Total Body Washout (TBW)

Donald L. Groat, AMSECT

Department of Cardio-Thoracic Surgery, Temple University Health Sciences Center, Philadelphia, Pennsylvania 19140.

Asanguineous hypothermic total body perfusion or total body washout (TBW) as a successful therapeutic procedure for treating Stage IV Hepatic Coma.

ABSTRACT

Asanguineous hypothermic total body washout is the ultimate extension of extracorporeal total bypass technology if conducted properly and the disease and state of coma diagnosed promptly enough to enable the patient to undergo the simple but traumatic procedure.

CASE REPORT

History

This was the first Temple University Hospital admission for this 21-year-old black female on August 15, 1972. She had been well until two weeks PTA, at which time she noted the onset of malaise, anorexia and mild nausea without vomiting. Three days PTA, the patient, who was a known asthmatic since age 3, suffered an acute asthmatic episode for which she received epinephrine and aminophylline at another hospital. She had noted dark urine and light stools for two days PTA. Nausea, this time accompanied by vomiting, occurred the night before admission. She was given Thorazine at another hospital without relief. She denied receiving any blood or blood products, use of parenteral drugs, contact with any known case of hepatitis or recent shellfish ingestion. PMH other than occasional asthmatic attacks were negative.

Physical Examination

On physical examination the vital signs were: pulse, 140 and regular; BP 140/80; respirations, 28; temperature 98°F and weight, 192 lbs. She was alternately lethargic and hyperexcitable. Significant physical findings included icteric sclera. Fundi were grade I. Chest was clear to auscultation and percussion. Cardiac examination revealed a grade IV/VI SEM at the apex. The liver was 10 cm. in breadth by percussion. There was no right upper quadrant tenderness. The spleen was not palpable. The bowel sounds were active. Stool was brown and hematest positive. Neurological exam was notable in that the patient had an inappropriate affect, laughing and crying without apparent reason. Asterixis was not present. Neurologic exam was otherwise within normal limits.

Hospital Course

This can best be depicted by assessing the laboratory data on admission, Hgb. 14.6, Hct. 43.7, WBC 27,000 with a shift to the left of 80 neutrophils, 1 band, 19 lymphocytes, half of which were atypical. These atypical cells were compatible with the later diagnosis. A heterophile agglutination test was negative excluding a diagnosis of infectious mononucleosis. The SMA6 was disturbing in
that it showed a CO₂ of 10.8, Na 138, Cl 100, K 5.6, BUN 11 and glucose 72.
Arterial blood gas pCO₂ 28, pO₂ 95, O₂ Sat. 96, HCO₃ 10.1, pH 7.38. This showed
that the patient had two acute primary processes, (1) metabolic acidosis and (2)
respiratory alkalosis. Arterial lactic acid 8.8, Pyruvate 0.153, Prothrombin time
19%. Mono spot test was negative.

After assessing all of these values it became apparent that the patient had
acute fulminant hepatitis. Further lab studies confirmed this diagnosis with lab
data showing LDH 2036, SGOT 5250, alkaline phosphatase 304, bilirubin 10.6,
HAA was positive and the BUN had dropped from 11 to 3.

The patient was started on a conservative regimen of neomycin bowel steriliza­
tion, glucose infusions, placed NPO and given parenteral vitamin K trial. Twelve
hours after admission she was stuporous and asterixis was prominent. Twenty-four
hours after admission she was in a wild delirium alternating with deep obtundation.
In view of her rapid downhill course, an exchange transfusion with 10 units of
fresh citrated whole blood was performed. However, 48 hours after admission in
spite of this treatment the patient progressed into Stage IV hepatic coma. On
August 10, 1973, total body washout was recommended.

Preparation

The entire procedure was performed aseptically in the operating room after
the patient was anesthetized and intubated with an endotracheal tube. The radial
artery was cannulated for constant monitoring of arterial blood pressure. The CVP
and EKG were also closely monitored. A Foley was inserted and attached to a
ureometer. At this point the patient was prepped and draped.

The right internal jugular vein was exposed and cannulated with a 24 Fr.
catheter both distal and proximal to the venous flow (Fig. 1) with the distal
reaching to the right atrium and the proximal ascending 10 cm. cephalad. The
two cannulas were now attached to a “Y” connector and that to a 3/8” venous
extension line (Fig. 1). At the same time the left femoral vein and artery were isolated and the vein cannulated with a 24 Fr. catheter inserted distal to the femoral vein bifurcation. This catheter is attached to a straight ¾” metal connector and that to another ¾” venous extention line which connects parallel to the jugular ¾” venous extension line, coming from the head, with a “Y” connector and those to the ½” common venous line (Fig. 1). This common venous line is filled with normal saline to prevent a vapor air lock and extends off the field. Just before this line enters the oxygenator column a waste drain line is attached with a “Y” which leads to a 20 gallon narrow neck waste container. The venous line is maintained by gravity flow.

The arterial line leaving the Travenol 6LF Oxygenator enters a Travenol Modular pump head then to a heat exchanger and back to the already cannulated femoral artery.

One suction line, Pump No. 1 (Fig. 2), is placed in a 5 gallon sterile container on a separate back table, in which the ice cooled perfusate is contained. This suction line empties into one of the two gas escape vents, to fill and replenish the oxygenator reservoir, making sure to leave the other vent open for excess O₂ and CO₂ to escape.

The other suction line, Pump No. 2 (Fig. 2), is placed in another sterile container which will contain 10 units of whole blood A positive and 4 units of packed cells and 20 mg. of heparin per unit which is used to replace the asanguinous toxic blood removed by the washout perfusate. This suction line empties into the cardiotomy reservoir which should be filled to capacity before washout. A Swank Filter was implanted just before this line enters the base part of the oxygenating column. Clamp proximal to the filter.

