Hematological changes in preeclampsia

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Abstract

Preeclampsia occurs in approximately 7% of all pregnant women. This study was aimed to determine the relative frequency of alteration in some of the haematological and biochemical parameters in patients with preeclampsia. To identify the association between different clinical and haematological parameters in preeclampsia. Prospective clinico-haematological study. The study was conducted in Mosul teaching obstetric hospitals during a period of six months (January-June 2002). Sixty patients with preeclampsia were included in this study. This study include clinical evaluation, complete blood cell count with platelet count, erythrocyte sedimentation rate, coagulation profile, Liver function tests and renal function tests, serum uric acid and test for proteinuria were done. Anaemia (depending on packed cell volume) was encountered in (15%). Erythrocyte sedimentation rate values were related to plasma fibrinogen levels. The most common haemostatic abnormalities in descending order were; shortened activated partial thromboplastin time (60%), positive D-dimer reaction (51.6 %), hypofibrinogenaemia (35%), borderline prolonged bleeding time (28.4 %), shortened prothrombin time (10%), thrombocytopenia (5%) and prolonged bleeding time (1.6%). Preeclampsia is an augmentation of the hypercoagulable state (prothrombotic state) of normal pregnancy. Haemostatic parameter abnormalities were more evident in mild preeclampsia , lower systolic and diastolic blood pressure, anaemia, lower packed cell volume and 1+ proteinuria, Packed cell volume and activated partial thromboplastin time had a positive correlation (R=0.5). Liver function tests were normal in the majority of the studied patients. HELLP syndrome had an incidence of (3.3%). Renal impairment was found in over (50 %) of the studied patients. In the present study, anemia is not a common feature in preeclampsia (15%). Lower mean erythrocyte sedimentation rate value was found in hypofibrinogenic group (35%) (37.6 mm/h) than normal fibrinogen group (65%) (49.9 mm/h). The most common haemostatic abnormality was shortened activated partial thromboplastin time (60%). Preeclampsia is an augmentation of the hypercoagulable (prothrombotic) state accompanying normal pregnancy inducing a state of chronic disseminated intravascular coagulopathy. Mild preeclampsia shows more haemostatic changes compatible to hypercoagulable state than severe preeclampsia. Renal impairment was encountered in over 50% of our studied patients. S.urea and s.uric acid had a close positive correlation. Severe preeclampsia had higher mean s.uric acid compared to mild preeclampsia. Liver impairment was not commonly encountered (10%). HELLP syndrome incidence was (3.3%).

Key wards: Preeclampsia, hypertension in pregnancy, coagulopathy.

Introduction

Preeclampsia (PE) is a condition specific to pregnancy. It arises after the 20th week of gestation and is characterized by hypertension and proteinuria. Oedema may also be present [1]. Preeclampsia occurs in approximately 7% of all pregnant women. The condition is induced by pregnancy and resolves after childbirth [2, 3].

Patients and Methods

During a period of six months (January-June 2002) a total of 60 patients were studied from the departments of gynecology and obstetrics in Al-batool hospital, Al-khansaa hospital and AL-Salaam general hospital .The diagnosis was based on clinical and laboratory criteria, and clinical evaluation was done for each patient.

Pooled plasma obtained from three healthy males and processed along- with patient's plasma and the results were within normal range to ensure the test validity.

This study includes clinical evaluation, complete blood cell count with platelet count, erythrocyte sedimentation rate

(ESR). Coagulation profile including skin bleeding time (B.T) prothrombin time (PT), activated partial thromboplastin time (APTT), plasma fibrinogen level and plasma D-dimer reaction .Liver function tests including total serum bilirubin (TSB), direct (DSB) and indirect serum bilirubin (IDSB), s.alanine transaminase (ALT), s.aspartate transaminase (AST) and s.alkaline phosphatase (ALP) and renal function tests (serum urea and serum createnine), s. uric acid, general urine examination and proteinuria were done . All the above procedures were done according to Practical Haematology [4] and according to manufacturer illustrations supplied with each kit.

Anova test, Z-test two mean, post hock (LSD), chi- square and correlation analysis were performed using statistical computer programs: Excel program under windows 98, Mini-tab program under windows 98. At the university of Mosul / Medical College / Department of Community Medicine. P-value of less than 0.05 was considered significant.

