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UNDERSTANDING *OJODUSHTI* IN RELATION TO PATHOPHYSIOLOGY OF SHOCK

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Abstract

Ojas is the vital force that sustains life formed as an essence of all the dhatu. The decline of *ojas* is an inevitable sequel of any disease. Shock which is a condition that results from reduced perfusion to tissues and organ dysfunction is relatable to the 3 stages of *ojakshaya* as mentioned by Susrutha. *Sannipatajwara* which is also called as *Hritaujas* and its classification into 13 types can be aligned to the stages of different types of shock. The contradiction of *visha* and *ojas* and the importance of the *trimarma* in the pathology of ojodushti are also significant in understanding and managing *ojodushti*. The emergency management system has to focus on the concept of *ojodushti*.

Keywords – *Ojodushti*, different types of shock, *Sannipata jwara*

BACKGROUND

A severe mismatch between the supply and demand of oxygen is the common feature of all types of shock.[1] Modified shock index is a significant clinical tool in comparison to heart rate and systolic blood pressure in assessing mortality of emergency department patients.[2] Similarly, amongst all parameters to assess *arishta lakshanas* and *sadhya – asadhyaatha* in ayurveda, *ojakshaya* is an important demarcation. Emergency management in Ayurveda always aims at *ojo-vardhana* to enable body to activate its self-support mechanism in catastrophic medical situations. This article thus opens avenues to the discussions about similarity in clinical features in *ojodusht*i and shock.

INTRODUCTION

Ojas is derived from '*ubj' dhatu* which means to hold and *'asun' pratyaya*, altogether meaning shine or lustre. *Ojas* is the finest essence formed from all the seven dhatus.

Ojas is present in dormant form in sperms *(shukra)* and ovum *(shonita)* of parents.[3] After conception, it is manifested as the first body component in intra-uterine life.[4] It is also termed as the basic constituent of embryo *(garbha sara)* and nourishes it.[5] After birth, it is formed as the supreme essence during the metabolism of each dhatu.[6] Some scholars opine that it is the essence of reproductive tissues *(sara* of *shukra dhatu)*.[7] *Sharangadhara* considers it as a metabolic by-product (*upadhatu*) of reproductive tissues *(shukra),* whereas *Vagbhata* opines that it is a metabolic waste product of reproductive components *(shukra dhatu)*.

Analogy

Just as ghee is present in a dormant form in milk and can be prepared after suitable processing, *ojas* is present in every body tissue (*dhatu*) in quiescent form and is manifested in its supreme quality after its proper metabolism.[8] Hence, it is clear that *ojas* is formed as a purest secondary component after the metabolism of primary body components *(dhatu)*.

Location

Ojas is circulated all over the body with *rasa* and *rakta dhatu* through channels *(ojovaha sira)*. These channels originate from the heart.[9] *Bhela* has opined about twelve sites as the location of *ojas*. These include seven *dhatus*, three *malas*, and two *doshas (pitta* and *kapha).*

Types & Quantity

Ojas is present in two forms: *para* and *apara*][10]

1. *Para ojas* is associated with the vitality of life and is located in the heart. Its quantity in the body is measured as eight drops (*bindu*) and is generally stable in the body in an equilibrium state. Destruction of this component can cause death.
2. *Apara ojas* is formed during the metabolism of each *dhatu* is present all over the body. Its quantity in the body is measured as half *anjali* and manifests in the form of the essence of all *dhatus (sara)*.[11]

Functions

1. Functioning of all external and internal senses including the mind, thereby responsible for the sensory functions and perception of knowledge.[12]
2. To maintain vitality and sustain the integrity of life.
3. Providing nutrition and enhancing the immune strength of the body.

