THE ELECTROCARDIOGRAPHIC CHANGES IN ORGANOPHOSPHATE POISONING (DICROTOPHOS "CARBICRON") -A CASE REPORT

Pages with reference to book, From 44 To 50

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Abstract

A case of Organophosphate poisoning in an agricultural pesticide sprayer who had electrocardiographic changes consistent with myocarditis has been presented. The changes appeared on the third day of poisoning and cleared almost completely as the condition of the patient improved. The changes in E.C.G. were considered due to the direct effect of the poison on the myocardiam. This is probably the first reported case of Electrocardiographic abnormalities due to organophosphate poisoning.

Introduction

In an attempt to achieve self-sufficiency in food and to obtain a higher per acre yield in agriculture, modern means are being increasingly applied in Pakistan and other agricultural countries. Proper and in time use of pesticides is an important measure which can help the farmer in realizing this aim. These highly poisonous compounds are used by the un-educated and un-trained farmers or the agricultural workers. The instructions circulated by the manufacturers of the pesticides and those taught by the agricultural authorities are ignored. As the use of pesticides for pest control is increasing the incidence of accidental poisoning by these compounds is also on increase. Because Anti-Cholinestrases the carbamates and organo phosphorous compounds are more frequently used today for pest control; the Syndrome of Anti-Cholinestrase poisoning is one of the commonest accidental poisoning met in the clinical practice. Of 1280 (1974-75) total annual admissions in the District Headquarters Hospital, Sargodha, for two accidental or suicidal tranquilizer poisoning and one copper sulphate poisoning, six cases of pesticide poisoning were admitted. Out of these nine cases of poisoning admitted in this hospital, three died. One died due to Barbiturate poisoning (Sondryl), one due to Copper Sulphate poisoning, and the third due to Organo Phosphate poisoning.

The purpose of this paper is to familiarize the medical practitioners with the problems posed by the Organo Phosphate poisoning and their management. I also specially report a case of Organo Phosphate poisoning 'Carbicron' (DICRO-TOPHOS) who developed electro-cardiograpnic abnormalities during the period of his illness.

Case Report

G.H. 30 years old male, an employee of the Agricultural Department had been engaged in pesticide spray for the last fifteen years without ever having contracted ill-effect. His job demanded daily pesticide spray amounting to many gallons. He and his colleagues had been properly taught the handling of the pesticides but with familiarization they tended to ignore precautionary instructions. On the 9th June, 1975 he did his routine spray work with Dicrotophas (CARBICRON) till lunch time. While spraying he insufficiently covered his eyes, face, hands and feet. He took his lunch without taking a bath or washing his hands or face. He went off to afternoon nap in the same clothes which he wore while spraying the pesticides. Two hours later he woke up severely giddy, perspiring profusely, had troublesome nausea, salivation, lacrimation with disturbed vision and started vomiting incessantly.

He developed colicky abdominal pain with loud borbory gmi and started having diarrhoea with faecal in-continance. He rapidly developed generalized severe muscular cramps, weakness and twitchings. By the evening he had to be helped up and was almost paralyzed all over. He had also become some-what disoriented in time and space. He was "brought to hospital a few hours after the onset of symptoms. He had been washed up by his colleagues and clothes had been changed but contrary to standing instructions by the department and manufacturers the supplied injection of Atropine 'Ready for Use' was not injected by the attendants or the officials accompanying the victim.

