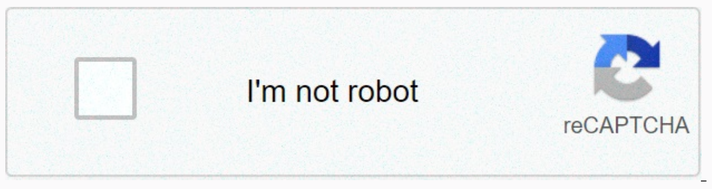

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Subarachnoid haemorrhage guidelines uk

Details of the instructions These guidelines confirm the level of treatment of patients with aneurysmal subaracnoid haemorrhage (aSAH). They provide a framework for best practices for managing the presenting space and preventing complications. Recommendations are made to lead patient care and LTHT care in non-LTH hospitals. Back to the top Background Aneurysmal (spontaneous) subaracnoid bleeding occurs in about 10 cases per 100,000 person-years. It most commonly affects people between the ages of 40 and 60. It is twice as common in women as in men and is associated with high mortality and morbidity, as more than a third of patients die by the end of the first week of ictus treatment. Risk factors include smoking tobacco, hypertension, moderate to heavy alcohol consumption of sympathotometic drugs (e. g. cocaine), use of family aneurysm and (rarely) genetic disorders such as autosomal dominant polysystic kidney disease, and type IV Ehlers-Danlos syndrome. Early diagnosis and treatment are crucial to improving survival from this devastating disease. All cases must be discussed with the Regional Neuroscience Centre. Risk factors and prevention of ASAH Treatment of hypertension is recommended to prevent coronary heart disease, ICH and cardiovascular, injuries to blood and other end organs (Level 1/ A evidence) Hypertension should be a treatment and such treatment may reduce the risk of ASAH (Level 1/ B evidence) Tobacco use and alcohol abuse should be avoided to reduce the risk of aSAH (Level 1/ B evidence) Back to peak diagnosis If an aneurysmal (spontaneous) subaracnoid leakage (aSAH) is suspected, diagnosis as soon as possible after the first resuscitation phase, when the patient's clinical condition is stable. Typical presentations include thunderstorm headache, collapse with or without loss of consciousness, convulsions and related nausea, vomiting and photophobia. The study may reveal focal neurology, including neck stiffness. Complications of ASAH, for example, acute hydrokefalus, may require neurosurgical emergency response. The classification scale of the World Association of Neurological Surgeons shall be recorded in clinical notes Fisher's CT scores (see Appendix 1) shall be recorded in clinical notes. Fisher's score of 3-4 predicts the occurrence of vasospasm and poor clinical outcome. The presence of intra-brain haematoma, which causes mass effect, is a conducive to threne pisto (LP), as well as the presence of obstructive hydrokephalus. Ingestion should not be performed if no prior CT scan has been performed in these patients. A positive CT scan result excludes the need to puncture the lumbar spine. Screen level B Patients without visible blood for CT scans should have a diagnostic thenic injection; This applies in particular to patients whose presentation has been delayed and to patients whose clinical condition suggests that there may have been a small haemorrhage. No, it's. Is. discarding of the first cerebrospinal fluid (CSF) ml (i.e. to collect a medium current sample for analysis), which reduces the possibility of contamination of the sample by epidural blood, which may give a false positive result. The sample is analysed for red blood cell count and xanthochrome. The number of red blood cells uniformly seen in three consecutive samples above 10,000/ml ensures diagnosis. However, patients who have suffered minor subaracnoid bleeding or patients present in a delayed manner may have fewer red blood cells in the brain. In the latter patient population, the presence of xanthochrome (either with the naked eye or with spectrophotometry) is diagnostic. The result of the CSD can only be interpreted correctly on the basis of clinical history. Patients with a convincing history despite negative CT scanning and unclear CSF analysis should be discussed with the neurosurgic group. Screen level B Back to peak care/management at the primary receiving hospital If the patient originally appears at Leeds General Infirmary, follow the assessment and the original treatment plan in accordance with the primary care of the hospital. Although in specialist centres final treatment is carried out, stabilisation, diagnosis, initial treatment and transplantation often take place in specialist hospitals, and this is of the utmost importance. 