Colloidal Osmotic Pressure in Chronic Subdural Hematoma

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Abstract

The colloidal osmotic and hydrostatic pressure of chronic subdural hematomas were measured with a manometer in 35 patients. Manometric measurement showed an increase in the hematoma fluid corresponding to 5% of the total hematoma volume. The maximum colloidal osmotic pressure of the hematoma fluid, as measured with a transducer, was 23 ± 12 mmHg (mean ± standard deviation). Hemorrhage occurs continuously or intermittently within a subdural hematoma, accompanied by red cell hemolysis and hemoglobin degradation. The colloidal osmotic pressure within a subdural hematoma is higher than that in plasma or cerebrospinal fluid, so that the hematoma is able to absorb water through semipermeable membranes or capillaries. However, since the absorption force can transport only 5% of the hematoma volume, the colloidal osmotic pressure is a contributory process rather than a primary etiologic factor.

Key words: colloidal osmotic pressure, chronic subdural hematoma, subdural effusion

Introduction

In 1932, Gardner\(^1\) published his hypothesis that spinal fluid is drawn into a hematoma through the semipermeable arachnoid membrane and adjacent cyst wall by the osmotic tension of the blood proteins within the cyst. Intracystic blood disintegrates slowly over a period of months, causing an increase in the osmotic pressure and progressive dilution of the blood within the hematoma.\(^2\)\(^3\) Using the freezing point depression method\(^4\) and an oncometer,\(^5\) Weir found no significant differences in the osmolalities of hematoma, venous blood, and cerebrospinal fluid. The mechanism of osmotic pressure regulation in chronic subdural hematoma remains controversial. With the recent advancement in manufacturing semipermeable membranes, the colloidal osmotic pressure in chronic subdural hematoma could be easily measured and clarified.

Materials and Methods

Our study included 32 patients with chronic subdural hematoma and three with subdural effusion. Through a small, trephination-like craniotomy, hematoma specimens were carefully aspirated through the dura with a number 18 needle. Care was taken to avoid contamination with peripheral blood.

We obtained bags made of a collodion semipermeable membrane and having a capacity of 7 ml (Sartorius Membrane Filter Co., West Germany). The bags had a pore size of either 8 or 15 nm and a maximum permeability corresponding to 12,400 or 25,000 molecular weight proteins, respectively. For each measurement a bag was filled with hematoma fluid, corked, and connected to a glass pipette (Fig. 1 left). Physiological saline, lactate, and Elliott's B solution were used as the media. A bag was placed in 350 ml of each medium at room temperature and the height of the hematoma fluid in the glass pipette were periodically observed. The capillary effect of the pipette (inner diameter, 1 mm), which was approximately 30 mmH\(_2\)O, was subtracted from each measured height. Pressure data were also obtained by recording the colloidal osmotic pressure with a pen-recorder (model CDR-12A, Toa Denpa, Japan) connected to a pressure transducer (model MD-4, Nihon Koden, Japan) (Fig. 1 right).
Fig. 1 For measurement of colloidal osmotic pressure, a collodion bag with a 7-ml capacity was filled with hematoma fluid, corked, and placed in 350 ml of medium in a large bottle. The collodion bag was connected to a glass manometer with a 1-mm inner diameter (left) or a pressure transducer (right).

**Results**

I. Manometric measurement of colloidal osmotic pressure in chronic subdural hematoma

The colloidal osmotic pressure exponentially increased to 480 ± 75 mmH2O within 3–5 hours, and thereafter stabilized or slightly declined (Fig. 2). Averaged maximum heights of the hematoma in the manometer was 480 mmH2O, so that the fluid volume in the glass pipette was 353 mm³ [(1/2)²π(480 – 30) = 353] due to colloidal osmotic pressure. This volume constituted 5% of the hematoma fluid in the bag.

II. Colloidal osmotic pressure in chronic subdural hematoma and subdural effusion determined by pressure transducer

When physiological saline, lactate solution, or Elliott’s B solution was used as the medium, the colloidal osmotic pressure of the hematoma fluid increased for 30 minutes and then gradually decreased (Fig. 3A–C). When the collodion bag filled with hematoma fluid was placed in 20% mannitol, the pressure initially decreased, began to slowly increase after 30 minutes, returned to zero after 7 hours, and reached a plateau of 18 mmHg between 12–15 hours (Fig. 3D). The maximum colloidal osmotic pressure in subdural effusion was 5 ± 2 mmHg; it returned to the initial level within 30 minutes (Fig. 3E).

