

A Patient of Peripheral Vascular Disease in Type 2 Diabetes

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62 year old man was admitted to Jaslok hospital on 4.8.97 with complaints of a non-healing wound over left great toe and uncontrolled diabetes.

Patient was a known non-insulin-dependent diabetic since 25 years and was inadequately controlled on oral hypoglycemic agents. (DBI & Daonil)

In the month of Jan-Feb '97 he developed a pustule over his left great toe. He was treated with antibiotics at Rajasthan, without relief. Pustule developed into an ulcer and surrounding area was inflamed with pain and swelling. He was admitted to a hospital at Rajasthan, local debridement was done. Discharge card of the hospital showed impaired renal parameters. (BUN 72 mg%, Creat 3.0 mg%).

Post-debridement wound did not improve and swelling and purulent discharge from wound increased which gradually developed into gangrene. X-ray showed erosion and osteoporosis.

He is a known hypertensive since 5 years and was put on Dytide once a day and nefedipin R 1 OD. He was taking these drugs irregularly.

History of tingling and numbness of feet was noted for the past 8 – 10 years. Five years back there was history of renal colic followed by passage of a stone in the urine. Presently there was no history of pyuria, nocturia or urinary tract infection.

Patient was referred to Jaslok Hospital for further management. On admission on 4 Aug. 1997, examination revealed Pulse 90/min, BP – 150/90 mmHg, respiratory rate – 16/min, JVP was normal. Mild oedema of the feet was noted.

Examination of peripheral pulses revealed bilaterally nonpalpable dorsalis pedis, left popliteal was poorly felt. Femorals were bilaterally well felt. Carotids were palpable without any bruit. Radials were normal.

Local examination of left great toe showed gangrene upto I – P joint, with purulent discharge. Margins were III defined. There was cellulitis upto proximal

1/3 of the foot on left side. Marked wasting of muscles on both the legs was noticed. Left foot and leg was cold as compared to right (Fig. 1) at the time of Initial presentation.



Fig. 1: At the time of initial presentation.

Ocular examination showed left eye cataract and right operated with IOL. Background diabetic retinopathy and hypertensive changes were seen.

Respiratory system examination showed occasional crackles in sub-scapular region.

Cardio vascular system was clinically normal.

CNS examination revealed glove and stocking type of anesthesia. Bilaterally ankle jerks were absent.

On routine examination biochemistry showed – Hb – 8.8 gm%, WBC – 15,000/cmm, ESR – 80 mm after 1 hr, Creatinine 3.0 mg%, Bun 41.6 mg%, Ca 9.5 mg%, P 3.5 mg%, Uric acid 5.8 mg%, protein 6.9 gm%, Alb – 3.7 gm%.

E.C.G. – RBBB pattern. No ischemic change. Chest X-ray – Normal. Urine routine – 2-3 pus cells. Alb - +++ Granular casts +++. Pus c/s – Pseudomonas sensitive to ceftazidime. Urine c/s – Klebsiella and citrobacter. (significant colony count).

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On preliminary examination diagnosis of NIDDM with diabetic neuropathy, nephropathy, retinopathy and peripheral vascular disease was made.

The patient was investigated further –

X-Ray Left foot showed bony erosion and osteoporosis.

U.S.G. (abdomen) – Bilaterally normal sized kidneys. Gross atherosclerotic disease of aorta. Arterial doppler was done in view of gangrene of the left great toe. It showed generalised atherosclerotic disease of aorta. Distal superficial femoral artery showed 70-80% obstruction with marked increased velocities at stenotic site distal to popliteal. Post. Tibial and dorsalis pedis showed dampened flow.

COURSE IN HOSPITAL

Diabetes was managed by 3 dose insulin therapy, (combination of Actrapid + Monotard), 1500 calorie diabetic diet, modified according to renal parameters. O.H.A. were omitted in view of poor control and renal dysfunction.

Hypertension was tackled by Nefidipine-R (10 mg) OD. ACE inhibitors were avoided in view of possibility of atherosclerotic involvement of renal arteries.