1.1 Initial treatment 1.1.1 Assessment, oxygen therapy and fluid status Evaluate cardiovascular, respiratory and neurological function. Conduct a targeted neurological examination to determine the level of consciousness (using 15 points glasgow Coma Score - GCS) and degree of neurological deficit, including higher brain functions associated with language and comprehension, skull nerves II, III, IV, VI, VII and XII, and peripheral engine system if possible. Give extra oxygen through the face mask and get at least one (preferably two) large perforated peripheral vein bottles. Initiating fluid resuscitation with 0.9% sodium chloride. Patients with clinical signs of hypovolaemia may be given 0.9% sodium chloride (e. g. 200-500 ml). Continue hydration with a continuous infusion of 0.9% sodium chloride or Hartmann solution administered between 125 ml and 166 ml per hour (3 to 4 L in 24 hours). A urine cooker should be placed on the patient. 1. 1. Level of treatment patients with SAH should be treated in a Level 2 or Level 3 environment (HOBS, HDU or ICU) as soon as practicable, depending on GCSes and early complications. See Appendix 1 on the clinical and radiation classification of SAH. Some patients considered low-risk (those in class 1 WFNS who are considered likely to be perimesencephalyeal SAH) may be treated in a Level 1 (Compartment) bed if agreed with a neurosurgical consultant. Start continuous monitoring of pulse, blood pressure (by invasive (arterial line) monitoring, if demonstrated), ECG, breathing rate and 1.1.3 Nursing and observations Basic observations - observations pulse, blood pressure and oxygen saturation, as well as GCS evaluation and peripheral limb efficacy assessment. Glasgow's coma scale and pupils' reactions in adults The decline in consciousness or the development of a new neurological deficit should trigger a review and computer investigation of nursing staff. Repeat the observations every 15 minutes until the transplant is approved for the neurosurgeon unit. If a patient is deemed unfit for transfer treatment, he or she will return as a supporter, which will be delivered at the initial hospital. 1.1.4 Referral and www.LeedsNeurosurgery.com and transfer Once aSAH diagnosis has been established (see above), the patient should be referred from 1 January 2007. Phone: 079799289120 All patients should be discussed regardless of age and clinical condition. The referral should be made by an experienced clinician who has been involved in the patient's treatment. All patients should be discussed with the consultant responsible for the patient. Patients with reduced consciousness should be consulted by an anaesthetist. Be prepared to provide a full history the neurosurgic readiness team will request the following information. Name, grade, specialty and telephone number of the referrer Patient's name and age. Consultant in charge of the patient. The name or number of the department and the telephone number of the contact person. Brief information on the history of subaracnoid bleeding, in particular the date and times of ICT, and whether the seizure was detected. Significant parallel incidence, history of medicine and allergy. General and neurological condition of the patient, including pulse, blood pressure, oxygen saturation, level of consciousness and degree of neurological deficit (motor deficit, pupillaar reaction to light and presence or absence of dysphasia). The level of consciousness assessed at glasgow coma points is particularly important and must be given orally for each sub-section (e.g. 1999). The patient opens his eyes spontaneously, follows verbal commands and is fully orientated and the patient is not Glasgow Coma Score 15). CT observations (and, if applicable, lying-on results). There is a neurosurgic referral system online, but SAH patients need a telephone conversation in addition to sending an online referral. Based on the information received, the neurosurgic group may decide not to transfer some patients to the neurosurgic unit, but this decision must be made on a case-by-case basis. Screen level B After the supervisor has approved the neurosurgic registrar, a transfer to a Level 3 (ICU) or Level 2 (HDU) unit is arranged through the medical staff. Some patients may be suitable for treatment in High Observation wards (HOBS). Nurse in charge of the receiving department to arrange the transfer to a basic hospital. Patients in stable neurology can be transferred with a convoy of nurses. If there are doubts about the neurological or general condition of the patient, it is mandatory to use a medical escort with appropriate airway skills. Patients with (or at high risk of loss of consciousness) may require intubation and ventilation prior to transplantation. For example, after a seizure or on a related significant hydrokephus ship. This decision is at the consideration of the primary hospital anaesthetist and the referral team. If intubation is considered necessary for safe