The Diagnostic Criteria for PE are [5]:

1-Hypertension.

2-Proteinuria.

3-Pregnancy beyond 20th weeks of gestation.

4- Negative history of hypertension before 20th weeks of gestation and before pregnancy.

5-Negative history of renal problems.

6-Negative history of blood transfusion at least one year before pregnancy.

The Definition of hypertension is [5]: Blood pressure should be measured at sitting position with the cuff enough for the subject's arm. Several definitions of hypertension exist. In United Kingdome, the most widely used is that of Davey and MacGillirray(1986) which is based on diastolic blood pressure (Krotkoff phase K4). One measurement of diastolic blood pressure of (110) mmHg or two consecutive measurements of diastolic blood pressure equal or more than (90) mmHg four hours or more apart.

The Definition of proteinuria is [5]: Two random clean catches of urine specimens with (2+) or more or (1+) if specific gravity is less than (1030).

So mild PE has both blood pressure with diastolic equal or more than 90 mmHg and proteinuria (1+) to (2+), while the severe type is considered if the patient had one or more of the following in addition to what was mentioned above [5] :

1. Systolic blood pressure equal or more than (160) mmHg or diastolic blood pressure equal or more than (110) mmHg .These levels of blood pressure must sustained at least four hours apart.
2. Proteinuria (3+) or more [6, 7] .
3. Oliguria (equal or less than 400 ml in 24 hours)
4. Cerebral or visual disturbance.
5. Epigastric pain.
6. pulmonary edema or cyanosis.
7. Impaired liver function.
8. Thrombocytopenia.

Results

Liver function tests were normal in the majority of the patients; TSB was normal (up to 1 mg/dl) in 56/60 (93.3%) and elevated in 4 /60 (6.7%), ALT was normal (up to 12 IU/L) in 54/60 (90%) and elevated in 6/60 (10%); ALP was normal (up to 32.5 K.A.U/dl) in all cases except in one case (1.6%). Proteinuria: 28/60 (46.6%) had (1+) proteinuria, 19/60 (31.7 %) had (2+) proteinuria, and 13/60 (21.7 %) had (3+) proteinuria. General urine examination was performed for all our studied patients. Any patient with urinary tract infection was excluded from our study.

In anaemic (9/60) and nonanaemic (51/60) groups of patients depending on the packed cell volume (PCV).There was a statistically significant difference ($P<0.05$) concerning the points below between the anaemic and nonanaemic groups:

1- The anaemic group 5/9 patients (55%) had borderline bleeding time while the non-anaemic group only 12/51(23%) had borderline bleeding time (χ^2).

2- Mean APTT was shorter in anaemic group (22.3 second) compared with non-anaemic group (27.2 second) (Z-test two mean).

3- D- dimer reaction was positive in 8/9 patients (88%) in anaemic group while in non-anaemic group there were 23 /51

patients (45%) cases with positive reaction(χ^2).

4- Mean (ALT) level was lower in anaemic group (5 U/L) than the nonanaemic group (7.65 U/L) (Z-test two mean).

There is a difference between mean ESR in patients with the normal fibrinogen level 39/60 (65%) (49.9 mm/ h), and in patients with hypofibrinogenaemia 21/60 (35%) (37.6 mm /h) (Z-test two mean). There is difference in mean PCV (33.2 %) in patients with positive D-dimer reaction 31/60 (51.6%) compared to patients with negative D-dimer reaction 29/60 (48.4%) here the mean PCV was (36.2 %)(Z-test two mean). The patients with shortened APTT 36/60 (60%) (less than 30 seconds) have mean PCV of 33.4 % while patients without this shortening 24/60 (40%) have mean PCV of 36.5% (Z-test two mean). Percentage of severe cases in patients with and without history of PE The first group had 14/17 (82%) of severe cases while the second group had 9/21 (42%) of severe cases (χ^2), primigravida were excluded.

Mild PE 28/60 (47%) had a lower mean s.uric acid (6.0 mg/dl) while severe PE 32/60 (53%) had a higher mean s.uric acid (7.11mg/dl) (Z-test two mean). There is a positive correlation between PCV and APTT (R=0.5). There is a positive correlation between the s.urea & the s.uric acid (R=0.6). Patients with both shortened APTT& positive D- dimer reaction were in mild PE group 14/28(50%), while in severe PE group, they were 8/32 (25%) (χ^2).