Physical characteristics of *Ojas*

The physical characteristics of *Ojas* are shown in table 1.[13]

Table 1: Physical characteristics of *Ojas*

Sr.No	*Parameter*	*Characteristics*
1	Touch *(sparsha)*	Cold *(sheeta)*, unctuous *(snigdha)*, soft *(mrudu)*, smooth *(shlakshna)*, slimy *(mrutsna),* sticky *(pichchila)*
2	Visible form *(rupa)*	Pure white *(shuddha shukla)*, slightly red yellowish *(ishat rakta-pita, lohit pita),* resembling to ghee-clarified butter *(sarpi varna)*
3	Taste (*rasa*)	Sweet *(madhura)*, like honey *(madhu rasa)*
4	Smell (*gandha*)	Parched cereal (*Laja gandha*)
5	Others	Heavy (*guru*), cherishable (*prasanna*), bulky (*bahala*), stable (*sthira*), cool (*somatmaka*), mobile (*sara*), clear or distinctly formed (*vivikta*)[5]

Pathological aspects

Causes of vitiation of *Ojas:*

Ojas is affected due to the following causes:

- Any injury or trauma (*abhighata*)
- Malnourishment or impaired metabolism (*kshaya*)
- Anger (*kopa*)
- Excessive thinking (*dhyana*)
- Grief (*shoka*)
- Vigorous physical work or exertion (*shrama*)
- Voracious appetite (*kshudha*)[14]

Pathophysiology

Due to these causes, the *vata* and *pitta dosha* are aggravated. This leads to the quantitative and qualitative depletion of *ojas*. It is then easily displaced from its prime seat - *hridaya*.

The abnormalities in quantity and quality of *ojas* are specifically observed in three stages.[15]

1. Displacement of Oja from its own site (*Oja visramsa*)
2. Qualitative deterioration (*Oja vyapat*)
3. Depletion of quality and quantity (*Oja kshaya*)

General clinical features:

- Fearfulness or phobia
- Weakness
- Fatigue especially at its sense and motor organs
- Altered skin complexion
- Lack of self-confidence
- Dry and rough skin
- Emaciation of body.[16]

The three stages are increasingly severe and harmful leading to death. The signs and symptoms of each state are shown below in table 2.

Table 2: Clinical features of deviation in *Ojas*[17]

Sr.No	*Oja visramsa*	*Oja vyapat*	*Oja kshaya*
1	Feeling of looseness and displacement in joints or synapses (*sandhi vishlesha*)	Feeling of stiffness and rigidity in body (*stabdha guru gatrata*)	Fainting or unconsciousness (*murchcha*)
2	Fatigue (g*atra sadanam*)	Unstable swelling due to vitiation of Vata dosha (*vata shopha*)	Emaciation and muscle wasting (*mamsa kshaya*)
3	Migration of vitiated dosha to other sites (*dosha chyavanam*)	Abnormal change in normal complexion (*varna bheda*)	Confusion (*moha*)
4	Impairment and blockage of all functions in body and mind (*kriya sannirodha*)	Lassitude (*glani*)	Delirium (*pralapa*)
5	-	Drowsiness (*tandra*)	Death (*marana*)
6	-	Excess sleepiness (*nidra*)	-

A careful evaluation of the location and functions of *ojas* and the observations of *Acharya Susruta* that three stages of *oja kshaya* can lead to death aligns with the emergency medical condition of shock. Shock is a life-threatening condition of circulatory failure that causes an imbalance between cellular oxygen supply and demand resulting in organ dysfunction. It has several causes including hemorrhage injury, burning, diarrhoea, myocardial infarction, bacterial infection, spinal cord injury, anaphylaxis and structural obstruction like aortic stenosis. It has two stages - reversible and irreversible. In the reversible stage, the condition can be improved by compensatory mechanisms such as tachycardia, increased vasoconstriction, capillary fluid shift and stimulation of RAAS. The later stage is irreversible where compensatory mechanisms fail to improve the patient's conditions. In severe conditions of hypovolemic shock, effective circulatory volume loss leads to tissue hypoperfusion, tissue hypoxia especially to vital organs and ultimately renal failure. It is important to recognize promptly as it may result in multiorgan failure and subsequent death. This condition is strikingly similar to *oja kshaya*. This article thus aims to explore this prospective relationship between *ojodusti* and shock.