transplantation, the patient's condition change should be discussed with the neurosurgic group, which may then communicate with the neurointensive unit. Appropriate monitoring of the transfer should continue. At least (if not intubated) - non-invasive or invasive blood pressure (at least every 5 minutes), continuous 3-lead ECG monitoring and peripheral oxygen saturation. Intubated (in addition) - tidal carbon dioxide monitoring and invasive arterial blood pressure monitoring are essential. ICS guidelines for making a critically ill adult available www.ics.ac.uk Hospital notes do not need to be sent, but the patient must be accompanied by a detailed transfer letter, as well as photocopies of the relevant notes. CT images shall be described linked to LGI from the radiology department of the referring hospital. If this is not possible, the patient must be accompanied by copies. Back to the top Continuous treatment at the Regional Neuroscience Centre 2.1 Further evaluation - After transfer to a special neurosurgic service, the following systems will be reassessed: 2.2 Resuscitation Further oxygen administration will continue regardless of oxygen saturation readings. Make sure the patient has safe intravenous access and continue to infusion 0.9% sodium chloride. The majority of patients have clinical evidence of intra-vein depletion due to either delayed withdrawal, insufficient fluid replacement or acute SAH-related fluid transfer. This should be taken into account when prescrimming fluid replacement therapy and fluid resuscitation may include rapid bolus use - 200-500 ml of 0.9% sodium chloride or colloid suspension. Hypotylous solutions such as 4% glucose-0.18% sodium chloride and 5% glucose should not be used to revitalize fluid in SAH as they can exacerbate swelling of the brain. Confusion or restlessness may indicate hypoxia, intravastic fluid depletion, pain or hydrocephus development and should not be considered to have the brain effects of SAH without excluding these reversing causes. 2.3 Level of care As these patients demonstrate the possibility of rapid and dramatic changes, invasive monitoring is often required to facilitate appropriate fluid therapy and control their management in Level 2 (HDU) or Level 3 units. Decision on the transfer to the department take a senior neurosurgeon together with an intensive care consultant and a chief nurse. 2.4 Treatment Treatment Continuous monitoring and vigilance is essential in this patient population. Rapid deterioration of the clinical condition may lead to loss of consciousness and impairment. Significant changes in the patient's condition should be immediately notified to appropriate medical staff to facilitate early intervention and prevent secondary neurological damage. 2.4.1. Installing patients Traditionally, SAH patients have been treated horizontally. There is little evidence that this posture reduces the in appearance of reflow and can even be harmful by endangering the respiratory system. Patients are treated in a position where they find comfortable reducing anxiety and pain; This can mean lifting the bed to a semi-flexible or seated position. 2.4.2 Environmental staff should strive to reduce anxiety and physiological stress by maintaining the calmest and most relaxed environment possible. Noise and lighting levels for which the patient may be acutely sensitive shall be taken into account. 2. 4. 3 Symptom management Pain - See pain relief Section 9.9. Vomiting can cause transient increases in blood pressure and put the patient at risk of aspiration and the ensuing pneumonitis. Antiemetic drugs should be prescribed regularly if patients are sickening. Pyrexia can be an indication of infection, but in the early stages it is likely to have a central effect as a result of SAH. Pyrexia should launch appropriate investigations to determine its cause. Pyrexia causes an additional metabolic burden on the injured brain and can adversely affect the end result. Symptoms can be controlled with regular paracetamol and/or ibuprofen and a cooling fan. In severe cases (e.g. temperatures above 40 °C or above), refractory cooling blankets or the use of cooled intravenous liquids may be reported in accordance with the above methods. 2.4.4 Nutrition Whenever possible, patients should be encouraged to eat and drink normally. Oral fluids should be replenished with intravenous fluids. Patients with reduced consciousness may not be able to use the food orally. In these cases, nasal gastric feeding should be introduced. Nasal feeding should be provided in cooperation with the diet section. In a minority of patients with onion problems, SALT referral may be necessary. Bowel therapy constipsion can become a problem for patients with reduced mobility and patients treated with opiate analgesia. Fae faeces stress should be avoided in these patients, so aperientes and faeal softeners should be given regularly from the start date. Whenever possible, patients should be allowed to use the bedside toilet instead of the bedside table. 