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Review

Priapism in Patients with Chronic Myeloid Leukemia (CML): A Systematic Review

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Summary. *Background:* Priapism is defined as a penile erection that persists four or more hours and is unrelated to sexual stimulation. Priapism resulting from hematologic malignancy is most likely caused by venous obstruction from microemboli/thrombi and hyperviscosity caused by the increased number of circulating leukocytes in mature and immature forms. In patients with leukemia, 50% of cases of priapism are due to Chronic Myeloid Leukemia (CML). We present a systematic review of priapism in CML. *Acquisition of evidence:* An extensive literature research was carried out in PubMed, Google Scholar, SCOPUS, and Science Citation Index databases. The search included cases up to 4th August 2020. *Synthesis of evidence:* A total of 68 articles were found and included in our review, including 3 reviews from three different centers. We found 68 articles (102 patients; figure 1) and several case reports on priapism in CML. Priapism was noticed in some patients at the first presentation of CML. However, it was infrequently reported during the start of treatment, following the stop of medication and post-splenectomy. The mean age at presentation was 27.4 years, and the mean time from onset of priapism to the time to get medical attention (presentation) was 78.2 hours. The mean white blood cell count associated with priapism was 321.29x10^9/L, and the mean platelet count was 569 x10 ^{9}/L. The chronic phase of CML was the most common phase where priapism occurred. Most patients were Asian (>50%). Nearly a quarter of patients (27.4%) developed permanent erectile dysfunction. *Conclusions:* Priapism is a urological emergency requiring urgent multidisciplinary management to prevent erectile dysfunction. Because of the relatively rare occurrence of priapism in CML patients, there is no standard treatment protocol. (www.actabiomedica.it)

Keywords: Chronic myeloid leukemia, Chronic granulocytic leukemia, Erectile dysfunction, Priapism, Male fertility.

Introduction

Hematological disorders are the leading cause of priapism, accounting for 20% of the cases and include SCD, hyperviscosity syndromes as seen with the myeloproliferative diseases, hypercoagulable states such as deficiencies of proteins C and S, antiphospholipid syndromes, And amyloidosis (1). Priapism is defined as a penile erection that persists four or more hours and is unrelated to sexual stimulation (2). The condition is classified into three subtypes: ischemic (low-flow), non-ischemic (high-flow), and stuttering (intermittent) priapism (3). Stuttering priapism is characterized by a recurrent and intermittent erection, frequently occurring in a specific patient population with SCD and less commonly with thalassemia (4). The clinical presentation of CML consists of lymphadenopathy (80%), asthenia, and fatigue (60%), spleen or liver enlargement (50%), weight loss (15–20%), and bleeding (10%), hyperleukocyto-

sis about 80%, central nervous system affection (15%) kidney (5%) and priapism (≤ 3%) (5). Priapism due to hematological disorder is most likely due to venous obstruction from microemboli/thrombi as well as hyperviscosity due to an increased number of circulating leukocytes n mature and immature forms. Other accessory mechanisms are venous congestion of the corpora cavernosa secondary to mechanical pressure from the abdominal veins draining the spleen or infiltration of the sacral nerves or the central nervous system by leukemia cells (6). It is also seen that increased production of cytokines and adhesion molecules by leukemia cells result in endothelial cell activation and lead to increased sequestration of cells in the microvasculature (7).

Prolonged corporeal ischemia lasting more than 24 to 48 hours may result in varying extents Of irreversible fibrosis with endothelial and trabecula destruction of the erectile tissue and Subsequently in permanent erectile dysfunction and, therefore, is considered as a urologic Emergency. Reduced sperm count related to TKIS in patients with CML as well as priapism adversely affects the quality of life, particularly in populations where adolescent and young adults represent the majority of patients (8,9).

The objectives of this review were to: (a) assess the characteristics and risk factors of CML patients with priapism, (b) realize the common type of priapism in CML, (c) describe the management options adopted for priapism in CML, and (d) investigate the outcome and erectile dysfunction.

Acquisition of evidence

We searched the English literature (Google Scholar, PubMed, SCOPUS, and Science Citation Index databases) including original articles, reviews, case series, and case reports using the terms: "chronic myeloid leukemia," "chronic myelogenous leukemia", "chronic myelocytic leukemia" and "priapism". A total of 68 articles were found and included in our review, among them 3 reviews were found from three different centers. The search included cases up to 4th August 2020.

Synthesis of evidence

We found 68 articles on priapism in CML (102 patients; figure 1) and several case reports (10-77). The youngest patient was 7 weeks old, and the oldest was 60 years old. The first case was reported in 1960 and the last in 2020. Most patients had priapism at their first presentation of CML. Three patients developed priapism after starting CML treatment, two after stopping treatment, and two were previously diagnosed. 80 /102 patients had splenomegaly (on clinical examination or by US abdomen), and 31/102 had hepatomegaly. The lowest white blood cell count (WBC) associated with priapism was 37 x10 ^{9}/L, and the highest was 782 x10 ^{9}/L.

