Summary of Product Characteristics

1 NAME OF THE MEDICINAL PRODUCT

Glucose Injection BP Minijet 50%w/v, Solution for injection

2 QUALITATIVE AND QUANTITATIVE COMPOSITION

Glucose anhydrous 500 mg in 1ml as 5g/10 ml and 25g/50 ml

For a full list of excipients, see section 6.1.

3 PHARMACEUTICAL FORM

Solution for injection
The clear, colourless solution, is contained in a type 1 glass vial with an elastomeric closure. The container is specially designed for use with the IMS Minijet injector supplied.

4 CLINICAL PARTICULARS

4.1 Therapeutic Indications

a) As a source of energy in parenteral nutrition.

b) In severe hypoglycaemia due to insulin excess or other causes.

c) For the treatment of *delirium tremens* or acute alcohol intoxication.

Glucose injection 50% w/v is strongly hypertonic and is used partly because of its dehydrating effects. In delirium tremens or acute alcohol intoxication the patient must be rehydrated.

4.2 Posology and method of administration

Hypertonic solutions of glucose should be administered via acentralvein. The dose is variable and depends upon the indication, clinical condition and size of the individual.

The rate of utilisation of glucose varies considerably from patient to patient. In general, the maximal rate has been estimated at 500-800mg/kg body weight/hour. If the patient's capacity to utilise glucose is exceeded, glycosuria and diuresis will occur.

Use as a calorie source in Total Parenteral Nutrition (TPN)

Fluid balance, serum glucose, serum sodium and other electrolytes may need to be monitored before and during administration, especially in patients with increased non-osmotic vasopressin release (syndrome of inappropriate antidiuretic hormone secretion, SIADH) and in patients co-medicated with vasopress in agonist drugs due to the risk of hyponatraemia.

Monitoring of serum sodium is particularly important for physiologically hypotonic fluids. Glucose containing solutions may become extremely hypotonic after administration due to glucose metabolization in the body (see sections 4.4, 4.5 and 4.8).

Adults, elderly, children over 6 years:
Hypoglycaemia: 20-50ml of a 50% w/v solution, repeated as necessary according to the patient's response, by slow intravenous injection, e.g. 3ml/minute. After 25g of glucose has been given, it is advisable to interrupt the injection and evaluate the effect. The exact dose required to relieve hypoglycaemia will vary. After the patient responds, supplemental oral feeding is indicated to avoid relapse, especially after insulin shock therapy.

Acute alcoholism: 50ml of glucose 50% w/v solution should be administered intravenously. Thiamine hydrochloride (100mg) should be added to the infusion. Soluble insulin may need to be given simultaneously. Blood sugar should be measured frequently until stable.

4.3 Contraindications

The intravenous use of strongly hypertonic solutions of glucose is contraindicated in patients with anuria, intracranial or intraspinal haemorrhage, or delirium tremens *if the patient is already dehydrated*.

Known sensitivity to corn or corn products, hyperglycaemic coma, or ischaemic stroke.

4.4 Special warnings and precautions for use

Hypertonic solutions of glucose should be administered via a large central vein to minimise the damage at the site of injection.

Use with caution in patients with diabetes mellitus, severe under nutrition, carbohydrate intolerance, thiamine deficiency, hypophosphataemia, haemodilution, sepsis and trauma. Rapid infusion of hypertonic glucose solution may lead to hyperglycaemia. Patients should be observed for signs of mental confusion or loss of consciousness.

Prolonged use in parenteral nutrition may affect insulin production; blood and urine glucose should be monitored. Fluid and acid-base balance and electrolyte status should also be determined during therapy with dextrose.

Although Glucose Injection, BP Minijet 50%w/v is a hypertonic solution, glucose intravenous infusions are usually isotonic solutions. In the body, however, glucose containing fluids can become extremely physiologically hypotonic due to rapid glucose metabolization (see section 4.2).

Depending on the tonicity of the solution, the volume and rate of infusion and depending on a patient's underlying clinical condition and capability to metabolize glucose, intravenous administration of glucose can cause electrolyte disturbances most importantly hypo- or hyperosmotic hyponatraemia.

Hyponatraemia:
Patients with non-osmotic vasopressin release (e.g. in acute illness, pain, post-operative stress, infections, burns, and CNS diseases), patients with heart-, liver- and kidney diseases and patients exposed to vasopressin agonists (see section 4.5) are at particular risk of acute hyponatraemia upon infusion of hypotonic fluids.

Acute hyponatraemia can lead to acute hyponatraemic encephalopathy (brain oedema) characterized by headache, nausea, seizures, lethargy and vomiting. Patients with brain oedema are at particular risk of severe, irreversible and life-threatening brain injury.

Children, women in the fertile age and patients with reduced cerebral compliance (e.g. meningitis, intracranial bleeding, and cerebral contusion) are at particular risk of the severe and life-threatening brain swelling caused by acute hyponatraemia.

Hyperglycaemia may be caused by physiological stress during ischaemic stroke, and this worsens cerebral ischaemic damage and impairs recovery. During cerebral ischaemia, cellularhypoxia causes a shift from aerobic to anaerobic metabolism of glucose leading to intracellularl acticacidosis, which is toxic to the cell. Hyperglycaemia provides more glucose for anaerobic metabolism, further worsening intracellular acidosis. Blood-glucose concentrations should therefore be monitored and hyperglycaemia avoided or treated. Hypoglycaemia must also be avoided and for patients who do require glucose, it should be given by continuous infusion, avoiding large infusions or boluses that can cause hyperglycaemia.

Glucosesolutionsshould not begiven through thesameinfusion equipment aswholeblood as haemolysis and clumping can occur.