2.4.6 Psychological support and information Treat patients calmly and sensitively. Patients may: sekasortoisti sekasortoisti confusion after SAH. Together with pain and psychological stress, this can lead to seemingly inappropriate behavior in some patients. Explain in advance all procedures and confirm that the patient understands the procedure to be performed. Provide information in clear and simple terms and, if possible, support written information. Provide information in the presence of a third party (for example, a close relative) whenever possible, as this may affect the patient's understanding and memory. Confusion, confusion or inappropriate behaviour in patients who have not previously observed these symptoms may indicate a physical cause such as hypoxia, hydrocephus or infection. These possible causes must be investigated. 2.4.7 Caring for relatives Keep relatives informed of the patient's condition and care. Good communication with relatives helps the patient in the acute treatment phase and rehabilitation. Provide information in private, undisturbed and in clear, non-technical language. If possible, a close relative or trusted friend should be identified as a key contact between the nursing and nursing staff. Changes in the patient's condition or management priorities shall be communicated in a timely manner and the possibility of reflection and questioning shall be given. 2.5 Monitoring Use continuous ECG recording to identify arrhythmias or cardiac ischemia commonly associated with SAH. Use invasive monitoring of arterial blood pressure to allow accurate blood pressure assessment and regular arterial blood gas, haematological and biochemical analysis. Centralised renal pressure monitoring should be introduced in patients considered to be at significant risk of developing. delayed neurological deficit, cardiovascular instability, neurogenic pulmonary oedema, significant electrolyte and fluid disturbances. Seek medical attention if it is uncertain. Monitor urine output in hours and start 24 hours of urine collection for electrolyte and osmolality analysis. Use pulse oximetry and arterial blood gas analysis to evaluate breathing functions. Assess a patient's neurological condition using Glasgow Coma Score, pupillary light reaction and motor neurological deficit assessment. Compressed spectral array (CSA) analysis is recommended for patients sedated and ventilated to support early identification and treatment of seizures and ischemistry. Initially, observations should be made every fifteen minutes until the neurological condition is stabilized for one hour of observation. After that, official observations should be made hourly, although interactions with the patient during normal nursing should alert the nurse to a change in neurological condition. Report the deterioration of the patient's neurological condition to the medical staff for further examination/intervention and initiate the frequency of formal neurological evaluation every 15 minutes until stability is achieved again. appropriate invasive monitoring until the patient is considered appropriate to be discharged from the special unit or until the removal is considered medically appropriate. Back to interventional therapy 3.1 Choice of treatment The consultant's vascular neurosurgeon and interventional neuroradiologist should determine the most appropriate choice for final aneurysm therapy based on the patient' s clinical condition and aneurysm composition. Endovascular retraining is the recommended treatment for the majority of aneurysm in the front and rear cycles. If endovascular retraining is unlikely to produce satisfactory results or if endovascular therapy is not clogged with aneurysm or the aneurysm is subsequently declared as eligibility, surgical surgery should be performed. Once optimal treatment has been decided, treatment options should be discussed with the patient and relatives. A clear recommendation for treatment must be made so that the patient can make a decision. Evidence level B 3.2 Timing of treatment Aneurysms should preferably be secured within 24 to 48 hours of IT in patients considered appropriate for procedure and anaesthesia. 