Table-1 presents the characteristics of stuttering and ischemic priapism in patients with CML.

Most patients had lower hemoglobin (Hb) levels, and in two reviews, the CML patients with priapism had lower Hb levels than their matched CML patients who didn't have priapism.

Treatment modalities (Table 2) included medications, aspiration, and irrigation to the corpora cavernosa, radiotherapy, leukoreduction, and surgical shunts. Medications were used in 59 CML patients, aspiration of the corpora cavernosa in 49 patients, leukapheresis in 19 patients, radiotherapy in 9 patients, and shunt in 40 patients.

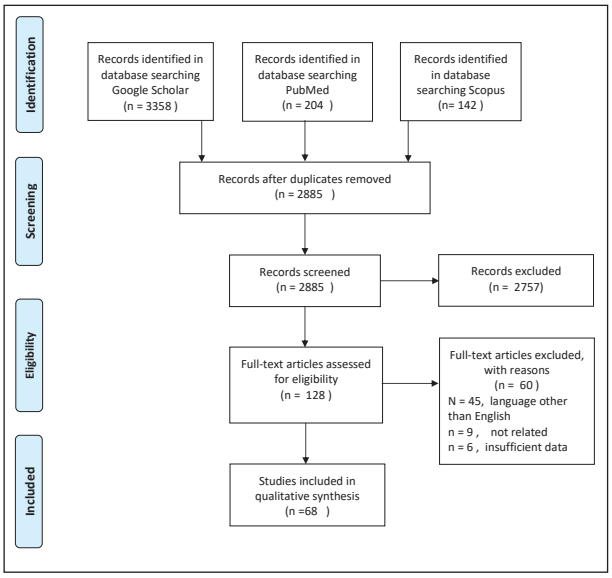

Figure 1. The PRISMA flow diagram detailing the cases of chronic myeloid leukemia (CML) presenting priapism.

Discussion

CML is a myeloproliferative neoplasm (MPN) characterized by the uncontrolled production of mature granulocytes. The three stages of the CML are the blast phase, accelerated phase, and chronic phase. Most of the patients are diagnosed incidentally with an elevated white blood cell count on the CBC during their chronic asymptomatic period. Unfortunately, in most cases, the diagnosis of CML is reached late, as it has a large variety of vague clinical manifestations that may include lymphadenopathy, fatigue, hepatosplenomegaly, weight loss, bleeding tendency, and thromboembolic phenomena due to hyperleukostasis (78).

The mechanism of priapism in leukemia is believed to be related to blood sludging with white blood cells (6, 79). Severe anemia implies tissue hypoxia, which may interfere with the NO cGMP balance and precipitate the occlusion. Repeated priapism (stuttering) episodes can lead to prolonged ischemia and tissue damage (80). Most patients with priapism had lower Hb levels compared to their matched CML patients without priapism (78,81).

Although anemia may be an essential factor in priapism's pathogenesis, it is not clear if blood transfusion is useful to alleviate an acute priapic attack, or it may worsen the condition. Blood exchange transfusion for treating patients with SCD and major priapism has been shown to be efficacious and safe.

WHO defines CML's blast phase as more than 20% blasts (large cells) in bone marrow or peripheral smear (82). Although this could lead to more stasis, unexpectedly, priapism is not more common in the blast crisis phase and accelerated phase (only 5/102 patients). The majority of priapism cases (n = 97/102) occurred during the chronic phase.

In the era before the introduction of tyrosine kinase inhibitors, the chronic phase of CML accounted for 85% of CML presentation at the time of diagnosis (5). Most patients were below the age of 40 years, with a mean of 27.4 years. This means that it is more common in younger patients with CML patients. The peak age of priapism in adults (without CML) is between 20-50 years (83).

Thrombocytosis, with platelets (PLT) count above 600 x10 ^{9}/L is seen up to 30% of CML patients (5,84). The high mean PLT count may have some impact on the occurrence of priapism in CML and/or may influence the type of priapism (stuttering versus ischemic) rather than its occurrence. In CML patients with stuttering type priapism, the PLT count was lower (506.8 x10 ^{9}/L) compared to those with the ischemic type (609 x10^{9}/L). Moreover, there was no significant difference in the PLT count between CML patients with and without priapism (78).

In essential thrombocythemia (ET), another form of MPN with extremely high PLT counts, priapism was much less reported compared to CML (85). The few reports might reflect the minimal role of PLT in occluding the penile circulation compared to WBC.

The enlarged spleen indicates an advanced CML stage, which supports the late presentation. Moreover, splenomegaly is of prognostic importance; massive splenomegaly indicates poor prognosis and increased risk of dying due to CML (86). In this review, splenomegaly was reported in 80/102 (78.4%) patients and hepatomegaly in 31 patients, but organomegaly was not addressed in 20 patients (Table-3). Splenomegaly was seen in 28/31 (90%) with the stuttering type and in 50/70 (73.2%) with the ischemic type.