TYPES OF SHOCK FROM THE PURVIEW OF STAGES OF *OJAKSHAYA*

Shock is a syndrome in which there is inadequate tissue perfusion associated with a relative or absolute decrease in cardiac output. Regardless of underlying pathology, shock gives rise to systemic hypoperfusion which can be caused either by reduced cardiac output or by reduced effective circulating blood volume. The end results are hypotension, impaired tissue perfusion and cellular hypoxia. The new classification comprises of four main categories:

- Hypovolemic shock
- Distributive shock
- Cardiogenic shock
- Obstructive shock.

Of these, hypovolemic shock is divided into four subcategories and distributive shock into three. Obstructive shock has been given a category of its own. Although this nomenclature and classification is schematic and there is some overlapping between the main groups, these four main groups can be basically assigned to four organ systems, owing to differences in their pathogenesis and pathophysiology.

The detailing of these clinical features has aroused curiosity regarding the current day understanding of *sannipatha jwara*. Any pathological condition causing diminution of *ojas* due to *tridosha prakopa* is cited as *hritaujasa/ abhinyasa* under *sannipata jwara. Bhela* has detailed about this by categorising it into 13 types – which has approximate parallels with different types of shock. They are:

- *Sandhikan*
- *Anthakan*
- *Rugdahan*
- *Chithavibhraman*
- *Sheetangan*
- *Tantrikan*
- *Kanthakabjan*
- *Karnakan*
- *Bhagnanetran*
- *Raktakshteevi*
- *Pralaapakan*
- *Jihwakan*
- *Abhinnyasam*

Of the numerous clinical signs under each type of *sannipatha*, a clinical comparative purview of them with those observed in types of shock are evaluated.

Hypovolemic shock

Hypovolemic shock is a condition of inadequate organ perfusion caused by loss of intravascular volume, usually acute. The result is a drop in cardiac preload to a critical level and reduced macro- and microcirculation, with negative consequences for tissue metabolism and the triggering of an inflammatory reaction.

Hypovolemic shock is divided into four subtypes:

- Hemorrhagic shock
- Traumatic hemorrhagic shock,
- Hypovolemic shock in the narrower sense
- Traumatic hypovolemic shock

The characteristic feature of both, hemorrhagic and traumatic hemorrhagic shock is bleeding. However, differences exist between the two subcategories in terms of the extent of soft tissue damage. Clinically, acute bleeding from an isolated injury to a large blood vessel triggers a critical drop in circulating blood volume; massive loss of red blood cells intensifies the tissue hypoxia. *Rasa kshaya lakshanas* clearly demarcates close parallels with these features including insipidity (*asya vairasya*), palpitation (*hrillasa*), heaviness (*gaurava*), drowsiness (*tandra*), feeling of darkness before eyes (*tama*), paleness (*pandutva*), malaise (*sada*) and scragginess (*krisha*). In addition, *rasavaha srotas* has its *moolasthana* in *hridaya* and *dasha dhamanis*; which is very similar to the major vessels carrying life sustaining humor - blood in the body. All these conclude

towards the possibility of *rasa* playing a vital role in sustaining *prana* and *ojas*; who also have their *moolasthana* in *hridaya* and are significant in nurturing life.

Traumatic hemorrhagic shock is distinguished from hemorrhagic shock by the additional presence of major soft tissue injury which aggravates the shock. Diffuse bleeding, hypothermia (especially = 34 °C), and acidosis leads to life-threatening coagulopathy. *Bhugnanetran*, a type of *sannipatha* mentioned in *Bhela* Samhitha enlists similar features to that of acidosis and hypothermia taken together, like – hypothermia (*jwarabala apachaya*), feeling tired and listless (*smruthi shoonyatha*), dyspnoea (*swasana bhugna*), delirium (*pralapa*), loss of consciousness (*bhrama*) and tremor (*kampa*). Similarly, as in *sheethagatran*, cold extremities and clammy body (*hima sadrusha shareera*), tremor (*vepathu*) are also seen in these clinical scenarios. The soft tissue injury leads to post-acute inflammation, further reinforcing the prospective analogy to swelling (*sopha*) in *bhugnanetra sannipatha*. Interestingly, both *bhugnanetra* as well as sheethagatra have poor prognosis and possibility of immediate death just as traumatic hemorrhage is life threatening.