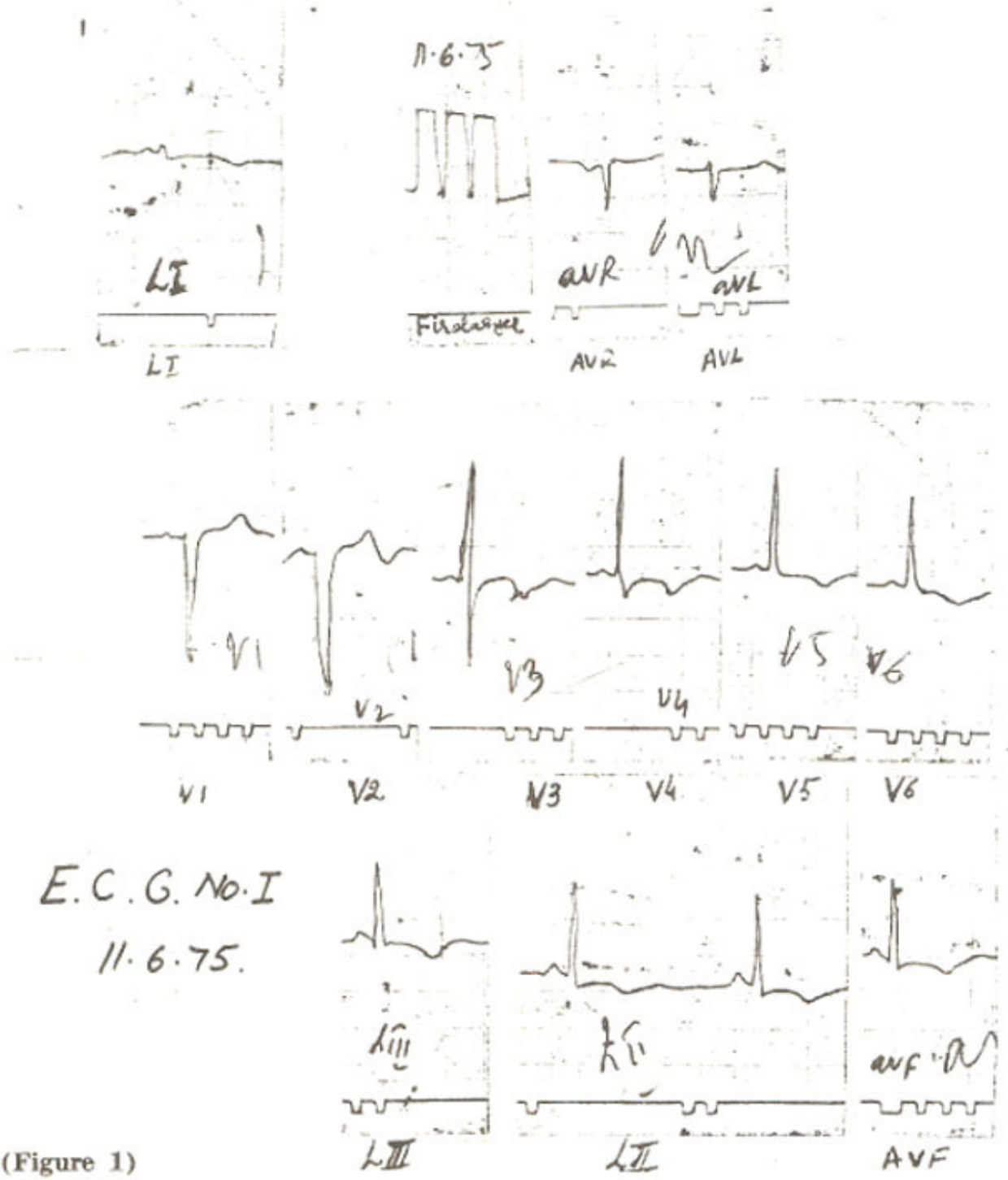
On admission he was disorientated in time and space. He had anxious look on the face and the complexion was red. Eyes were congested and had excessive lacrimation; pupils were small and fixed. He had excessive salivation. Skin was warm and he was sweating heavily. Temperature was normal. Pulse on admission was 60 per minute and was full in volume and regular in rhythm. Blood pressure on admission was 140/70 mm Hg. Phonation was weak and clear. Respiration was shallow and rather laboured, breath sounds and air entry was poor and a few scattered rates were audible. Apex beat was palpable in fifth inter-coastal space and norma 1 in character. Heart sounds were both audible and normal. Mentally he was slightly disoriented and inattentive but was answering question. Eyes were fixed in central position, blinking was infrequent, pupils were small and non reactive. Generalized muscular twitching and fasiculations were visible and there was almost complete paralysis of all the four limbs and trunk. Respiratory excursion was also poor. Cough reflex was present, deep tendon reflexes were all markedly depressed. Plantar reflexes were normal. Sensations were normal.