Perfusate is in volume equal to at least 3 times the blood volume of the patient (80 cc/kg for adult man, 60 cc/kg for adult female and children).³

Perfusate formula/liter

1) Albumin 25% Sol. 100 cc.
2) Sodium Bicarbonate 50 cc.
3) Ringer’s Lactate 850 cc.
4) Heparine (1000 u.) 1 cc.³

Whole Blood Replacement: type specific and compatible equal to the blood volume of the patient and prepared as:

1) 2/3 whole blood in heparin
2) 1/3 packed cells in ACD³

It is important to keep the Ringer’s Lactate cooled at 5°C in crushed ice until the last possible minute before mixing with the rest of the perfusate to insure minimal hypothermal loss. Now fill your oxygenator reservoir and prime with perfusate by flowing Suction Pump No. 1 (Fig. 2). Fill your clamped cardiotomy reservoir with blood by flowing Pump No. 2. Sterile drapes constantly covered the blood and perfusate containers to insure minimal air contamination. There is no need to pre-cool the patient with a hypothermal blanket before or after washout. Now all is ready and the washout can begin.
Washout Procedure

Clamps are on the two venous extension lines (Fig. 1), one on the waste drain line and one distal to the waste line “Y” on the ½” common venous line to prevent any toxic blood from entering the oxygenating column (Fig. 2). Now the surgeon unclamps the two venous extension lines.

You, the pump technician, release the clamps on the waste drain line leading to the non-sterile waste bucket which starts the exsanguination of toxic blood from the patient. Simultaneously start your arterial pump head which now contains the perfusate at 35 cc/min/kg and increase to 50 cc/min/kg or more using rate of venous effluent as a guide for infusion rate along with CVP pressures. Your arterial pressure will soon become 50-60 mm Hg. pump mean pressure. Constantly replenish your oxygenator reservoir with perfusate by flowing your No. 1 Suction pump (Fig. 2) at the same rate as your Arterial Pump. At this point your core temperature will have dropped to below 30°C and coarse fibrillation will have begun. The washout is continued until the hematocrit in your venous line is unreadable and your patient’s esophageal temperature has dropped to 20 to 25°C. Now the EKG parameter is flat and the patient’s respirations are continually mechanically maintained. The bypass heat exchanger is in the cooling mode as cold as possible. The washout should continue until the venous effluent is visibly light pink and almost totally translucent. At this point “zero hematocrit” has been reached, about 4 to 6 minutes from the start of the washout. Note that this light color pink is seen at both the jugular and femoral venous cannulation sites.

Now all your reserve perfusate should be exhausted and your oxygenator reservoir bag reaching half full. Slow down your Arterial Pump flow and open the clamp on the Cardiotomy Reservoir (Fig. 2) which contains the fresh whole blood and packed cells. Not until now do you turn on your 100% oxygen. Flow it at a sufficient rate to oxygenate at a 2:1 ratio. At this point put your heat exchanger in the warming mode, noting not to exceed 5°C between core blood temperature and rewarming water mixing valve thermometer to insure no free oxygen is liberated from the blood. Constantly replenish your cardiotomy reservoir by flowing your No. 2 Suction Pump at the same rate as your arterial pump is flowing. Increase your arterial pump back to 50 cc/min/kg if possible. Note that your washout perfusate is now reversing and starting to darken your venous drain line. When all of your whole blood reserve is exhausted in the sterile container on the back table and the cardiotomy reservoir is near empty, you will now notice your exsanguinous venous waste line is darkened to near bypass normal at 25 to 30 hematocrit. Note again that both the jugular and femoral vein cannula sites have darkened equally. Slow down your arterial flow to 35 cc/min/kg and unclamp the common venous line distal to the “Y” of the waste drain line and clamp the waste drain line leading to the waste container simultaneously. Now you are on a conventional bypass and flow should be increased to a rate which will balance your venous return, 50 cc/min/kg, maintaining an arterial pressure of 50 to 70 mm. Hg. Continue to rewarm and at 30 to 34°C ventricular fibrillation should reverse, if not defibrillate until normal sinus reappears. After a satisfactory temperature has been reached and the normal sinus rhythm is stable, you are ready to come off bypass in the conventional method.
When breaking down equipment at the end of the case, take every precaution to avoid contact with the waste blood. Recommend use of disposable drapes and scrub suits. Before covering the waste blood and washout perfusate, pour a gallon of chlorox or cidex in the container and cover tightly with screw cap. Also avoid contact with the old urine and any fecal material for your own personal safety. The entire TBW procedure should not exceed 65 to 75 minutes in duration.

Post TBW

Following the procedure the patient was comatose and supported on a Bird respirator. Within 48 hours her vital signs stabilized and she showed progressive improvement neurologically, regaining complete consciousness 62 hours after TBW. BUN showed a rise from 4,8,15,26. Bilirubin subsequently dropped back down to 1.3.

The liver function studies improved greatly and at discharge the SGOT was 47, LDH 246 and the alkaline phosphatase was 142. The Australian antigen which originally was positive reverted back to negative on four determination. It was felt at the time of discharge that the patient’s liver status was remarkably improved, although a follow-up liver biopsy in several months would be of value in determining the amount of regeneration that the liver had undergone.

Discussion

Our immediate post TBW serological findings and six month post discharge finding of Hgb 13.1, WBC 6,700, Pro Time 100%, alkaline phosphatase 80, SGOT 41, and LDH 235 prove that a marked improvement in liver functions was achieved.

Today this woman is back working and carrying on normal everyday tasks without difficulty.

Hepatocellular regeneration is a function dependent upon a nonhepatic metabolic overload. We believe this was accomplished by TBW, therefore providing time for the liver to recover and hold pace with everyday metabolic stresses.

REFERENCES