Discussion

Anaemia was found in 9/60 (15%) of our studied patients. No significant changes were noted concerning the white blood cells in our study. Thrombocytopenia was observed in 3/60 patients (5%), 2 of them had HELLP syndrome. So, thrombocytopenia was less frequent than other haemostatic anomalies. 55/60 (91.7%) of our patients were with normal platelet count. This agrees with other similar study [9]. Patients with hypofibrinogenaemia had lower mean ESR than the patients with normal fibrinogen level. This is supported by the fact that defibrinated blood has lower ESR values than normal blood [4].

Haemostasis:

The following abnormal haemostatic values were more frequently observed towards lower PCV, anaemia , 1+ proteinuria , lower systolic and diastolic blood pressure and mild form of PE (no similar observation is encountered in other studies). The duration of illness could be the real explanation for that as severe PE had earlier termination of pregnancy while mild PE had later termination of pregnancy. So, there is more time for pathological process to act in mild PE than in severe PE. Generally speaking, severe PE is an indication of earlier termination of the pregnancy [5,8].

Bleeding time, platelet count, PT and APTT:

Bleeding time is prolonged in 1/60 (1.6%) of our patients. Accordingly prolonged bleeding time is not a common haemostatic abnormality in PE. Cases with borderline bleeding time were more frequent in anaemic PE than the non anaemic PE. This supports the fact that haemostatic abnormalities were more frequent in anaemic group.

Thrombocytopenia was not a common finding in our study, as we discussed earlier. In chronic disseminated intravascular coagulopathy (DIC), platelet count is usually within normal limits or borderline but most patients developed decrease platelet survival and increased turnover of platelets resulting from platelet consumption, this is often referred to as a compensated state [9].

From the onset of pregnancy there is a hypercoagulable state reflected as shortened PT and APTT [10]. This hypercoagubility might be explained by the increase in several procoagulant factors (I, VII, VIII and X) [5] and Von Willebrand factor antigen [11] . PE is an augmentation of the hypercoagulable state accompanying normal pregnancy [5] especially if we know that in PE there is reduction in antithrombin III and enhanced inactivation of protein C [8, 12] . So shortened PT and APTT are expected to be more pronounced. The following were present in PE; increased VIII, vWF, fibrinogen, increased blood viscosity [13] ,platelet activation [5] .They may all lead to acquired thrombotic tendency by producing

hypercoagulibity and all may cause shortening of PT and APTT [4] . Thrombosis leads to placental insufficiency which is one of the most frequent causes of intrauterine growth retardation [8] . Hence, thrombosis is expected to be encountered in our study specially if we know that one of the main haemostatic problems in pregnancy resulting from the hypercoagulable state is thrombosis [5] .It is now quite clear why some authors call the hypercoagulable state as prothrombotic state [14] , and why PE is a risk factor for venous thrombosis [15] , so the role of the anticoagulant therapy in PE is clear [5] .

Prolonged PT and APTT are features of acute rather than chronic DIC, as PT and APTT depend on the ultimate conversion of fibrinogen to fibrin. In DIC there is usually hypofibrinogenaemia, PT and APTT are prolonged when fibrinogen level is equal or less than 1g/L [9]. In our studied patients, the minimum plasma fibrinogen level was 2g/L and that is why PT and APTT prolongation were not encountered in the majority of our studied patients.

Shortened APTT was due to the activation of coagulation system in very early stage of DIC [16, 17] or as a reflection of the hypercoagulable state of pregnancy [10]. Mean APTT in anaemic PE group was shorter than the non anaemic group. Patients with shortened APTT had lesser mean PCV than the patients without this shortening. This could be due to the fact that the activation of coagulation system was more in anaemic group than nonanaemic group. Anaemic group had 78% mild cases; nonanaemic group had 42% mild cases as explained earlier.

A positive correlation was observed (R=0.5) between APTT and PCV, the higher PCV the longer APTT and the vice versa. The percentage of cases with shortened APTT in patients who had diastolic blood pressure less than 110 mmHg was higher than that of patients with diastolic blood pressure equal or more than 110 mmHg , and as 70% of the patients with diastolic blood pressure less than 110 mmHg were mild cases, the duration of illness might explain these changes.