Immediately, after admission he was given 2 mg of Atropine Sulphate inter-venously and repeated every ten minutes till the signs of atropinization appeared i.e. stoppage of salivation and dialatation of pupils. His respiration improved, sweating, abdominal pain, diarrhoea, pulse rate all got markedly improved but muscular weakness persisted. This state was maintained. From then on he was kept under close observation and Atropine was repeated two to four hourly. Next day on routine examination third heart sound was audible at the apex and along the lower end of sternum. This is when the first E.C.G. was taken on 11.6.75. By this time his general condition had improved. He was not having diarrhoea, vomiting or sweating.

During the management, the dehy-deration was corrected by 5% Dextrose in Isotonic saline infusion. Nasogastric tube was passed for feeding. Liberal quantities of fruit juices, soups and milk feeds were given regularly. Throat was kept clear by frequent suctions and he was also given Penicillin and Streptomycin for the minor chest infection. He was also given Oxygen at the rate of 2-3 litre per minute. Urinary output remained satisfactory. Blood Urea and electrolytes remained normal throughout. X-ray chest showed no abnormalities. Routine haemotological and urinary investigations were normal. E.C.G. was recorded daily.

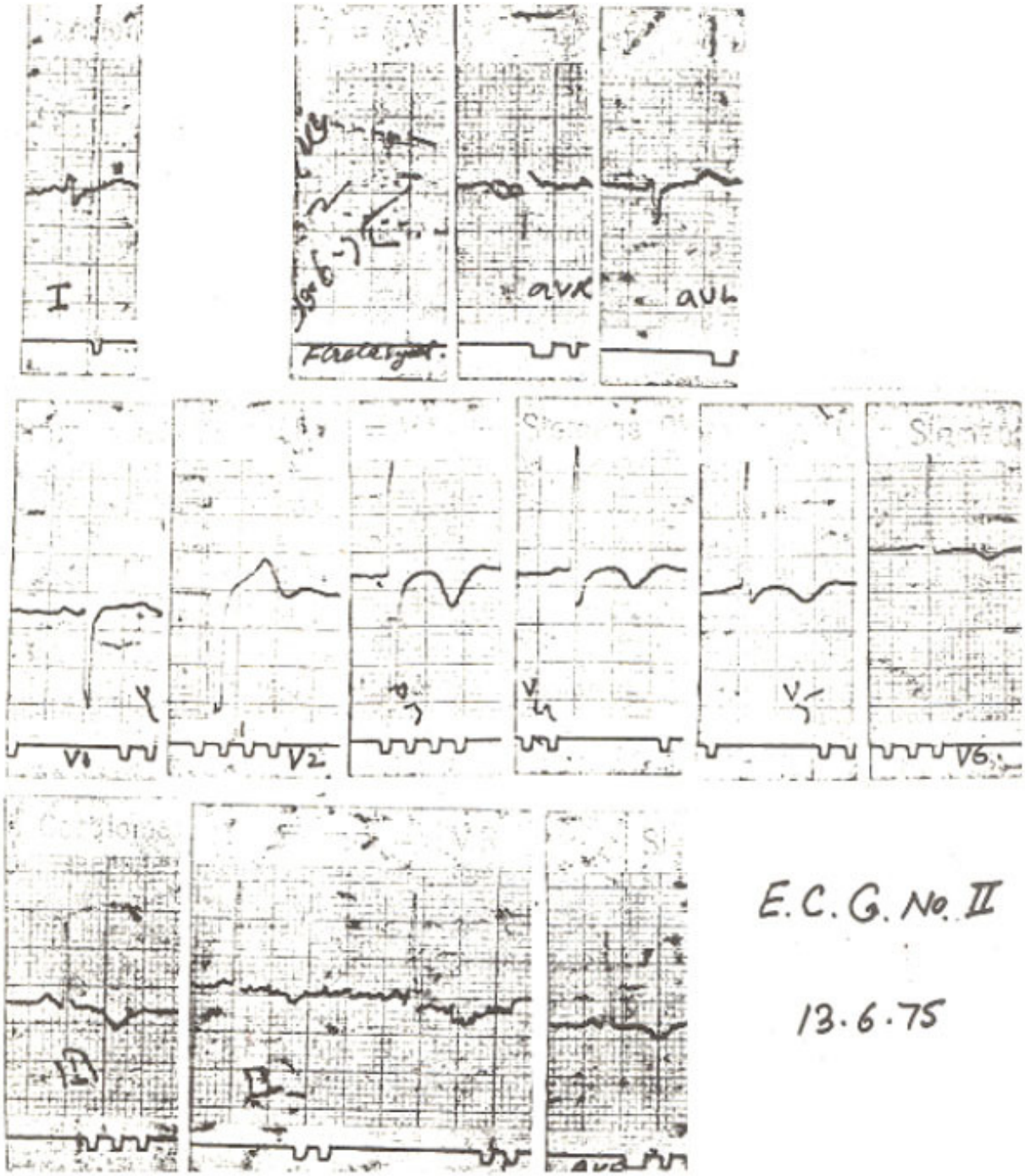
Within 8-9 days after the onset of poisoning he showed no clinical signs of toxicity, E.C.G. had returned to normal, and he was discharged from hospital. He was re-examined twice at fortnightly intervals and routine examination and E.C.G. were found to be normal.

