CASE REPORT

Hypertension After Multiple Transfusions

by

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Abstract

Two patients are reported who developed hypertension with or without convulsions after receiving mutiple blood transfusions. The pathogenesis of this complication of transfusion is briefly discussed.

Received 4th September 1981.

Introduction

It is well known that blood transfusion carry a risk of complications that in the course of time has been reduced to a minimum level. For a substantial review of the possible complication that may occur after transfusions one is referred to Mollison's textbook (1979) and more recent article (Barton, 1981).

One of the complication that has so far not came to the fore, is the occurrence of hypertension. Recently we have encountered this complication in two patients after multiple transfusions.

Case reports

Case 1. H., a 9 years old Indonesian boy was admitted to the hospital with the main complaints of fever and an enlarged abdomen. The fever had lasted for 10 days and the abdomen was noticed to be enlarged for about six months.

On physical examination the boy looked pale, was undernourished and had hepatosplenomegaly. The blood pressure was 110/70. Significant laboratory findings were as follows: Hb. : 4.6 g/dl. decreased osmotic fragility, a high Hb F content (22%) and the presence of Hb E (41%), while the blood smear showed a picture compatible with hemoglobinopathy. The diagnosis of Hb E - Thalasemia was made. During a period of 10 days the patient received 5 units of pecked red cells of ± 150 ml. each, after which he was discharged with a Hb. 14.6 g/dl. His blood pressure was 110/80.
Two days later he was rushed back to the emergency ward because of severe headache, oliguria and vomiting. The blood pressure was 175/125, the heart beat and pulse rate were 88/minute and irregular, there was no fever. Intravenous fluid drip with 10% dextrose and furosemide (20 mg) were administered. Two hours after admission the boy got convulsions upon which diazepam 5 mg was given intravenously. The seizure susided, but the blood pressure remained at 150/130. A lumbar puncture yielded a normal cerebrospinal fluid. Serum elec trolytes were as follows: Na: 130,7 mEq/L., K: 4,7 mEq/L., CO2: 24,4 mMol/L., Cl: 97 mEq/L BUN was 10 mg %, serum creatinine 0,7 mg %. The patient became soporous.
Reserpin and lanoxin were administered upon which the blood pressure began to fall to 140/100. The ophthalmologist and pediatric cardiologist could not find any abnormality in their respective fields, the neurologist confirmed the presence of hypertensive encephalopathy. The blood pressure gradually dropped to normal levels., and 3 days later it remained stable at 110/80-70. The patient also regained full consciousness, the intravenous fluid drip was discontinued. Cessation of antihypertensive therapy did not cause a rise again in the blood pressure and the patient could be discharged after 20 days of hospitalization. One month later a follow-up examination revealed the boy to be in good general condition without any sign of neurological sequelae, his blood pressure was 110/70.

Case 2. N.K., an 8 years old Indonesian girl, was admitted to the hospital for typhoid fever and treated with chloramphenicol. After 13 days of treatment she suddenly produced copious bloody stools.

The hemoglobin concentration dropped to 3,2 g/dl., but her blood pressure remained normal, 110-120/60-80 and her temperature was 37,2 C. She was immediately transfused with whole blood after which the hemoglobin rose to 7,8 g/dl. The next evening she had melena again, the Hb. dropped to 4,9 g/dl. She was transfused again, partly with whole blood and partly with packed red cell until her hemoglobin rose to 10,1 g/dl. Two days later a third bout of melena necessitatated more transfusion. Altogether she received 600 ml. of whole blood and 300 ml. of packed red cells. Her body temperature remained normal and so did her blood pressure. However, 4 days after the last trasfusion she complained of bursting headache for which she was given novalgin. It apeeared that her blood pressure had risen to 140/120, the following day it became even higher, 170/120.

Serum electrolytes were as follows : Na : 133 mEq/L, K :4,5 mq/L., Chloride: 105,9 mEq/L. and CO_2 : 16,5 mMol/L. BUN and serum creatinine were 25 mg % and 0,9 mg % respectively. The patient remained alert and did not complain any more of headache.