The maximum colloidal osmotic pressures of hematoma, whole blood, and fresh plasma were 23, 17.3, and 20 mmHg, respectively (Fig. 4A, C, F). When hematoma fluid or cell components of blood were stored in a refrigerator for 30 days, their colloidal osmotic pressure increased two- to threefold.
A tear in a bridging vein near the sagittal sinus following minor head injury can cause slight hemorrhage into the subdural space. Shortly thereafter a clot forms and, for about 3 weeks, it is encased in a neomembrane composed of connective tissue. The presence of foreign substances, such as subdural clot, effusion, or pus, may contribute to the formation of the neomembrane. The neomembrane produces plasminogen-activator to absorb the foreign substance, because the lymphatic system does not reach into the intracranial space. The plasminogen-activator converts plasminogen to plasmin, which degrades fibrin and fibrinogen into their degradation products and thereby liquefies the clot. Chronic subdural hematoma develops if too much plasminogen-activator is produced and local fibrinolysis and hemorrhage continue to occur.

Red cells in the hematoma fluid are hemolyzed and the large hemoglobin molecules that are liberated in increase the colloidal osmotic pressure of the fluid. If fresh blood, plasma, or interstitial fluid, enclosed by a semipermeable membrane, is placed into water, the osmotic pressure determined by electrolytes, so-called crystal osmotic pressure, becomes 5092 mmHg. The crystal osmotic pressures are similar in subdural hematoma fluid, cerebrospinal fluid, plasma, and fresh blood. However, the colloidal osmotic pressure of fresh blood is 17 mmHg (Fig. 5), which is small in comparison with the crystal osmotic pressure. The capillary wall is permeable to water as well as to all of the electrolytes and other small molecules. In contrast, capillaries are impermeable to charged, high molecular weight proteins, and electrolytes exert no significant osmotic effect across the capillary wall.

Starling theorized that most of the colloidal osmotic pressure is exerted across the capillary wall. Subdural hematoma fluid neighbors plasma in the many capillaries of the outer membrane, and is separated from cerebrospinal fluid by the inner and arachnoid membranes. Therefore, the colloidal osmotic pressure imbalance draws water in plasma and cerebrospinal fluid into the hematoma cavity. Thus, after a latent interval, subdural hematomas grow and produce symptoms because of high colloidal

Discussion

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Fig. 4 Colloidal osmotic pressure of hematoma fluid and blood components against physiological saline.

(Fig. 4A, B, D, E). However, when plasma was refrigerated for 30 days, the colloidal osmotic pressure changed only slightly (Fig. 4F, G). When red blood cells were hemolyzed with saponin, the colloidal osmotic pressure greatly increased (Fig. 4H). The maximum colloidal osmotic pressure of hematoma fluid was 23 ± 12 mmHg, and that of subdural effusion was 5 ± 2 mmHg (Fig. 5).

Fig. 5 The maximum colloidal osmotic pressures. Circles denote the mean and bars represent the standard deviation.
osmotic pressure.\(^{1}\) The albumin:γ-globulin and albumin:total protein ratios in the hematoma fluid are considerably higher than their corresponding ratios in serum, so that fluid accumulation is a consequence of effusion through irritated or damaged capillary walls.\(^{2}\)

Colloidal osmotic pressure in hematoma fluid can transport only 5% of the hematoma volume. Moreover, the hematoma fluid is diluted within a few hours and the osmolalic balance among hematoma fluid, cerebrospinal fluid, and plasma is restored. Mannitol can be used to lower osmotic pressure,\(^{3}\) but it must be administered repeatedly because its effect is temporary (Fig. 3D). However, daily hemorrhaging contributes 6.7% of the hematoma content.\(^{6}\) With continuous or intermittent hemorrhage, the degradation of hemoglobin and fibrin or fibrinogen keeps the colloidal osmotic pressure high. Therefore, continuous hemorrhage can be considered the essential cause of chronic subdural hematoma; it is the single most important factor in the increase of hematoma volume and production of symptoms. High colloidal osmotic pressure per se does not cause chronic subdural hematoma, but plays a limited role in the sequelae of continuous hemorrhage.

References

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