Interpretation



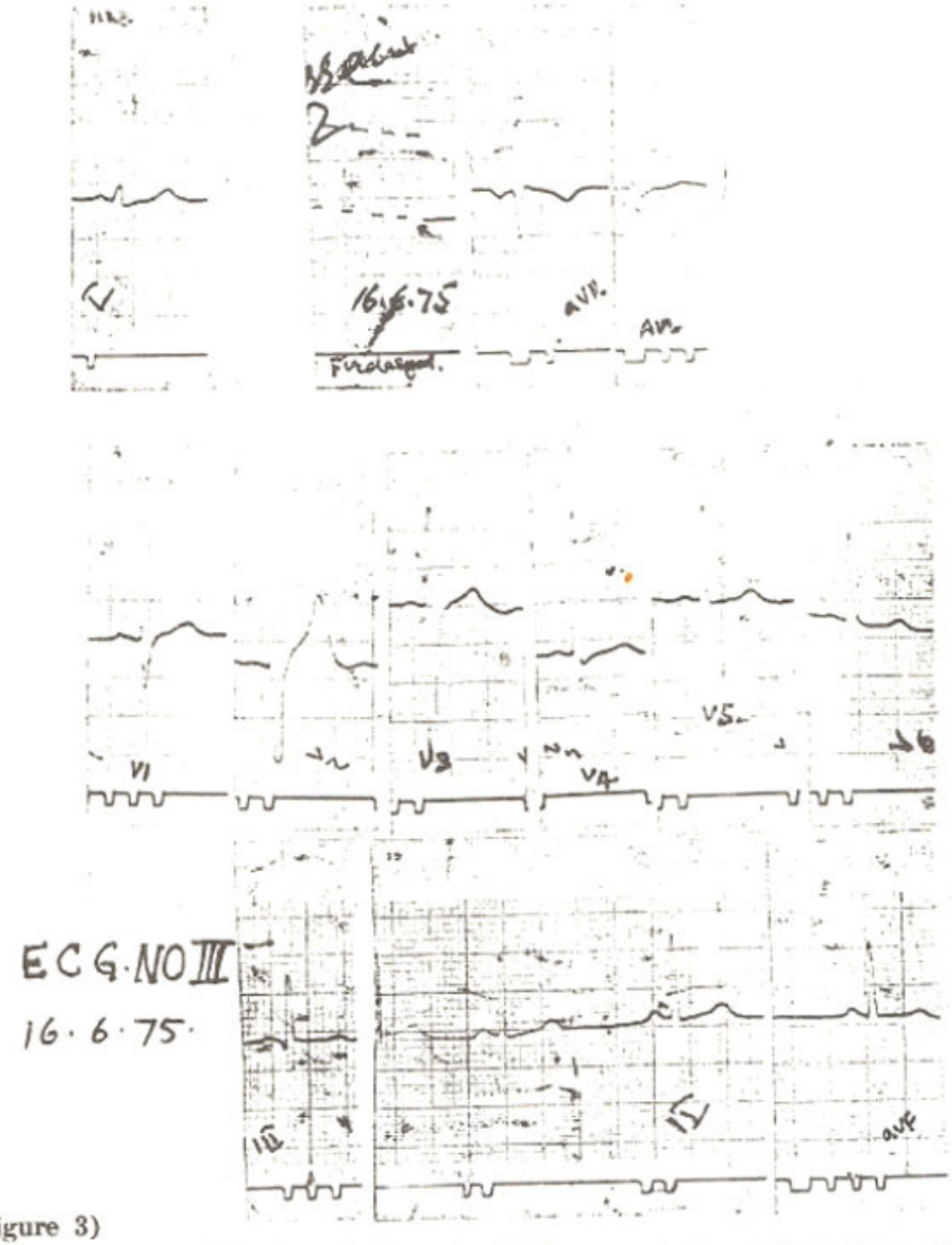
(Figure 1)