3.3 Anaesthetic aspects A full anaesthetic assessment shall be carried out and the necessary pre-optimisation shall be carried out. The majority of patients smoke and are known to be hypertensive. Consideration should be given to continuing to take antihypertensive medication, as unprotected aneurysm has a high chance of rebbled (40% in 4 weeks), which can be reduced by avoiding extreme blood pressure increases. See Table 1 for the proposed blood pressure targets. This can be partially achieved: adequate oxidation of careful fluid management of the normoccant (PaCO2 4.0-4.5 kPa) proper sedation to avoid exertion/cough effective pain relief General anaesthesia must be induced using a familiar range of drugs for anaesthetist. The technique should take into account keen blood pressure control, avoiding coughing and strain. It should also allow smooth birth and exhalation if necessary with a rapid neurological evaluation - remifentanil is an ideal substance in this regard. Display level B 3. Interventional neuroradiology issues After rewinding patients should be treated either at level two (HDU) or level three (intensive care unit) according to their basic condition. Patients usually have a 6F angiocesal device inserted into the common femoral artery to ensure hemostasis. Patients are allowed to sit almost immediately. Patients will be returned with an Angioseal information card, which must be attached outside the notes. 3.5 Anticoagulation Most patients are given heparin during the retraction/ stent procedure, and act (Activated coagulation time) is monitored using the ACT treatment score machine for the angiography suite. Heparin can usually be allowed to wear off without formal change of direction with protamine Occasionally, when there has been a thrombotic or thromboembolic complication or this is a high risk after surgery (reel penetration into the parent artery) heparinisation of heparinisation is required for at least 24 hours. Additional aspirin is required (either intravenously or laden after the procedure or with the help of NGT). This may be continued over a variable period after the procedure. A small proportion of cases of Eptifibatide (Integrelin®) are administered during the procedure. This is a stubborn acute platelet-rich (white) thrombus that can accumulate during the procedure and threaten the patensity of large intracial arteries. . Eptifibatide: 180 micrograms/ kg bolus (IV) and continued as a vein infusion at 0,5 micrograms / kg / minute – 1 microgram /kg / minute up to 24 hours From theatre to ICU/ HDU Eptifibatide there is a short half-life; platelet function returns to 50% within 4 hours of discontinuation Optional patients in the Inr group should be clearly documented and passed on to a critical treatment/neurosurgical team.: GPs are advised to start taking platelets according to the instructions of the interventional radiologist of aspirin and clopidogrel, if Prasugrel has been selected instead of clopidogrel, this is loaded on the day of the operation, 2-3 hours before the stent is inserted, the dose of prasugrel is 30 mg (stock in the neurosurgic ward/ contact pharmacy) All delays in administration should be reported to the consultant Prasugrel dose is off the label and no weight or age dependent Oral platelets (treatment after the introduction of a post-OP stent) Treatment options include : Aspirin 75 mg OD (lifetime + clopidogrel 75 mg OD or aspirin 75 mg OD (lifetime) + Prasugrel 5 mg OD Prasugrel dose is off the label and no weight or age-dependent co-medication Lansoprazole (or alternative gastro protection) for the second platelet (6 months) Dosage data are documented in postoperation notes with INR rules. Contact them in possible questionnaires/ concerns Indications and start/stop dates with platelets should be documented in the prescription and eDANS 3. This can be done either by mri or by catheter angiography. This is carried out 6 months and 2 years after the procedure. Sometimes the follow-up of some lesies is no more than 5 years. Clinical monitoring is usually after 6 weeks. This is ideally in a combined neurovascular clinic, especially if there are concerns about the initial durability of the procedure or if there are additional aneurysmes that may need to be considered for treatment. Back to the top Platelet treatment Pharmacological treatment 4.1 Nimodipine dose: 60 mg every 4 hours (fractionas dose to 30 mg every 2 hours if hypotension post-dose) The tablets may be crushed for NGT administration C/ I: within one month the error indicator/unstable angina/hypersensitivity to active/excitable substances (in both cases contact with the consultant neurosurgeon) Display level A Conditions, where enteral (PO/NG) nimodipin cannot be given/absorbed intravenous nimodipin use should not occur without discussion, between the neurosurgeon and the relevant ICU consultant on the relevant risks and benefits. Intravenous nimodipine can accelerate deep hypotension and exacerbate existing brain ischemia. It should be administered through the central vein and in accordance with the instructions of an anaesthetist/neurosurgeon consultant. For more information, see the Summary of Product Characteristics and Appendix 5 to Section 4.4. Allow dehydration to be higher in patients with SAH as a result