Ethnicity might have a role in the predisposition to priapism as 57% of the CML patients with priapism were Asian. Most of these reports came from India.

Table 3. Characteristics of stuttering and ischemic priapism in patients with chronic myeloid leukemia (CML)

Priapism type	Stuttering (n=32)	Ischemic (n=70)
Mean time to presentation	220.9 hours (n. 20)	77.8 hours
Mean Age (year)	23.78	27.93
Mean WBC	314.9 x10^{9}/L	320.49 x10^{9}/L
Mean PLT	506.8 x10^{9} /L	609 x10^{9}/L
Mean Hb	9.6 g/dl	8.78 g/dl
CML phases	All chronic phase	1 accelerated phase 4 blast phase 67 chronic phase
Erectile dysfunction	16 not addressed 13 had erectile dysfunction 3 no erectile dysfunction	41 not addressed 16 had erectile dysfunction 14 no erectile dysfunction
Splenomegaly (n)	29	51
Hepatomegaly (n)	9	22

This could be due to genetic susceptibility or difficulty accessing health care for the nonspecific symptoms of CML until WBC reached high levels, causing vascular stasis and priapism. Also, priapism occurred in patients with a problem with compliance or stopped medications (30, 46,76). Surprisingly, priapism developed after starting cytoreductive therapy in 3 patients (32, 49, 50). It is hard to conclude that initiating cytoreductive therapy will raise the risk of priapism.

Priapism is a urological emergency, which must be treated early to prevent erectile dysfunction. It is predicted that if priapism lasts more than 24 hours, the risk of permanent erectile dysfunction is more than 90% (87). Therefore, a rapid and expert reversal of the priapism is highly required. The mean time that a CML patient with priapism sought medical advice was 78.28 hours (n=76), which carried a high risk for developing permanent erectile dysfunction (88). However, despite this delayed presentation to medical attention, the reported erectile dysfunction was not high. The erectile dysfunction after the episode(s) of priapism in CML patients was reported in 29/102 (28.15%) and did not occur in 17/28 (60.7%). Probably, the lack of information and methods used to assess the erectile function in CML patients may explain the low reported percentage of erectile dysfunction in CML patients.

Over the past decade, we have witnessed significant advances in knowledge of CML's biology and treatment. Imatinib is a first-line tyrosine kinase inhibitor for treating CML and has dramatically improved the prognosis of this disease. Chang et al. (89) have shown that Imatinib crosses the blood-testis barrier and reduces sperm density, sperm count, sperm survival rates, and sperm activity in CML patients in the chronic phase. But did not affect the structure of reproductive organs or sex hormone levels.

Forty patients needed a surgical shunt to relieve priapism, 5 of them had a partial response and continued chemotherapy to control the priapism. Forty-nine patients responded to aspiration and irrigation, and 2 of them required chemotherapy to control priapism due to incomplete response. Irradiation to the penis and spleen was used in 9 patients; leukapheresis was used in 19 patients, 6 of them required surgical shunts (43, 51, 63, 66).

Medications alone were used to treat priapism in 14 patients. However, reversing priapism using medications required longer duration compared to other modalities (mean duration in 7 patients was 14.4 days). Medications used included: hydroxyurea (n= 48), cyclophosphamide (n=3), busulfan (n=15), terbutalin (n=1), prednisolone (n=2), vincristine (n=1), priscoline hydrochloride (n=1), hyaluronidase injection (n=1), imatinib (n=5), cytarabine (n=1), diethylstilbestrol (n=2), cytarabine (n=2), low-molecular-weight heparin (n=3), anticoagulation (n=2), opioids (n= 1), blood transfusions (n=2).

The response to systemic therapy alone (medication) is usually prolonged and may represent the natural history of ischemic priapism rather than the effect of the medications (90). The American Urology Association (90) recommended an early treatment in a step-wise fashion starting with therapeutic aspiration (with or without irrigation) or intra-cavernous injection of sympathomimetics.

For CML patients, two points shall be considered. First, the late presentation to medical attention, which means that the slowly acting medications are less likely to be effective alone. In contrast, urological aspiration and irrigation within the first 24 hours decrease the risk of erectile dysfunction (91). Therefore, it is reasonable to start with aspiration and irrigation not to rely on oral medication for the treatment of CML alone. Secondly, treating the underlying mechanism of increased WBC count is needed to control and prevent priapism recurrence. Leukapheresis was used as a modality to treat both the high WBC of CML and priapism. The pitfalls of leukapheresis are that it is not available everywhere, is costly, and may require several sessions before a significant reduction in WBC count, which may take days. Besides, there is no clear-cut WBC value below which priapism is anticipated to be controlled. The mean WBC count after which priapism was controlled, is 22 x10 ^{9}/L. However, other patients were controlled only with WBC count between 3-10 x10 ^{9}/L. It remains an option for patients who failed aspiration and refused surgical shunt.

