Delirium and Hyponatraemia in the Elderly Post-Surgical Patients

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The authors of this article [1] mentioned in table 1 that out of a total of 27 patients, who suffered delirium postsurgical, had Hepatobiliary + Pancreas = 9, Upper GI = 7, Colorectal = 5, Vascular = 3, Others - general surgery = 2 and orthopaedic 1. Of whom there were 11 patients who had hyponatraemia (HN), 5 had fluid overload and 6 had hyperglycaemia. All these complications are induced by excessive volumetric overload particularly 5% Dextrose fluid during surgery. Delirium occurred during the first postoperative day in most patients.

<table>
<thead>
<tr>
<th>Gender</th>
<th>Male = 17, Female = 10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age range</td>
<td>Mean 82 (66-94)</td>
</tr>
<tr>
<td>Type of surgery</td>
<td>Hepatobiliary + Pancreas = 9, Upper GI = 7, Colorectal = 5, Vascular = 3, Others - general surgery = 2, Ortho = 1</td>
</tr>
<tr>
<td>Onset of delirium (from postop day)</td>
<td>0 = 2, Day 1 = 17, Day 2-4 = 4, Day 10 and beyond = 4</td>
</tr>
<tr>
<td>Electrolyte abnormalities</td>
<td>Hyponatraemia = 11, Hyperglycaemia = 6, AKI = 6, Acidosis = 4, Others = hypokalaemia, hypocalaemia, hypo PO4, No electrolyte abnormalities = 3</td>
</tr>
<tr>
<td>Medical issues contributing to delirium</td>
<td>Constipation (no BO ≥ 2days) = 26, Fluid overload = 5, Poor oral intake = 5, NSTEMI = 5, Hb drop &gt;2g = 4</td>
</tr>
<tr>
<td>Drugs</td>
<td>15 had been given drugs with anticholinergic properties- maxolon, anarex, cough syrup, antihistamines, pethidine. Alcohol withdrawal = 1</td>
</tr>
<tr>
<td>Pain</td>
<td>Inadequate pain control = 21, No complaints of pain = 6</td>
</tr>
<tr>
<td>Sepsis</td>
<td>Related to surgery or hospital acquired = 17</td>
</tr>
<tr>
<td>Indwelling urinary catheter</td>
<td>Present = 16, 3 were inserted for ARU</td>
</tr>
<tr>
<td>Hx of dementia</td>
<td>Positive in 18, 9 not known to have dementia</td>
</tr>
<tr>
<td>Depression</td>
<td>Positive = 2 both in presence of dementia.</td>
</tr>
<tr>
<td>Premorbid Instrumental activities of daily living</td>
<td>Independent = 5, Assisted = 22</td>
</tr>
<tr>
<td>Premorbid Activities of daily living</td>
<td>Independent = 10, Assisted = 17</td>
</tr>
<tr>
<td>Types of delirium</td>
<td>Hypoactive delirium = 4, Hyperactive delirium = 6, Mixed hypo and hyperactive = 17</td>
</tr>
<tr>
<td>Restraint use</td>
<td>Physical restraint = 16, Chemical restraint = 3, Physical+ chemical restraint = 2</td>
</tr>
</tbody>
</table>

Table 1: Postoperative delirium.
Delirium and Hyponatraemia in the Elderly Post-Surgical Patients

The authors did not mention how this HN was managed and did not link the two conditions of delirium and HN together. Here I demonstrate that not only HN causes delirium among wide range of cerebral features but it is also induced by sodium-free fluid volumetric overload (VO1), such as 5% Glucose solution. Sodium-based fluids volumetric overload (VO2) induce fluid overload without HN markers. I shall also mention that there is an effective treatment for the condition, namely hypertonic sodium therapy (HST) of 5% NaCl or 8.4% NaCO₃.

Incidence and prevalence of HN

Hyponatraemia in which serum sodium is < 130 mmol/l is one of the most commonly encountered biochemical abnormality in clinical practice. Batuman., et al. [2] found that HN affects 33% of patients receiving parenteral nutrition (range 114 - 129 mmol/l). In a prospective study involving surgical patients, Chung., et al. [3] found post-operative HN to affect 23.1% cardiovascular, 18.9% Gastrointestinal and biliary and 92% renal transplantation patients. Anderson., et al. [4] found that 1 - 2.5% of hospitalized patients had HN and was associated with a 60-fold increase in fatality.

The morbidity and mortality of acute HN is dependent on the level to which serum sodium drops and the rate of this fall. An acute reduction in serum sodium concentration of > 20 mmol/l or to a level below 120 mmol/l is usually serious but may be asymptomatic if time is allowed for adaptation [5] or osmolality is maintained. At levels less than 130 mmol/l, HN causes delirium and confusion.

The cerebral features of HN

Manifestations of the cerebral nervous system are recognized early in the conscious patient who received regional anaesthesia. Mild signs such as confusion, delirium, disorientation, restlessness, apprehension, burning all over the body, irritability, lethargy, headache, muscle twitching, nausea and vomiting are commonly observed early features. Visual disturbance of transient or permanent bilateral blindness have also been reported [6].

