8% maternal stress can cause abnormality in child development. Laura Palagini [14] in August 2014 in a review article suggested that insomnia in pregnancy is a determinant of pregnancy outcome. As per our reports, 44.4%

suffered from insomnia during pregnancy. Helen Christensen [15] in their reports suggested that pregnancy mothers become forgetful during pregnancy. As per or reports, 42.2% mothers become forgetful due to stress. W. Hofhuis and his team [16] in their research suggested that breathing problems like apnea occurs during pregnancy and also due to maternal smoking. As per our reports, 46.7% pregnant women suffer breathing problems. Matthew W Gillman [17] in his reports showed that chronic diseases can be the cause of prenatal stress. As per or reports, we concluded that 22.2% maternal women are being chronic towards diseases like hypertension, diabetes type 2 and glucose intolerance. According to our reports; 53.3% feel craving towards anything, 71.1% feel pain in the body, 60% feel some sort of pressure from family and 51.1% have pressure about baby's gender from their in-laws.

Conclusion

As per our reports, we come up with the conclusion that: Excessive use of abuse material such as alcohol, cocaine, caffeine, or tobacco is one of the major causes of fetus growth retardation. Unhealthy relationships, physical or mental abuse, breakups of the relations or persistent hypertension also play a major role in fetus retardation and in depressed fetal physical or mental growth. By this cross- sectional study, we have tried to aware the majority of women about prenatal stress and its consequences regarding fetal development. To overcome the consequences and inconveniences of prenatal stress, we have to take some healthy and beneficial steps, in order to let the people know about it, awareness programs, seminars, and workshops should be arranged, NGO's should be run to eliminate the marital abuse from the society, special units should be developed in the rehabilitation centers for pregnant women.

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