Similarly, the effects of irradiation were not rapid (6 days, 2 weeks, and 19 days), as documented in a few patients (45,58,69).

Four major types of surgical shunts are used for

the treatment of priapism. These include percutaneous distal shunts, open distal shunts, open proximal shunts, and vein anastomoses/shunts (92). The goal of surgery is to create a channel or fistula that allows the deoxygenated blood to drain from the corpora cavernosa. For all shunt procedures, the patient should receive preventive perioperative antibiotics.

Guidelines advocate for an aggressive approach in treating patients with refractory priapism by proceeding in a serial fashion from distal to proximal shunts to vein shunting as quickly and safely as possible to achieve penile flaccidity (92).

A delayed penile implant was used in one patient. It is generally used for patients who developed erectile dysfunction; however, it can be used acutely to control priapism and prevent fibrous tissue formation (93). The erectile dysfunction occurs more frequently following proximal or vein shunts compared to the distal shunts. However, it is difficult to attribute the erectile dysfunction to the shunt operation as patients had received different modalities of treatment and had different duration before seeking medical attention (89).

Conclusion

Priapism is a rare complication of CML. It is mostly seen at the first presentation of the disease and much less during the start of treatment. It may occur after stopping the medication or post-splenectomy. Late presentation negatively affects the response to treatment as well as erectile function. Therefore, physicians must interfere early and follow a timely plan and shall follow the patients closely for developing erectile dysfunction. Conservative and medical therapy without urological intervention is less likely to be sufficient. Starting treatment of CML to decrease the high WBC count might accelerate the resolution of the priapism and sometimes is needed for a complete resolution.

Conflicts of interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

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Table 1. shows the characteristics of CML patients with priapism

Referenced	Age in Years	Priapism from onset to presentation to the hospital	First presentation or previously	Type of priapism	WBC x10^9/L	PLT x10^9/L	HB g/dL	Splenomegaly/ hepatomegally, below costal margin in cm or if presdnt	stage of CML
10	18	72 hours	Yes	ischemic low flow	100	1,002	6	spleen 2–3 cm	chronic phase
11	15	2 days	Yes	ischemic low flow	135	197	9	spleen to the umbilicus	chronic phase
12	52	4 hours	Yes	ischemic low flow	239	625	8.9	spleen 8cm liver 3 cm	chronic phase
13	19	18 hours	Yes	ischemic low flow	513	NA	NA	N/A	chronic phase
14	38	30 hours	Yes	ischemic low flow	378	155	10.5	spleen 18 cm	chronic phase
15	52	6 days	Yes	ischemic low flow	282	368	10	N/A	chronic phase
16	21	72 hours	Yes	ischemic low flow	619	N/A	7.4	splenomegally	chronic phase
17	30	9 days	Yes	ischemic low flow	261	86	9.2	spleen 10 cm	chronic phase
18	29	3 days	Yes	ischemic low flow	366.34	622	6.7	spleen 5 cm, liver 2 cm	chronic phase
19	20	4 hours	Yes	ischemic low flow	158	N/A	10.9	spleen 8 cm	chronic phase
20	18	14 hours	Yes	ischemic low flow	363	527	9.7	spleen 9 cm	accerated
21	55	2 days	Yes	ischemic low flow	420	280	9.5	spleen up to umbilicus , liver 2 cm	chronic phase
22	17	NA	Yes	ischemic low flow	377,31	730	11.3	Hepatosplenomegaly Blasts+ Promyelocytes-12%	chronic phase
23	21	19 hours	Yes	ischemic low flow	216	1746	8.3	spleen 7 cm, liver 6 cm	chronic phase
24	28	2 days	Yes	ischemic low flow	294	94	6.6	spleen 2	chronic phase
25	22	4 days	Yes	ischemic low flow	218.6	324,2	8.2	spleen 6	chronic phase
26	21	6 days	Yes	ischemic low flow	410	N/A	N/A	spleen 3 fingers	chronic phase
27	18	75 hours	Yes	ischemic low flow	323	N/A	N/A	N/A	chronic phase
28	18	7 days	Yes	ischemic low flow	257	5450	N/A	spleen 2–3	chronic phase
29	18	4 days	Yes	ischemic low flow	144	350	9.6	spleen 9-10 cm liver 2-3 cm	chronic phase
30	30	16 hous	after stopping	ischemic low flow	210	45	10.3	huge splenomegaly	chronic phase
31	18	5 days	Yes	ischemic low flow	215	470	6.9	spleen 10 cm	chronic phase
32	18	10 days	after start of treatment	ischemic low flow	320	normal	6.5	hepato-splenomegaly.	blast crisi
33	21	8 hours	Yes	ischemic low flow	316	670	8.3	liver palpable 6 cm , spleen 7 cm	chronic phase
33	55	12 hours	Yes	ischemic low flow	282	260	9	spleen 7 cm	chronic phase
34	36	5 days	diagnosed at 33	ischemic low flow	N/A	N/A	N/A	N/A	chronic phase
25	33	22 hours	Yes	ischemic low flow	400	1 200	10.53	spleen 4 cm	chronic phase
36	16	11 days	Yes	ischemic low flow	614.8	907	5.7	liver 2cm spleen 4cm	blast crisis
37	30	20 hours	Yes	ischemic low flow	285	462	11.2	spleen 2cm liver 3 cms	chronic phase
37	25	2 days	Yes	ischemic low flow	670	320	11.3	spleen 1 cms	chronic phase
37	28	7 hours	Yes	ischemic low flow	441	422	7	spleen 15 cms	chronic phase
38	14	24 hours	Yes	ischemic low flow	226.9	310	9.9	spleen 6 cm	chronic phase
39	16	NA	Yes	ischemic low flow	320	417	11	spleen 4 cm	chronic phase
40	25	16 hour	Yes	ischemic low flow	501 570	269	10.3	no organomegally	chronic phase
41	9	9 hours	Yes	ischemic low flow	274	1235	8.2	splenomegally	blast crisis
42	55	8 hours	Yes	ischemic low flow	184	277	9	spleen 3 cm liver 2 cm	chronic phase
43	23	3 days	Yes	ischemic low flow	660	321	8	hepatosplenomegaly	chronic phase
44	47	5 days	Yes	ischemic low flow	297	N/A	N/A	hepatosplenomegaly	chronic phase
44	42	7 days	Yes	ischemic low flow	390	N/A	N/A	spleen 6 cm	chronic phase
44	28	6 days	Yes	ischemic low flow	206	N/A	N/A	no hepatosplenomegally	chronic phase
45	7 weeks	5 days	Yes	ischemic low flow	37	344	10	liver and spleen were palpated below the costal margin.	blast crisis