Mean APTT in patients with 1+ proteinuria was 24.86 sec., while patients with 3+ proteinuria had mean APTT of 29.23 sec. As patients with 1+ proteinuria had 65% mild disease while the patients with 3+ proteinuria all had severe PE, this difference might also be related to the duration of the illness. Fourteen out of twenty eight (50%) of mild PE cases had both shortened APTT and positive D-dimer reaction, whereas in severe PE the result was 8/32 (25%). So activation of the coagulation system was more evident in mild PE .

D- dimer reaction:

The most common haemostatic abnormality in this study after shortened APTT, was the positive D-dimer reaction. This is supported by other similar studies example ([18] , and is quite expected as D-dimer increases in thrombotic disorders [17] . The appearance of positive D-dimer reaction is prior to other clinical and laboratory indicators of coagulopathy and indeed under circumstances in which the other coagulation studies are normal. The presence of positive D-dimer reaction may reflect either acute low grade DIC or chronic compensated DIC [5, 20].

The percentages of D-dimer positive cases in anaemic PE were more than the non anaemic patients. The mean PCV in D-dimer positive group was lower than D-dimer negative group, this support our initial findings.

Negative plasma D-dimer reaction was found in 7/21(33.3%) patients with hypofibrinogenaemia in the present study. This agrees with another study by AL-Ubadye [21] as (37.5 %) of hypofibrinogenic cases had negative D-dimer reaction.

Liver impairment as a cause of hypofibrinogenaemia without positive D-dimer reaction was not accepted in our study as we have 6/60 (10%) patients with liver impairment, 3 of them with positive D-dimer reaction and the other 3 were with normal plasma fibrinogen level.

Renal function:

There was a renal impairment in more than 50% of our studied patients. Createnine clearance (ml/min) was needed to solve this problem but it was not available, however the following equation might be helpful (not

valid with severe renal insufficiency, serum createnine > 5 mg/dl) but it was not applied because body weight was not available. For women, the value should be reduced to 85 percent of that estimated by this equation [22]:

Createnine clearance = (140-Age)xBody weight (Kg)/ 72 x patients createnine (mg/dl)

Renal affection was present in all the studied patients as proteinuria was part of our criteria [23] , after the exclusion of urinary tract infection by general urine examination and negative history of renal problems.

Mean s.uric acid was higher in severe PE (7.11mg/dl) than in mild PE (6.00 mg/dl) . Accordingly s.uric acid is related to the degree of severity. Moreover, s.uric acid is a good indicator of the detection of hypertensive states of pregnancy [24]. However, Odendaal (1997) [25] stated that there was no evidence of association between perinatal deaths and higher s.uric acid levels in patients with severe PE.

Mean s.uric acid was higher in patients with 2+ proteinuria (7.35 mg/dl) than the patients with 1+ proteinuria (5.93 mg/dl) . This gives a clue to the relation between the two parameters as both are indicators of renal function .Comparing mean s.uric acid in patients with 3+ proteinuria and patients with 1+or 2+ poteinuria, no significant correlation was found probably due to the small number of patients with 3+ proteinuria.

Hassan et al,1991 [26] stated that s.urea and s.uric acid were higher in preeclamptic patients than normal pregnancies. In our study, there was a strong positive correlation between s.urea and s.uric acid (R=0.6) .This reflects that the association between these two parameters is more than that between other parameters, as a reflection of prerenal impairment, as there is decrease in glomerular filtration rate in preeclamptic patients .

It was normal in 54/60 (90%) patients of the studied group . So liver impairment in PE is not common. Our initial differential diagnosis included viral hepatitis, toxic hepatitis, acute fatty liver of pregnancy, the Budd-Chiari syndrome, and liver injury due to PE. In a similar study no common aetiologic factors other than pregnancy could be found [27].

HELLP syndrome was encountered in our study in 2/60(3.3%) patients according to our criteria (thrombocytopenia less than 100 $x10^9$/L, S. alanine transaminase greater than 22 U/L, s.aspartate transaminase greater than 18 U/L, microangiopathic haemolysis - presence of schistocytes, burr cell and / or polychromasia) [28]. So it is not a common complication of PE. This is supported by Campbell (2000) [29] who stated that HELLP syndrome incidence was 2-4%, further more, in a study by Haggi, Al Hayali 1996 [30] performed in Mosul, 1/50 (2 %) patients with PE had HELLP syndrome according to our criteria.

