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A Case Study on Liv.52 an Adjuvant in Diabetes Mellitus

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An established case of diabetes mellitus was followed up from 6-10-1977 at which stage the fasting blood sugar was 200 mg% and two hours after food, when it was 300 mg%. The blood sugar level had to be kept under control by administration of Glibenclamide in the doses of 5 mg t.i.d. The patient, Mrs. M.D., aged 25 years, whose obstetric record was somewhat poor, was admitted to the Rajendra Medical College for her confinement.

Prior to this, when her diabetes had not been detected, the lady had a full-term forceps delivery. Unfortunately, the baby had expired after four days. Subsequently, she had an abortion of $1\frac{1}{2}$ month's gestation. This time, she delivered a premature female baby by L.S.C.S. on 21-11-1979. The patient was discharged from the hospital on 3-12-1979. During her hospitalisation, her blood sugar was kept in check by administration of insulin and Glibenclamide.

Four months after her confinement, the lady developed infective hepatitis. It was at this stage that I examined her on 4-4-1980. I found her icteric, her liver being enlarged 1½ finger below the right costal margins and tender to the touch. Her serum bilirubin assessed on 7-4-1980, was 2.5 mg% and her urine also showed an excess of urobilinogen. I prescribed a course of Liv.52, 2 t.i.d. for three months.

Five weeks later, her icterus had disappeared and she reported 'feeling absolutely normal'. The patient had on her own stopped taking Glibenclamide and yet, her urine was free from sugar. On completion of the course of three months, she even stopped taking Liv.52 tablets.

A few days after, she came to my clinic again complaining of extreme exhaustion, fatiguability and lack of interest in surroundings. When subjected to an examination, her urine once again revealed the presence of sugar-more than 1.5 mg%. I prescribed her Glibenclamide 5 mg t.i.d. alone for one week. However, there was no perceptible improvement either in the symptoms of the patient or her urine. So, I added Liv.52, 2 tablets t.i.d. as an adjuvant and later, gradually withdrew the antidiabetic. On re-examination 4 days later, her urine sugar was found to be negative. The patient reported feeling energetic and showed active interest.

The patient has been maintained on Liv.52, 2 tablets t.i.d. alone for quite some time. A recent blood sugar estimation on 27-6-1981 showed the fasting blood sugar level to be 131 mg% and post prandial 2 hours to be 184 mg%. She is stated to be on a liberal diet and takes recourse to Chlorpropamide only when she has been very indiscreet with her diet. Otherwise, she is routinely on Liv.52 alone.

Further to the above mentioned case, I have had the opportunity to follow-up closely the effect of Liv.52 in 5 other established cases of maturity-onset diabetes. Of these, 3 cases had gradually become resistant to Glibenclamide, which they had been using for control of diabetes since several years. They had been taking 15 mg (3 tablets) Glibenclamide daily and still their urine was positive for sugar. Adding Liv.52, 2 tablets t.i.d. to daily regimen, brought down their urine sugar to negative within a week. Thereafter the dosage of Glibenclamide could be reduced to 10 mg (2 tablets) per day, after 20 days.

Similarly, the other 2 cases that had become resistant to Tolbutamide, benefited from the addition of Liv.52 to the regimen. Their urine became free from sugar. So also, the Tolbutamide dose could be reduced.

It is my surmise that besides improving the hepatic function, Liv.52 probably also improves the pancreatic function, thereby regulating the release of insulin from beta cells of islets of Langerhans. Liv.52 also restores the well being of the patient and minimises diabetic neuropathy.