46	22	8 hours	Known CML, stopped medications	ischemic low flow	157	670	5.4	hepato-splenomegaly	chronic phase
47	43	48 hours	NA?	ischemic low flow	N/A	N/A	N/A	N/A	blast crisis
48	37	96 hours	Yes	ischemic low flow	150	215	13.9	N/A	chronic phase
49	22	N/A	after day 4 of treatment	ischemic low flow	297	N/A	N/A	hepatosplenomegaly	chronic phase
50	29	deveoped priapism after starting treatment busalfan and allopyranol in the 5th hospital admission	5th day	ischemic low flow	296	800	N/A	spleen 4 cm	chronic phase
51	46	duration after presentation 22 days	Yes	ischemic low flow	782	400	N/A	spleen 14 cm	chronic phase
51	42	nonsustained	Yes	ischemic low flow	367	179	N/A	spleen 9 cm	chronic phase
51	42	24 hr	Yes	N/A	402	500	N/A	spleen 10 cm	chronic phase
51	18	4 dasys	Yes	N/A	451	726	N/A	spleen 18cm	chronic phase
52	45 year	18 hrs	Yes	N/A	N/A	N/A	N/A	N/A	chronic phase
52	58	48 hrs	Yes	N/A	N/A	N/A	N/A	N/A	chronic phase
52	40	12 hrs	Yes	N/A	N/A	N/A	N/A	N/A	chronic phase
52	38	29 hr	Yes	N/A	N/A	N/A	N/A	N/A	chronic phase
52	60	6 hrs	Previously diagnosed	N/A	N/A	N/A	N/A	N/A	chronic phase
52	39	72 hrs	Yes	N/A	N/A	N/A	N/A	N/A	chronic phase
53	9	3 Days	Yes	N/A	155	N/A	N/A	N/A	chronic phase
53	11	5 days	Yes	N/A	210	N/A	N/A	N/A	chronic phase
53	13	4 days	Yes	N/A	120	N/A	N/A	N/A	chronic phase
53	10	3 days	Yes	N/A	200	N/A	N/A	N/A	chronic phase
53	8	2 days	Yes	N/A	170	N/A	N/A	N/A	chronic phase
54	22	6 days	Yes	N/A	580	N/A	7.2	spleen 22 cm, liver 10 cm	chronic phase
54	30	8 days	Yes	N/A	300	N/A	6	hepatosplenomegally	chronic phase
55	28	11 days	Yes	N/A	400	adequate	11.75	spleen 13 cm	chronic phase
56	26	N/A	Yes	ischemic low flow	365	625	N/A	spleen 17 cm liver 4 cm	chronic phase
57	36	N/A	Yes	ischemic low flow	231	113	12	hepatosplenomegaly by US	chronic phase
58	8	1 day	Yes	ischemic low flow	430	200	9	liver 1 cm, spleen down to the left inguinal ligament	chronic phase
59	12	3 days	Yes	ischemic low flow	172	N/A	N/A	splenomegally	chronic phase
60	45	2 days	Yes	ischemic low flow	410	350	6	massive spleenomegally	chronic phase
61	22	5 days	Yes	iscemic low flow	200	increased	7.5	spleen 15 cm liver just palpable	chronic phase
62	12	2 days	Yes	stuttering - ischemic	460	350	8.2	spleen 22 cm and hepatomcgaly 4 cm.	chronic phase
63	24	5 days	Yes	stuttering - ischemic	207	545	10	spleen 8 cm	chronic phase
64	30	8 days	Yes	stuttering - ischemic	240	186	7.5	liver 1 cm spleen 6 cm	chronic phase
64	18	18 hour	Yes	stuttering - ischemic	288	388	8.7	liver 3 spleen 10	chronic phase
64	32	4 days	Yes	stuttering - ischemic	540	210	12.4	liver 3 spleen 15	chronic phase