Ophtalmologic examination was negative. This second case occurred not long after the experience of the first case, so it was thought that the hypertension would soon disapear spontaneously. However, the blood pressure kept rising and 6 days later it reached a level of 210/130. It was then decided to treat the hypertension with serapes upon which the blood pressure gradually fell to normal levels. Six days later serapes could be discontinued because the blood pressure stabilized at 110/80. In the meantime signs and symptoms of the typhoid abated and the patient could be discharge after 39 days of hospitalization. No neurological sequelae were observed.

Discussion

The occurrence of hypertension after tranfusions has been reported earlier (Royal and Seeler, 1978; Yetgin and Hicsonmez, 1979).

Wasi et al (1978) described 8 thalassemic patients who experienced hypertension, convulsion and cerebral hemorrhage after multiple blood transfusions in preparation for splenectomy. He considered these complications as a specific syndrome after multiple transfusions that started with hypertension and could lead to hypertensive encephalopathy and cerebral hemorrhhage.

The occurrence in thalassemic patients would suggest that host factors might contribute to the rise in blood pressure. Vandongen and Gordon (1969) reported that angiotensin was generated in plasma pools that had been at ambient temperature for a reasonable period. This generated angiotensin was relatively resistant to destruction by endogenous angiotensinases in plasma and could affect the

blood pressure and aldosteron secretion of human recipients.

Our experience revealed that host factors were probably not contributory to the rise in blood pressure since the second patient was not thalassemic. The hypertension was not due to circulatory overload since it occurred a few days after the transfusions as was also observed by Wasi et al 1978. Besides, no congestive heart failure was present in both patients. Apparently the transfused blood contained vasoactive substances i.e. angiotensin that caused the hypertension.

The first case caught us by surprise and treatment was at first aimed at hypertensive encephalopathy due to acute renal failure. Although the condition was incorrectly interpreted symptomatic treatment gave relief to the hypertension. The experience of the first case guided our management of the second case. Although Vandongan concluded that the generated angiotensin was relatively resistant to angiotensinases in normal plasma we expected that its effecs on the blood pressure would be transient.

Therefore, the patient was only closely observed and no antihypertensive drugs were given. However, when the blood pressure kept rising we considered it inappropriate to withold treatment any longer. The choice of antihypertensive drug was guided by the assumption that rise in blood pressure was due to angiotensin. Serapes, containing reserpin, hydralazine and hydrochlorthiazide was chosen because its antihypertensive action includes the vasodilating effect of hydralazine. In both cases the blood pressure began to fall upon administration of antihypertensive agents and no rebound was observed after withdrawal of the drugs. This would indicate the temporary character of te hypertension.

Lovric had never received any report on the occurrence of hypertension after blood transfusion and suspected that the complication might be the result of substandard collection of blood donors and its subsequent storage as might happen in blood banks in developing countries. This statement might hold some truth, though not so much as concerning the collection and storage of blood from donors and regarding to the procedure of its administration to the recipient.

Firstly, the occurrence of hypertonsion is reported in tropical or subtropical countries such as Thailand, Turkey and in our case Indonesia where the ambient temperature can be quite high in wards that are not air-conditined. Secondly, ward nurses are often overburdened with work especially when the wards are overcrowded with patients some of who need intensive care. Insuch situations it may happen that a blood pack is exposed to room temperature for quite a while before it is administered to the patient. However, this reasoning would not explain its occurrence in Chicago as reported by Royal and Seeler (1978). It would also be hard to explain why such complications had not occurred more often. The fact that the second patient had complaints of headache only at the beginning of the hypertension and was symptom-free when her blood pressure peaked to 210/130 might suggest that the complication had probably occurred more often but had remained undetected.

After the above mentioned experiences it was decided that any patient receiving mutiple transfusions should be closely observed for the occurrence of hypertension while ward nurses were urged to avoid exposing blood packs to ambient temparature before administering the blood to recipients.

Addendum

Since the writing of this paper we have had a further patient who developed hypertension with convulsions six days after transfusion of 250 ml. of packed red cells. This complication occurred in a 5½ years old boy with acute lymphoblastic leukemia during maintenance treatment.

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