Case No.14 was an example of (class I-HELLP syndrome), note how the haemostatic parameters were worse in this case compared with case No. 19 (class II-HELLP syndrome) . This finding is supported by Martin et al, 1999 [31] who stated that class I HELLP syndrome showed more severe abnormal haemostatic laboratory findings compared to classes II and III- HELLP syndrome. Concerning the outcome case number 14 was discharged on her responsibility, case number 19 was delivered normally.

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Table 1: Haemostatic findings of the studied patients

Bleeding time	*No (%)*		*Platelet count*	*No (%)*	
Normal	**Borderline**	**Prolonged**	*Low*	*Normal*	*Increased*
42/60 (70.0)	17/60 (28.4)	1/60 (1.6)	3/60 (5.0)	55/60 (91.7)	2/60 (3.3)

PT	*No (%)*	*APTT*		*No (%)*
Shortened	*Normal*	*Shortened*	*Normal*	*Prolonged*
6/60 (10.0)	54/60 (90.0)	36/60 (60.0)	23/60 (38.4)	1/60 (1.6)

Fibrinogen	*No (%)*	*D-dimer reaction*	*No (%)*
Low	*Normal*	*Positive*	*Negative*
21/60 (35.0)	39/60 (65.0)	31/60 (51.6)	29/60 (48.4)

Table 2: Renal function tests and s.uric acid of the studied patients.

S.urea No (%)		*S.createnine No (%)*		*S.uric acid No (%)*	
Normal	*Elevated*	*Normal*	*Elevated*	*Normal*	*Elevated*
28/60 (46.6)	32/60 (53.4)	13/60 (21.6)	47/60 (78.4)	29/60 (48.4)	31/60 (51.6)

Table 3: Severe PE, mean APTT and mean s.uric acid in different grades of proteinuria.

Parameter	*Case No.14*	*Case No.19*	*Parameter*	*Case No.14*	*Case No.19*
Age (year)	20	38	Dyspnea	-	-
Residence	Mosul	Mosul	Previous cesarean section	-	-
Para	0	6	History of PE	-	+
Gravida	1	7	Family history of PE	-	-
Gestational age (wk)	25	35	Family history of hypertension	-	-
Chief complaint and duration	Headache, 1 wk	Headache & hypertension ,1 month	Oedema	-	ankle
Visual disturbances	-	-	Pallor	-	-
Ankle oedema	-	+	Blood pressure mmHg	140/90	230/120
Right hypochondrial pain	-	-	Ultrasound Gestational age	23 wks+2 days	31 wks