Hypovolemic shock in the narrower sense arises from external or internal fluid loss coupled with inadequate fluid intake. It can be caused by hyperthermia, persistent vomiting and diarrohea (e.g., cholera), or uncompensated renal losses (e.g., diabetes insipidus, hyperosmolar diabetic coma). *Bhela* has mentioned similar features in *sheethagatran* with persistent vomitting and diarrohea (*chardi, atisara*) and in *raktakshteevi*, he has also added intense thirst (*trisha*), loss of consciousness (*moha*) and hyperthermia (*jwara*). In *rugdahan*, escalating thirst inspite of water consumption (*niranthara thrishakara*) has been mentioned. Similarly, increased thirst in *taanthrikan* – (*prabhutha pipasaakula*), tiredness (*prabhutha tandra*), loss of energy (*shayanamisham*) are all points to ponder over in relation to hypovolemic shock.

Distributive shock is a state of relative hypovolemia resulting from pathological redistribution of the absolute intravascular volume. The three subtypes are septic, anaphylactic/anaphylactoid and neurogenic shock.

Septic shock is characterized by endothelial dysfunction, which leads to dysregulation of vascular tone resulting in vasodilation, impaired distribution, and volume shifting in the macro- and microcirculation. It is a mixed form of a variety of pathologies (hypovolemia, vasodilation, impaired cardiac function, and mitochondrial dysfunction) and is usually associated with complex coagulopathies. Non-infectious disease involving extensive mediator activation (e.g., acute pancreatitis) may lead to a clinical presentation similar to that of septic shock. The pathophysiology and pathogenesis of toxic shock syndrome (TSS) are related to those of septic shock. TSS is characterized by fever, severe hypotension, and skin rash as the main symptoms. *Sandhikan* in *Bhela Samhitha* resembles in some clinical features such as premonitory stage of fever (*jwara poorvaroopa*), abdominal colic (*poorvaroopakritha shoola*) - just as seen in acute pancreatitis and other abdominal emergencies, emaciation (*shosha*), pain due to *vata* (*vata prakopa*), loss of strength (*balahani*) and hyperpyrexia (*tapa*). This matches with many features mentioned under non infectious medical conditions causing septic shock. *Anthakan* has symptoms such as burning sensation (*daha*), dullness (*paritapa*) and loss of consciousness (*moha*) - which may occur due to uncurbed rise in core temperature, which is comparable to toxic shock syndrome.

Anaphylactic shock is characterized by massive histamine-mediated vasodilation and maldistribution with a shift of fluid from the intravascular to the extravascular space. The clinical presentation varies greatly individually, with skin manifestations, abdominal symptoms, or respiratory symptoms prominent amongst them. The respiratory manifestations aligns with some medical scenarios like *anthakan, rugdahan, sheethagatran, karnakan, bhugnanetran, raktakshteevi* and *jihwakan*. In addition, rakta prakopaja lakshanas in Charaka sutrasthana outlines a spectrum of dermatological complaints including – kushta, visarpa, pidaka, guda-medhra-asya-paka and abdominal symptoms like pleeha vrdhi, vidradhi and gulma. Dadru, charmadala, pama, kotha and asramandala are also comparably noticed.

Neurogenic shock is a state of imbalance between sympathetic and parasympathetic regulation of cardiac action and vascular smooth muscle. The dominant signs are profound vasodilation with relative hypovolemia while blood volume remains unchanged, at least initially. Numerous ayurvedic clinical scenarios with ojakshaya also have this aforesaid regulation at stake.

Cardiogenic shock

Cardiogenic shock is primarily a disorder of cardiac function in the form of a critical reduction of the heart's pumping capacity, caused by systolic or diastolic dysfunction leading to a reduced ejection fraction or impaired ventricular filling. The main symptoms of cardiogenic shock are agitation, disturbed consciousness, cool extremities and oliguria whereas obstructive shock is a condition caused by the obstruction of the great vessels or the heart itself. The symptoms are nonspecific and is characterized by the compensatory autonomic response in the form of tachycardia, tachypnea, oliguria, and altered consciousness. Hypotension may be quite modest initially and this can lead to underestimation of the clinical situation.