of fever, increased breathing rate and supplementary oxygen therapy. Avoid hypovolemia. Fluid therapy should maintain an adequate amount of blood flow based on clinical evaluation, pulse, central intravenous pressure, arterial blood pressure and urine output. It is recommended to feed at baseline at least 3 L 0.9% sodium chloride in 24 hours. This volume should be adapted to the individual needs of the patient according to clinical needs and re-examined over an 8-hour cycle over an uncomplicated cycle. Fluid therapy should be controlled hourly by input baseline measurements and a blood sample of 8-12 hours for urea and electrolytes. Patients able to use oral nutrition and fluids, although the recommended intravenous fluid therapy described above should be maintained within an acute time. Table 1 . Suggested target parameters for blood pressure, central intravenous pressure and urine outlets were not known for hypertension Known hypertension Systolic blood pressure (mmHg) 120-140 20% normal systolic BLOOD PRESSURE CVP (mmHg) 8-12 8-12 Urine output (ml/ kg/ h) 0, 5-1.0 0.5-1.0 Display level B These target parameters are for patients showing no signs of delayed neurological deficiency. Patients showing signs of delayed neurological deficiency will be considered in later sections of this paper. Back to controlling blood pressure Optimum blood pressure in patients with aneurysm SAH has not been established. The systolic blood pressure threshold below 100 mmHg for a previously normotensive patient or a reduction of less than 20% of normal value in patients with pre-existing hypertension should initiate intervention. Patients with suspected long-term but untreated hypertension should not be given antihypertensive medicine at an acute stage of treatment unless a clinical assessment indicates an increased risk of aneurysm rupture. These patients may need above normal blood pressure to maintain brain perfusion and antihypertensive medications jeopardize this. Patients without known or suspected hypertension with systolic blood pressure above 180 mmHg should consider acute reductions in blood pressure. In these patients, treatment should only be considered if blood pressure is monitored invasively and technical reasons for false elevated readings have been excluded. Transient increases in systolic blood pressure do not require treatment. Other causes of hypertension, such as pain and fever, should be excluded. If treatment is deemed necessary, the patient should be discussed with the NICU medical staff. Labetolol (up to 20 mg intravenous bolus followed by 20 to 160 mg / hour intravenous infusion) is a treatment. Alternatively, estmolol (50 to 200 micrograms / kg / min intravenous infusion) may be used. Screen level B Back to the top Managing seizures and prophylactic use Seizures are a relatively common problem that occurs after SAH and occurs in up to 25% of aneurysm leaks. Seizures may be related to the initial ICT of aneurysmarepeäma, the mass effect of major aneurysm, the metabolic consequences of aneurysmarepeäe (electrolyte disturbances) or delayed coronary neurological deficiency. After SAH, and especially in unprotected aneurysms, re-eligibility should be considered the cause of seizure activity. Bloodlucose should be assessed on the bedside table and any abnormalities should be treated at the beginning of the process. The treatment of seizures of SAH is multimodal and uses an increasing set of pharmacological strategies, depending on the severity of the seizures and the severity of the response to treatment. In the early stages, there is little difference between SAH and control of the scene from other starting points. Asah does not have RCT to control routine epileptic therapy, but short-term epileptic therapy is commonly used and influenced by intervention and other risk factors such as aneurysm in MCA, related cerebrovasing haematoma, retination, poor neurological quality, previous seizure, etc. If the levetirasetam initiated is a preferential AED (unless C/I) Accept prophylactic and ensure that: that the stop date is on prescription Dose: 500mg BD PO/IV/NG (if seizure activity occurs during prophylactic therapy, monitors management as described below and contacts the neurosurgeon consultant) 6.1 Suspected/established