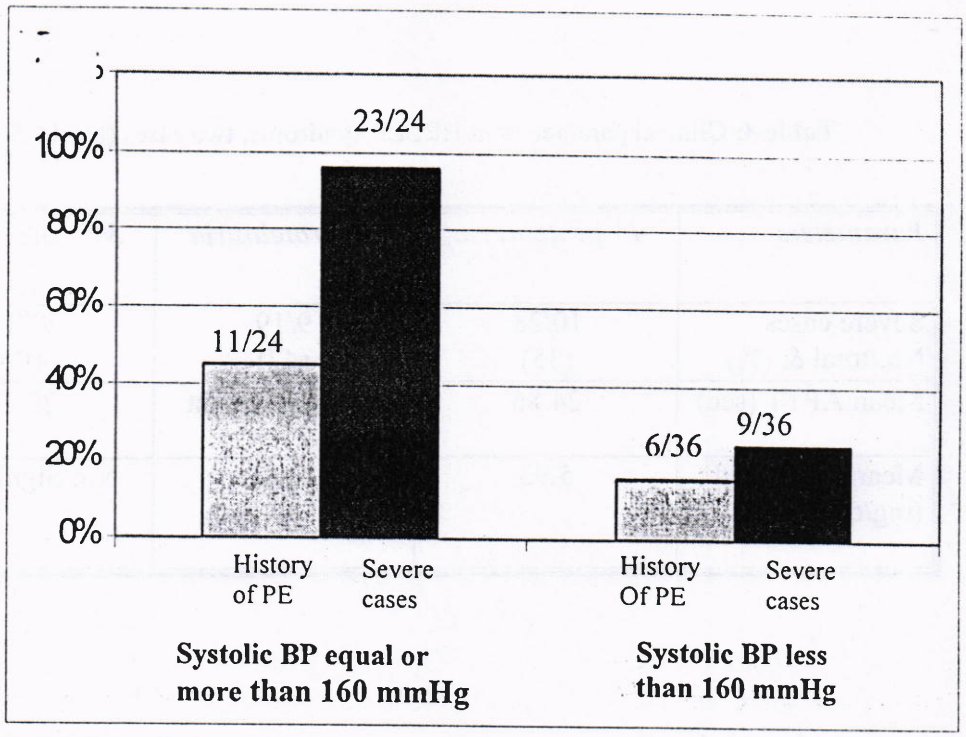
P<0.05

Table 4: Clinical parameters in HELLP syndrome, two cases (No.14 & No.19).

Parameters	*1+ proteinuria*	*2+ proteinuria*	*3+ proteiuria*
Severe cases No./total & (%)	10/28 (35)	9/19 (47)	13/13 (100)
Mean APTT (sec)	24.86	Not Significant	29.23
Mean s.uric acid (mg/dl)	5.93	7.35	Not Significant

Table 5: Investigations of HELLP syndrome, two cases (No. 14& No.19).

Parameter	*Case No.14*	*Case No.19*	*parameter*	*Case No.14*	*Case No.19*
Hb (g/dl)	11	16	BT (min)	10	10
PCV(%)	31	43	PT (sec)	11	12
RET(%)	5	5.3	APTT (sec)	30	36
T. WBC (x 10^9/L)	9	10	Fibrinogen (g/L)	2	4.5
N%	77	88	D-dimer	+	-
L %	19	10	S.Urea (mg/dl)	38	33
M%	2	1	S.Createnine (mg/dl)	1	0.9
E%	1	1	TSB (mg/dl)	1.4	1.7
B %	1	0	DSB (mg/dl)	0.4	0.6
Platelet count (x10^9/L)	20	60	IDSB (mg/dl)	1	1.1
Red cell morphology	Normochromic normocytic with fragmentation	Normochromic normocytic with fragmentation	ALT (U/L)	32	36
Platelet in film	Markedly Reduced	Mildly Reduced	AST (U/L)	24	40
ESR (mm/h)	40	23	ALP (KAU/dl)	10	28
Class	I	II	Proteinuria	2+	3+



P<0.05

Figure 1: Systolic blood pressure & its relation to number of patients with history of PE, number of severe cases .

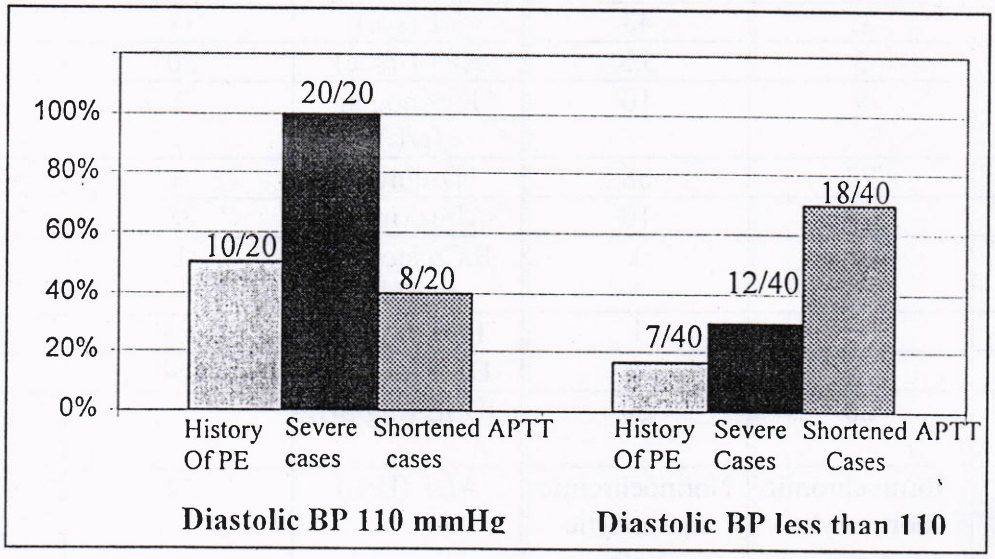


Figure 2: Diastolic blood pressure & its relation to number of cases with history of PE, number of severe cases , number of shortened APTT cases.