The patient may progress to severe signs such as convulsion, coma with fixed dilated pupils and bizarre types of paralysis which mimic cerebro-vascular infarctions. Coma and paralysis may be the presenting signs in patients who had general anaesthesia from which he fails to recover. Intensive investigations may be embarked upon causing delay in treatment. Not uncommonly the diagnosis may be missed leading to permanent brain damage with coma or death [7,8].

New discoveries on HN

Hyponatraemia (HN) is a condition that has many names [8] reflecting its multiple presentation masks that have eluded researchers of physicians, surgeons, urologists and anaesthetists over 6 decades. Most studies have repeatedly missed volumetric overload as the real patho-aetiology. My initial thoughts and concepts were reported in BMJ long ago [9,10]. The new provocative statements have proved remarkably correct via reported studies [11-13]. Such evidence is summarized and clarified here.

Serious HN affects men, women and children. It affects postsurgical patients particularly during or after endoscopy. All cases are traceable to iatrogenic complication of fluid therapy. Data suggest large volumetric overload (VO) of sodium-free fluid type (VO1), worsened by the wrong therapy using isotonic sodium-based fluid (VO2) that may mask HN and cause death. All reports lack a mention of exact VO1 causing HN. Most if not all prospective studies but one [11] has fallen short of identifying and quantifying the VO insult. The most well-known model of HN is the infamous transurethral resection of the prostate (TURP) syndrome, induced by large fluid gain during any endoscopic surgery [11]. It is the best model to reveal overlooked issues and highlight new concepts that can easily be applied to all cases of HN once the importance of VO/Time (VO/T) in pathogenesis is realized. The work of pioneers [14,15] and current authorities such as Professors Arieff, and Ayos [7,8] and Hahn [16] has markedly contributed to the understanding of HN, leading to my new concepts and discoveries.

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The TURP syndrome is induced by the absorption of any sodium-free fluid of > 3.5L in < 1h during any endoscopy surgery. Such fluids include Glycine, Glucose, Mannitol and Sorbitol. It presents acutely with vascular hypotension shock that is usually mistaken for one of the recognized shocks and treated with further volume expansion. Fluid gain of 3.5 - 5l in 1h can be serious or lethal. It causes severe systemic signs of multiple vital organ dysfunction/failure (MVOD/F) syndrome in both humans and animals [14,15]. Acute renal failure (ARF) occurs paradoxically which prevents excretion of sodium and urine. So HN is dilutional. The severity is directly proportional to VO but inversely to T.

Fluid type determines the changes of serum solute contents and presentation masks: Pure water causing haemolysis, VO1 is marked with HN nadir proportional to severity [9-13] and VO2 such as saline, plasma or substitutes though better tolerated induce the signs without HN marker, affirming the concept of VO/T as cause of shock [17-21]. About 3.5l is the normal daily fluid intake but causes signs when infused over 2 - 3 hours and is a serious gain in < 1h.

Although the systemic bizarre illusive signs of severe TURP syndrome and HN are well documented in case reports, it is extremely difficult to relate to VO/T even on monitored measure of gained volume. This is because the complex signs of cardiovascular disturbance of shock and MVOD/F syndrome of the TURP syndrome have wide severity range up to arrest/death and many presentation masks- each having enormous differential diagnosis. Hence, seen in the complex surgical setting, signs are wrongly attributed to known causes of shock, respiratory distress, coma, and/or renal and heart failure among MVOD/F signs occurring in any combinations. Of well documented postoperative presentation masks is shock to surgeons and coma to physicians. More important, neither the concept nor mechanism of VO/T causing shock by disturbing capillary dynamics are recognized despite explaining pathology of HN, TURP syndrome and link with MVOD/F [17-21].

Adding to the difficulties, the concept of VO/T as cause of shock is paradoxical to the received concept of treating all shocks indiscriminately with vascular expansion. The over incriminated blood loss and sepsis have no role in pathogenesis. Authors [16] have validated our volumetric measure of irrigant gain of 3.5L and blood loss of < 0.4l. So, VO is responsible for this vascular shock. Volumetric overload shocks (VOS) [17-21] and the proof and reasons why Starling’s law is wrong [20], providing the correct replacement of hydrodynamics of the porous orifice (G) tube have been reported.

Once the new concept and discovery are understood and accepted, resolving the puzzles of HN and MVOD/F syndrome and finding a successful readily available therapy for HN should be easy. Using HST of 5% Sodium Chloride or 8.4% Sodium Bicarbonate is life-saving [17-19]. Infusing 200 ml/10 minutes, repeated under clinical and biochemical monitoring corrects HN without worsening VO [4,5,11-15]. It acts as diuretic recovering the patient from his shock and coma while excess water and sodium is passed as urine. My colleague who tried this therapy called his saved patient “Lazarus”- the one who came back from the world of dead. It should be tried as primary therapy for postsurgical delirium.

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