65	24	14 hours	Yes	stuttering - ischemic	177.15	N/A	10.3	N/A	chronic phase
65	29	6 hours	Yes	stuttering - ischemic	402.24	N/A	8.2	N/A	chronic phase
56	60	2 weeks	splenectomy 16 months before	stuttering - ischemic	360	222	N/A	had splenomegally , splenectomy	chronic phase
66	13	3 days	Yes	stuttering - ischemic	350	450	8.5	spleen 4cm	chronic phase
67	24	prolonged	Yes	stuttering - ischemic	540	N/A	HCT 25%	spleen 7 cm	chronic phase
51	33	3-5 days	Yes	stuttering - ischemic	197	350	N/A	spleen 14 cm	chronic phase
51	27	NA	Yes	stuttering - ischemic	202	900	N/A	spleen 12cm	chronic phase
51	33	38 days	Yes	ischemic - stuttering	240	1150	N/A	spleen 19 cm	chronic phase
51	28	70 days	Yes	stuttering - ischemic	186	218	N/A	spleen 7 cm	chronic phase
51	28	24 hr	Yes	stuttering - ischemic	500	345	N/A	spleen 14 cm	chronic phase
68	11	12 hours	Yes	stuttering - ischemic	290	550	hematocrete 22%	spleen 4 cm, mild hepatomegally	chronic phase
51	23	duration after presentation 36 days	Yes	ischemic - stuttering	470	180	N/A	spleen 14	chronic phase
50	17	24 hours	Yes	stuttering - ischemic	290	230	hematocrit: 28 per cent;	massively enlarged spleen, occupying the entire left half of the abdominal cavity	chronic phase
69	7.5	few hours	Yes	stuttering ischemic	337	N/A	7	splenomegally	chronic phase
70	15	2 times resolved spontaneously	Yes	stuttering - ischemic	480	130	9.4	mildly enlarged on US	chronic phase
41	9	sveral days	Yes	stuttering - ischemic	509	1200	10.1	hepato-splenomegaly	chronic phase
41	9	4 days	Yes	stuttering - ischemic	169	663	10.3	splenomegally	chronic phase
71	36	34 hour	Yes	ischemic - stuttering	65	800	N/A	spleen 12cm	chronic phase
71	30	4 day	Yes	ischemic - stuttering	356.4	220	PCV. 22 L/L	liver 6cm, spleen 10cm	chronic phase
37	29	3 months duration	Yes	stuttering - ischemic	284	370	10.5	spleen 3 cm	chronic phase
72	18	12 days	Yes	stuttering - ischemic	199	504	HC 17	spleen 18 cm	chronic phase
37	26	3 days	Yes	stuttering - ischemic	292	490	8.9	spleen 15 cm	chronic phase
73	12	2 dyas	Yes	ischemic - stuttering	346	924	9	liver and spleen were enlarged, 5 cm	chronic phase
74	22	one month intermittent	Yes	stuttering-ischemic	185	N/A	10.7	splenomegaly on US	chronic phase
75	19	over 24 hours	Yes	stuttering ischemic	296	936	9.2	spleen 3 cm , liver 1-2	chronic phase
76	27	9 hours	diagnosed 19 y	stuttering - ischemic	450.01	509	11.4	splenomegally on US	chronic phase
77	18	6hours	Yes	ischemic stuttering	588	109	7.3	N/A	chronic phase

Table 2. Treament modalites of priapism and the outcome of erectile dysfunction

patient refernce	medications for priapism	Aspiration and irrigation	leukophresis	irradiation	shunt	Best responded to	Erectile dysfunction
10	Imatinib	No	Yes	No	No	leukapheresis	No
11	No	Yes	No	No	glanocorposal shunt, corporospongiosal shunt	shunt	N/A
12	No	Yes	No	No	Winter's shunt	Winter's shunt	No
13	No	Yes	Yes	No	No	leukapheresis	No
14	Yes	Yes	Yes	No	No	after leukophresis and medication	N/A
15	No	Yes	No	No	surgery penis shunts	surgery penis shunts	N/A
16	No	Yes	No	No	Winter's shunt	Winter's shunt partial response, hydroxyurea after combined complete response	Yes
17	Yes	Yes	No	No	bilateral T-shunts	afte shunt	Yes
18	No	No	No	No	Winter shunt	Winter shunt	N/A
19	No	Yes	No	No	No	cavernosa aspiration, epinephrine irrigation	No
20	No	Yes	No	No	No	cavernosa aspiration and irrigation with epinephrine	No
21	No	No	No	No	corpus cavernosa–glans shunt	corpus cavernosa–glans shunt	N/A
22	No	Yes	No	No	No	Aspiration following intra-cavernosal injection of phenylephrine.	N/A
23	No	Yes	No	No	No	cavernosa aspiration and epinephrine irrigation	No
24	hydroxyurea, allopurinol, Cytarabine	Yes	No	No	corporoglandular shunting	corporoglandular shunting	N/A
25	No	Yes	No	No	No	aspiration and irrigation of the corpora cavernosa	N/A