The insight Acharyas had while formulating these samhithas can be perceived while observing that several symptoms under *sannipatha have* mirrored the psychiatric ideals of these systemic medical conditions. Many of those are consistent with compensatory autonomic responses – inappropriate singing, dancing and talking (*geethanrtha pralapi*) as in *chithavibhraman, bhugnanetran* and *pralapi*. Disturbed consciousness can be recognised as similar to *moha* in *raktakshteevi* and *bhugnanetra*.

DISCUSSION

Acharya Charaka referred to *ojas* as one of the *pranayatanas* (locations of *prana*), suggesting that if *ojas* is lost, life will also come to an end. Acharya Vagbhata also asserted that the loss of *ojas* certainly results in the destruction of the individual, while its existence sustains life. Charaka introduced the idea that this *ojas* exists in the torso of the body. The *marma* points were thus identified and were found to be closely linked with ojas. These *marma* points in the trunk are more significant than those in the limbs, since the latter relied on the former. Among the *marmas* on the trunk, *trimarmas* hold greater significance. The *trimarmas* are categorized as some of the *pranayatanas* (locations of *prana*). Just as the dependent is annihilated when the base is compromised, harming any of the *trimarmas* could harm the *prana*.

Ojas is situated in the *hridaya*, one of the *trimarmas* and as both *ojas* and *hridaya* are *pranayatanas*; any conditions impacting the *hridaya* will also affect the *ojas*. This can result in *ojakshaya*, which ultimately causes death as *prana* is harmed. Cardiogenic shock occurs mainly by myocardial infarction and in obstructive shock there is cardiac tamponade that directly affects *hridaya* and compensatory mechanism affects kidney (*trimarma*). Septic shock is caused by microbial infection. The pathophysiological effects of endotoxins closely align with the concept of *visha*, which has opposite qualities of that of *ojas* and thus can lead to death. Any *kshaya* in *para ojas* will result in death. Therefore, the *kshaya lakshanas* described by Acharya Charaka and Vagbhata can be linked to the *kshaya lakshana* of *apara ojas*. In shock, patient either dies immediately just as depletion in *para ojas* can cause swift death or, eventually when he reaches the irreversible stage which is included under *ojakshaya lakshana* by Acharya Susruta. It can be understood either as a slight decline of *para ojas* or an excessive decrease of *apara ojas*.

The difference in timeline and scientific interest between Sushruta, Charaka and Vagbhata played a major role in the understanding of *ojakshaya*. Susruta focused mainly on *Shalya tantra* and classified the depletion of ojus into three stages. He focused more on emergency management and needed detailed classification to assess patient's viability for invasive procedure. Though Susruta had outlined 3 stages of *ojakshaya* in detail, further descriptions regarding these are missing. Here, Charaka and Vagbhata focused primarily on *Kayachikitsa* and their aim was to preserve and restore vitality. Thus it was not necessary for them to classify all these stages of derangement.

In addition, as observed by Acharya Susruta, in the three stages of *ojakshaya* there is a progressive decrease in *ojas*, that is the prognosis becomes graver as the degree of depletion of *ojas* increases. In *vyapat*, the patient should be discarded if he has reached the stage of coma and in kshaya the patient eventually dies. Charaka and Vagbhata at the same time have a slow pace - planned approach to the treatment of *ojakshaya*. Their treatment protocol included *rasayana* therapy which is similar to that of *visramsa* treatment by Susruta.

CONCLUSION:

The concept of *ojodushti* resembles the systemic collapse seen in shock, characterised by reduced tissue perfusion, multi-organ dysfunction and failure of physiological equilibrium. *Trimarma* injury and *visha* exposure are powerful triggers for *ojodushti* and correspond clinically into various types of shock. Injury to *trimarma* disrupts vital functions and leads to rapid depletion of *ojas*. *Visha* acts systemically to destroy *ojas*

through toxic, inflammatory or hemorrhagic mechanisms whereas *trimarma* injury is itself extremely fatal. Interpreting *ojodusti* in terms of shock enhances the clinical understanding of critical conditions and opens new perspectives for better management and patient care. It also opens up prospective avenues for *ojovrdhi* line of ayurvedic emergency management in shock afflicted patients.

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