Diabetic nephropathy was managed conservatively as follows :

Diabetic diet, 20 gms of protein of high biological value, low potassium, 4-5 gms of salt and normal fluid intake.

Left foot gangrene was managed in following systemic manner –

1. Cefotaxime as suggested by c/s report and adjusted according to creatinine, for wound infection.
2. Daily dressing with Eusol and Betadine solution.
3. Decision of surgical intervention was withheld and it was decided to do peripheral angioplasty to improve local blood circulation.

Peripheral angioplasty was done with 50 cc of nonionic dye for imaging. Great care was taken regarding adequate hydration and maintenance of

adequate urine output, in view of renal failure. (Fig 2)

Post angioplasty dorsalis pedis was well felt and significant change in local temperature was noticed. (Fig 3). Patient was put on Pentoxifylline and low molecular weight heparin 3500 U/d for 10 days.



Fig. 2: Baseline angiography.



Fig. 3: Post angioplasty angiography.

Patient developed a small blister on the plantar aspect of the foot following the procedure on second day (Fig 4). This was due to microembolisation produced during the procedure.

Patient was discharged with advice to come back after 10 – 15 days for consideration of surgical intervention.

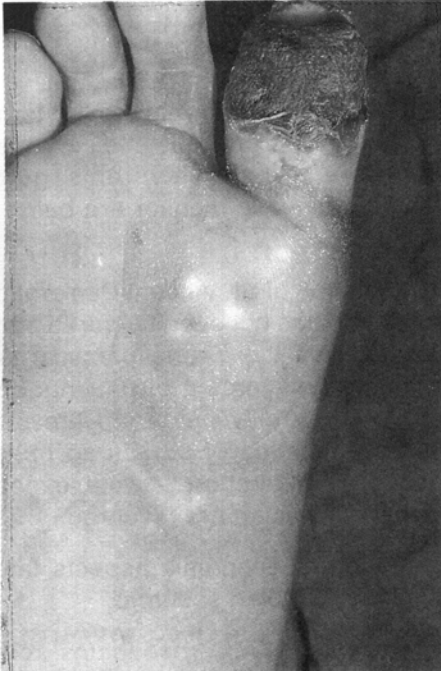


Fig 4: Blister on the plantar aspect Following angioplasty.



Fig. 5: Appearance following conservative amputation.

Patient was readmitted on 27-8-97. On admission, local examination revealed significant reduction in purulent discharge, marked improvement in proximal cellulitis. Margins of the gangrene were clearly defined. (Fig. 5).

Repeat doppler showed good distal blood flow upto dorsalis pedis.

Patient was referred to a plastic surgeon for conservative amputation of left toe. Excision of the left great toe was performed at I – P joint with primary flap closure on 30-8-97. (Fig 6). Post-operatively, patient made an uneventful recovery and was discharged on 3.9.97.

DISCUSSION :

This case history reveals difficulties encountered in treating long-standing diabetics with vascular problems. In this patient, significant peripheral neuropathy and infected gangrene. In addition, the patient suffered from diabetic retinopathy and nephropathy with renal failure. His treatment required constant caution to avoid aggravation of renal failure. Hence, fluid and electrolyte therapy had to be carried out meticulously and nephrotoxic drugs avoided scrupulously. The use of a low dose non-ionic dye was made for imaging procedure.

The most important decision required here was whether excision of gangrenous toe should be undertaken before or after the attempt to restore peripheral circulation by angioplasty. In this situation, we decided to attempt angioplasty first because of the possibility of non-healing of wound if excision were undertaken before angioplasty. Although the peripheral vascular disease is diffuse in diabetics, as was the case here, it is worth attempting angioplasty of proximal vessels to provide overall a greater pressure-head proximally with improved distal flow. This approach worked favourably in this patient although complications like microembolisation and further vascular occlusions are distinct possibilities as attending angioplasty in this situation. This patient had some microembolisation which improved with heparinisation.