seizures of pharmacological management If the above procedures do not control seizure activity, an expert report should be sought. Levetirasetam (Keppra ®) is increasingly used in LTHT to prevent seizures because it does not require plasma level monitoring and has good bioavailability orally. Other substances to be considered are phenobarbitone (ICU monitoring required) Pharmacological control of seizures should be parallel to attempts to narrow down the cause. CT scan of the brain should be considered as may reveal a surgically repairable cause that may limit the pharmacological movements. If there are concerns about the patient's conscious level of either post-ICT status, pharmacotherapy or the underlying disease process, anaesthetic staff should accompany the patient for scanning. Electrolyte aerations should be sought and corrected (hypomagnesemia is a particular problem, especially if the patient has had high levels of diuresis). Csf sampling may be appropriate, especially if sewerage equipment, such as an external chamber well or a lying well, has been used. This needs to be discussed with the neurosurgic team. 6.3 Post-seizure treatment The level of patient care after the end of the seizure largely depends on his clinical condition. If the patient has fully recovered to their appropriate level of knowledge without a focal gap, it may be appropriate to keep them at level two (HDU). If there are concerns about the conscious level, their ability to maintain a safe airway or other changes in neurology may be appropriate to escalate to level three . The patient should be closely monitored for the development of new seizures and atonement complications or seizure control medicinal products. Screen level B Back to the top Management of special complications 7.1 Delayed neurological deficit (DND) Delayed neurological deficit is a term that covers clinically detectable neurological impairment in a patient with SAH after initial stabilization that excludes a membrane caused by a broken aneurysm of a new SAH. There are several causes, all of which are potentially reversive, so early diagnosis and intervention can significantly change the clinical course of this complication. The causes include delayed brain ischemistry (DCI), hydrocephalus, cerebral oedema, fever, convulsions and electrolyte ailments. The onset of symptoms typically occurs 3-14 days after SAH, but not usually earlier. DND is seen in 20-30% of patients with aneurysm SAH and leads to completed stroke in 30% of these and death in a further 30%. Vasospasm in the brain is a term that applies to the narrowing of the arteries after SAH has been demonstrated radiologically or sonographically. This leads to a reduction in blood supply and oxygen supply to the brain, which can accelerate the brain's ischemistry or infarction. The term is often used interchangeable with DCI, but should only be used to describe radiographic observations. Delayed brain ischemia (DCI) is a term applied to neurological degeneration due to ischemia (e.g. haemiparesis, aphasia, altered level of data) that lasts more than an hour and cannot be explained by other physiological abnormalities observed in radiological, electrophysiological or laboratory studies. The presence of DCI may mask the patient's poor clinical condition or the giving of sedatives An urgent CT scan shall be arranged for a surgically treatable cause deterioration, in particular hydrocephalus, intracinal haematoma and intractal sepsis (in patients who have received surgical treatment for their aneurysm) and identify areas indicating ischemia. A full general clinical study should be conducted to identify other causes of pollution or other conditions associated with DND. Problems typically seen in SAH patients include respiratory infection, pulmonary oedema and pulmonary embolism, sepsis of invasive follow-up lines, surgical wounds or urinary tract infection. Conduct a full neurological examination to identify new changes, deterioration or response to treatment. The possibility of meningitis should be considered and the low threshold for puncture of the lumbar spine must be maintained. Laboratory tests must be carried out as a matter of urgency and the results must be called back to the ward. Mandatory studies include complete bleeding, urea and electrolytes, and arterial blood gas analysis. A full bloodshot analysis identifies anaemia and may give an indication of the patient's hydration level. Sodium abnormalities are a common finding in SAH. Low serum sodium levels may indicate the possibility of brain salt waste syndrome (CSWS) - see section below on fluid/electrolyte imbalance. Whether CSWS is a separate clinical unit or other manifestation