11.6.75—Sinus Rhythm with sinus bradycardia (rate 58 per minute) Non-specific T. wave inversion is present in L I, L II, L III, AVF, V3-6. T. wave is Biphasic in V2, S-T segment is slightly depressed in V4-5. Q-T internal is 0.44 second.



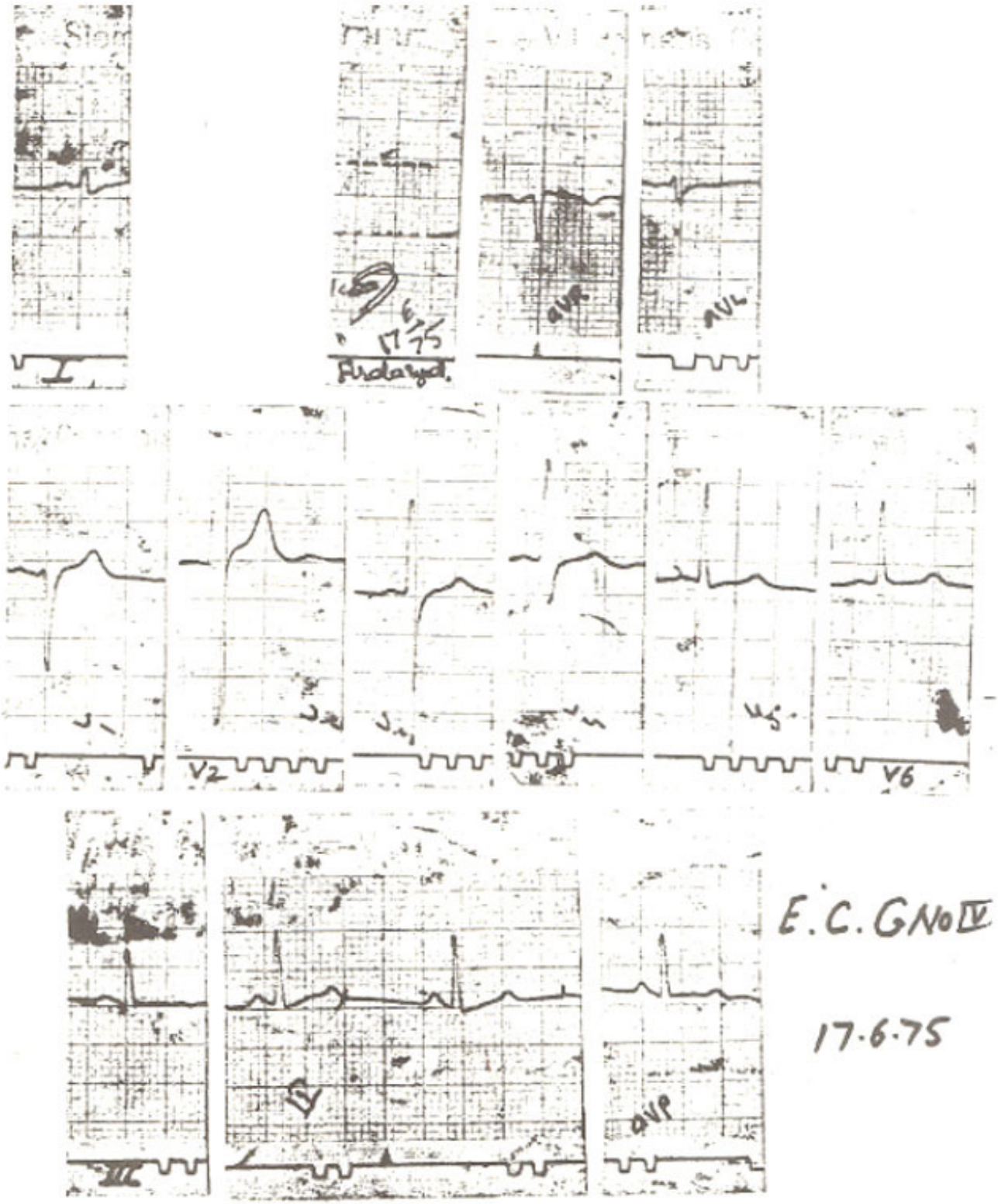
(Figure 2)

13.6.75—Findings are essentially similar to those of first E.C.G. dated 11.6.75 although the heart rate has improved to 75 p.m. T. wave is flatter in VI and Monophasic positive in V2 but U-wave is more prominent in V2, S-T segment is Isoelectric in all the leads. Q-T is 0.48 seconds. T-Inversion is slightly more pronounced.



(Figure 3)

16.6.75—Sinus Rhythm at the rate of 67 per minute and regular. T-wave in all the leads had reverted to normal upright and Q-T interval is 0.32 second.



(Figure 4)

E.C.Gs in figure 1 and figure 2 shows almost generalized inversion of T waves and plongation of Q-T interval. From the presentation of the patient and from the whole clinical setting it appears that these changes were the direct result of the toxic effect of the organophosphate (Carbicrone) which the patient

was heavily exposed to. As the general condition of the patient improved and the generalized toxic manifestation of the poisoning disappeared electrocardiographic abnormalities also reverted back to normal. This seems to be another evidence in the favour of the changes being direct due to the toxic. Thus this was taken as a case of toxic Myocarditis due to the Pesticide-Organo Phosphate-Dic-rotophos.

Conclusion

It is re-emphasised that the anti choli-nestrase poisoning by Organo phosphate and Carbanate Pesticide is not common. It is the most common accidental poison in this area and many cases of milder poisoning go unreported. The clinical presentation and the management of these very serious cases of poisoning should be widely publicised. Strict protective measures should be made obligatory for those who handle these products by education, persuation and legislation.

It is emphasised that the Organo Phosphate can give rise to electrocardiographic abnormalities and the possibility of myocarditis due to these compounds should always be kept in mind and every case reported should have Electrocardiogram recorded regularly.