26	imitinib	Yes	Yes	No	No	Failed aspiration, Leukapheresis ended in, penile prosthesis,	Yes
27	allopurinol hydroxyurea	Yes	Yes	No	transglandular cavemosum-spongiosum shunt	transglandular cavemosum-spongiosum shunt	N/A
28	No	Yes	No	No	proximal corpora cavernosa-corpus spongiosum shunt	surgical proximal corpora cavernosa-corpus spongiosum shunt	No
29	No	No	Yes	Yes	No	improved after 48 hr leukapheresis procedure	N/A
30	No	Yes	No	No	No	aspiration and irrigation with ephedrine	Yes
31	hydroxyurea	No	Yes	No	No	hydroxyurea ,leukopheresis 5 sessions	N/A
32	hydroxyurea allopurinol	No	No	No	Winter's shunt	shunt, hydroxyurea, allopurinol failed	Yes
33	oral pentazocaine, Allopurinol Hydroxyurea, busulpha n	No	No	No	No	Allopurinol Hydroxyurea	N/A
33	No	Yes	No	No	No	cavernosa aspiration and epinephrine irrigation	No
34		Yes	No	No	No	aspiration of the corpora cavernosa	N/A
25	No	No	No	No	Proximal surgical shunt	Proximal surgical shunt was performed	Yes
36	No	Yes	No	No	No	cavernosa aspiration and epinephrine	N/A
37		Yes	No	No	winter shunt	Winter shunting	No
37	hydroxyurea	Yes	No	No	Al-Ghorab shunt	Aspiration Shunt	No

37	hydroxyurea	Yes	No	No	Al-Ghorab shunt	Aspiration Shunt	EHS2. Grade 1 developed ED
38	hydroxyurea	Yes	No	No	Al-Ghorab shunt	Aspiration Shunt	No
39	No	Yes	No	No	No	cavernosal aspiration, irrigation and phenylepinephrin partial response after 5 days improved	N/A
40	Hydroxiurea ARA-C	No	Yes	No	No	Hydroxiurea ARA-C / LeukapheresisOne session per day for 3d /LMWH	N/A
41	No	Yes	No	No	No	corporal aspiration	N/A
42	Cyclophosphamide LMWH SC	No	Yes	No	No	N/A	N/A
43	No	Yes	No	No	transglanular to corpus cavernosal shunt	transglanular to corpus cavernos-al shunt	N/A
44	hydroxyurea	No	yes	No	transglandular cavernospongiosum shunt	transglandular caverno-spongiosum shunt	surgical curettage of the penis.
44	NA	Yes	No	No	winter's T shunt,	N/A	N/A
44	NA	Yes	No	No	No	N/A	N/A
45	NA	Yes	No	No	winter's T shunt	N/A	N/A
46	Vincristine sulfate and prednisone	No	No	Yes	No	radiation ,complete resolution after 6 d	N/A
47	imatinib	Yes	No	No	No	cavernosaaspiration was unsuccessful. Imatinib	N/A
48	NA	Yes	No	No	Distal corporoglanular shunt	N/A	N/A
49	analgesics, anxiolytics and steroids	Yes	No	No	No	bilateral aspiration and irrigation	N/A

50	hydroxyurea, allopurinol and intravenous fluids imatinib	No	Yes	No	No	leukapheresis	N/A
51	busulfan	No	No	No	No	subsided gradually over a two to three week period	Yes
51	hyaluronidase	No	No	Yes	No	NA	N/A
51	Busulfan	No	Yes	No	No	NA	No
51	Busulfan, hydroxurea	No	Yes	No	sapheno-cavernous bypass.	NA	Yes
52	Busulfan, hydroxurea	No	Yes	No	sapheno-cavernous bypass.	NA	N/A
52	pain killers	NA	No	No	Proximal shunt	N/A	N/A
52	Cavernosal Pseudo-Ephedrine Inj, pain killers	NA	No	No	No	N/A	N/A
52	pain killers	NA	No	No	Proximal shunt	N/A	N/A
52	Cavernosal Pseudo-Ephedrine Inj, pain killers	NA	No	No	No	N/A	N/A
52	pain killers	NA	No	No	No	N/A	N/A
53	N/A	Yes	No	No	No	N/A	N/A
53	NA	NA	No	No	Winter shunt	NA	Yes
53	NA	NA	No	No	Winter shunt	NA	Yes
53	NA	NA	No	No	Winter shunt	NA	Yes
53	NA	NA	No	No	Winter shunt	NA	Yes
54	NA	NA	No	No	Winter shunt	NA	Yes
54	allopyranol hydroxyurea	NA	No	No	cavernosoum-spongiuosum shunt	allopyranol hydroxyurea , cavernosoum- spongiuosum shunt	No