4.5 Interaction with other medicinal products and other forms of interactions

<u>Drugs leading to an increased vasopressin effect.</u>
The below listed drugs increase the vasopressin effect, leading to reduced renal electrolyte free water excretion and increase the risk of hospital acquired hyponatraemia following inappropriately balanced treatment with i.v. fluids (see sections 4.2, 4.4 and 4.8).
• Drugs stimulating vasopressin release, e.g.: Chlorpropamide, clofibrate, carbamazepine, vincristine, selective serotonin reuptake inhibitors, 3.4-methylenedioxy-N-methamphetamine, ifosfamide, antipsychotics, narcotics
• Drugs potentiating vasopressin action, e.g.: Chlorpropamide, NSAIDs, cyclophosphamide
• Vasopressin analogues, e.g.: Desmopressin, oxytocin, vasopressin, terlipressin

Other medicinal products increasing the risk of hyponatraemia also include diuretics in general and antiepileptics such as oxcarbazepine.

4.6 Fertility, pregnancy and lactation

Glucose Injection should be administrated with special caution for pregnant women during labour particularly if administered in combination with oxytocin due to the risk of hyponatraemia (see sections 4.4, 4.5 and 4.8).

Intravenous glucosemay result in considerablefoetal insulin production, with an associated risk ofrebound hypoglycaemiain thenew-born. Infusion should notexceed 5-10g/hourduring labourorCaesarean section.

4.7 Effects on ability to drive and use machines

This preparation is intended for use only in emergencies.

4.8 Undesirable effects

Anaphylactoid reactions have been reported in patients with asthma and diabetes mellitus. Local pain, inflammation, irritation, thrombophlebitis and fevermay occur.

Hypokalaemia, hypomagnesaemia or hypophosphataemia may result from the use of hypertonic solutions via the intravenous route.

Prolonged or rapid administration of hyperosmotic (>5%) solutions may lead to dehydration.

Theadministration of glucose with out adequate levels of thiamine (which form the coenzyme systems in its metabolism), may precipitate overt deficiency states, e.g. Wernicke's encephalopathy.

Excess glucose infusion produces increased CO2, which may be important in respiratory failure, and stimulates catechol amine secretion.

Tabulated list of adverse reactions related to use in TPN		
System Organ Class	Adverse reaction (MedDRA term)	Frequency
Metabolism and nutrition disorders	Hospital Acquired Hyponatraemia**	Not known
Nervous system disorders	Hyponatraemic encephalopathy**	Not known

** Hospital acquired hyponatraemia may cause irreversible brain injury and death due to development of acute hyponatraemic encephalopathy (see sections 4.2 and 4.4).

Reporting of suspected adverse reactions
Reporting suspected adverse reactions after authorisation of the medicinal product is important. It allows continued monitoring of the benefit/risk balance of the medicinal product. Healthcare professionals are asked to report any suspected adverse reactions via HPRAPharmacovigilance, Earlsfort Terrace, IRL- Dublin 2; Tel: +353 1 6764971; Fax: +353 1 6762517. Website:www.hpra.ie; E-mail: medsafety@hpra.ie.

4.9 Overdose

The patient becomes hyperglycaemic and glycosuria may occur. This can lead to dehydration, hyperosmolar coma and death.

Treatment: The infusion should be discontinued and the patient evaluated. Insulin may be administered and appropriate supportive measures taken.

5 PHARMACOLOGICAL PROPERTIES

5.1 Pharmacodynamic properties

Glucose, the natural sugar occurring in the blood, is the principle source of energy for the body. It is readily converted to fat and is also stored in the liver and muscles as glycogen. When a rapid rise in blood sugar is demanded by the body, glycogen is quickly liberated as d-glucose. When the supply of glucose is insufficient, the body mobilises fat stores which are converted to acetate with production of energy by the same oxidative pathways employed in the combustion of glucose.

It may decrease body protein and nitrogen losses. Glucose is also the probable source of glucuronic acid with which many foreign substances and their metabolites combine to form excretion products. It probably provides the basic substances required for the formation of hyalluronates and chondroitin sulfate, the supporting structures of the organism. It can be converted to a pentose essential for the formation of nucleic acids by the cells.

5.2 Pharmacokinetic properties

Glucose is metabolised to carbon dioxide and water with the release of energy.

5.3 Preclinical safety data

Not applicable since glucose has been used in clinical practice for many years and its effects in man are well known.

6 PHARMACEUTICAL PARTICULARS

6.1 List of excipients

Water for Injections

6.2 Incompatibilities

Glucose solutions which do not contain electrolytes should not be administered concomitantly with blood through the same infusion set as haemolysis and clumping may occur. This medicinal product should not be mixed with other medicinal products except those mentioned in section 4.2.

6.3 Shelf life

Unopened: 3 years
Once open: Use immediately. Discard any unused portion.

6.4 Special precautions for storage

Store below 25°C.

6.5 Nature and contents of container

The solution is contained in a type I glass vial with an elastomeric closure and is supplied with an IMS minijet injector which meets all the relevant specifications. The product is available as 10ml and 50ml.

Not all pack sizes may be marketed.

6.6 Special precautions for disposal of a used medicinal product or waste materials derived from such medicinal product and other handling of the product

The container is specially designed for use with the IMS Minijet injector. Do not use the injection if crystals have separated. For single use only. Discard any remaining contents after first use.

7 MARKETING AUTHORISATION HOLDER

DLRC Pharma Services Limited
Chesterfield House
Clonmannon
Ashford
Wicklow
Ireland

8 MARKETING AUTHORISATION NUMBER

PA22684/007/001

9 DATE OF FIRST AUTHORISATION/RENEWAL OF THE AUTHORISATION

Date of first authorisation: 08 September 1977

Date of last renewal: 08 September 2007

10 DATE OF REVISION OF THE TEXT

May 2019