55	allopyranol hydroxyurea	NA	No	No	cavernosoum-spongiuosum shunt	allopyranol hydroxyurea , cavernosoum- spongiuosum shunt	No
56	myeleran endoxan	No	No	Yes	No	radiation to the penis	N/A
57	NSAIDS and Diethyl Stilbesteol	No	No	No	glandulo-cavernosal shunt	improved after the shunt	Yes
58	No	Yes	No	No	No	N/A	N/A
59	cold compressision , priscoline hydrochloride , diethylstilbestrol	No	No	Yes	No	radiation therapy the penis	N/A
60	benzyle penicilin busulfan , trioxyphenbutazone	Yes	No	No	No	initial aspiration little response improved after irrigation (lowselys operation)	N/A
61	N/A	NA	No	No	NA	immediate surgical decompression	N/A
62	NO	NO	NO	NO	saphenocavernous anastomosis	N/A	N/A
63	Yes	Yes	Yes	no	corpus cavernosa–glans shunt	Aspiration and irrigation	Yes
64	Busulfan	Yes	No	No	No	with bothe busulfan , irrigation and aspirartion of the corpora cavernosa	Yes
64	busulfan	Yes	No	Yes	No	improved after aspiration and irrigation	Yes
64	No	Yes	No	Yes	No	after aspiration and irrigation of corpora cavernosa	Yes
65	Yes	No	No	No	No	N/A	N/A
65	Yes	No	No	No	No	N/A	N/A

56	hydroxyurea allopurinol Imatinib	Yes	No	No	No	improved after aspiration	Yes
66	allopyranol	Yes	Yes	No	Distal shunt	r distal distal shunt procedure	N/A
67	Myleran intravenous A-139 I, demerol	No	No	No	No	flaccid after two weeks of medical therapy	N/A
51	hydroxyurea	No	Yes	No	sapheno-cavernous bypass.	after sapheno-cavernous bypass.	Yes
51	N/A	Yes	No	No	No	N/A	N/A
51	Busulfan	Yes	No	No	No	N/A	Yes
51	Busulfan Steroids. Anticoagulants.	No	No	No	No	N/A	Yes
51	busulfan	No	Yes	No	No	N/A	Yes
68	allopyranol hydroxyurea	No	No	No	No	the priapism subsided after 24 hr of starting medicalk treatment no aspiration was done	No
51	Busulfan Anticoagulants	NA	No	Yes	No	no benefit	Yes
50	busulfan	Yes	No	No	shunt between the right saphenous vein and corpus cavernosus	shunt between the right saphenous vein and corpus cavernosus	N/A
69	busulfan then, 6-mercaptopurine, busulfan again	No	No	Yes	No	improbed after radiotheray on the 2 weeks period	N/A
70	No	No	No	No	No	on presentation no priapism was there , two episodes of priapism that resolved, and he was treated three times with metronidazole for presumed balanitis.	N/A

41	1 LMWH SC BID for one month , Hydroxyurea Cyclophosphamide	No	Yes	No	No	N/A	N/A
41	1 LMWH SC / hydroxyurea	No	No	No	No	Hydroxyurea	N/A
71	hydroxycarbamide	aspiration done	No	No	No	hydroxycarbamid aspirin 4 units of PRBC	No
71	cyclophosphamide 1g stat, hydroxycarbamide 1g 12 hourly	No	No	No	No	20th day of admission with significant healing of the penile shaft ulcers, significant detumescence	N/A
37	No	No	No	No	No	Imatinib	developed ED EHS2. Grade 1
72	Yes	No	No	No	No	By the 4th week of cytoreduction	Yes
37	hydroxyurea	Yes	No	No	Al-Ghorab shunt	Aspiration Shunt	developed ED EHS2. Grade 2
73	Yes	No	No	No	No	terbutaline 0.125 mg subcutaneously	N/A
74	Paracetamol Morphine Diazepam, prednisone Terbutaline terbutaline 5.0mg, hydroxyurea	No	No	No	Winter shunt	Corpora cavernosa aspiration (winter), prednisone 2, Terbutaline terbutaline 5.0mg, tab hydroxyurea	N/A
75	No	Yes	No	No	No	corporal aspiration and phenylephrine irrigation	N/A
76	No	Yes	No	No	No	aspiration followed by intracavernosal injection of 1 dose of phenylephrine	No
77	No	Yes	No	No	No	penile aspiration and irrigation	N/A