of DND is unknown, but the depletion of volume seen with csws exacerbates DND and needs to be addressed. Urea and creatinine levels can help in the volume assessment. Urea and electrolyte analysis should be performed every eight hours during DND until the patient's condition has reversed or a deficiency has been established. After that, daily urea and electrolytes should be performed or more frequently if clinical need so requires. Perform arterial blood gas analysis to monitor hypoxaemia and hypercompensation. Sometimes, when the diagnosis is uncertain and/or leads to better specific interventional therapy (in-arterial nimodipine infusion and/ or intracial angioplasty), CT angiography may be used. TT Perfusion usually plays a limited role, but it can be useful in selected individual cases. These further studies should involve a consultant interventional neuroradiologist. The main goal is to ensure adequate access to well-oxidized blood in the brain. The traditional approach of triple-H therapy (hypertension, hypervolemia and haemodilution) is now largely historical, and the latest evidence suggests induced hypertension when the intrasenarian volume alone is sufficient. Hypervolemia has not been shown to have any additional benefits for euvolemia, but has been associated with more complications, both as a result of a haemodilation reaction that reduces lung and blood oxygen carrying. All patients with suspected DND should have: Invasive monitoring of arterial blood pressure. Central cart boiler. Urine cooker. pulse oximetry to monitor the height of arterial ulpie sucerations and respiratory tract. Persistance of neurological deficiency for 24 hours or more despite inotropic/vasopressor or vasopressor may indicate a permanent deficit and continuous administration of substances is unlikely to lead to clinical improvement. Vigilance on ECG changes is needed. If cardiac ischemia is proposed, 12 lead ecg and troponin are required and an assessment of cardiology is warranted. Patients with pre-existing heart disease, pulmonary oedema or secondary cardiac function loss for aSAH should consider placing pulmonary arterial etching in order to closely monitor cardiac function. These patients need access to a Level 3 unit (ICU). All patients should receive nimodipin (60 mg / 4 hours daily, PO/ NGT.) routinely for 21 days. If this leads to significant hypotensiveness, consider converting the dose to 30 mg / 2 hours. Treatment of anaemia The optimal haemoglobin level in patients with aSAH has not been accurately determined. For adequate oxygen viability, haemoglobin levels above 8 g/l (80 g/l) are recommended, although a higher target (>10 g/1L) may be appropriate in patients with associated co-pollution and in patients at highest risk of PMI. Haemodilization to improve blood reology should not be performed except in the case of polycytamia. Reduced oxygen carrying capacity in the blood can have harmful effects. Treatment of groin Haematoma In patients undergoing endovascular procedures via a femoral artery puncture, the injection site should be checked for the underlying haematoma. Unexplained drops in Hb, groin/leg or back pain should lead to the removal of hematoma at the injection site. An urgent ultrasound examination should be requested for further examination. Early participation of endovascular and vascular surgical teams is recommended. Risk factors for groin haematoma include multiple injections, difficult access, the use of large drilling diapers and catheters, and patient factors that predispose them to the formation of haematoma, platelet and anticoagulation drugs. Taking pelvic CT scans into account is important in patients at increased risk of haematoma with severe back pain and/ or unexplained hypotension/ anaemia (ultrasound may be difficult to detect retroperitoneal haematoma and there may be no outward signs of bleeding at the injection site). Treatment of sodium balance The target plasma sodium ≥140mmol/l and <150mmol/l. Hypertical sodium chloride solutions can be used, for example, 1.8% sodium chloride in combination with oral sodium supplementation. Regular monitoring of plasma concentrations of Na is mandatory, especially if hypertone sodium chloride solutions are used to avoid excessively rapid correction and also to correct sodium levels. For more information on how to correct sodium content, see Appendix 2. Oral/natural gas modified release tablet (Slow sodium®) 600 mg/ 10mmol Dose: Two tablets BD (prophylactic) (prophylactic) may be increased with appropriate fluid yield by reference to BNF The liquid is available for NG administration Hypertonic saline solution (HDU/ ICU only) 1, 8% sodium chloride Rate: 50-150mL/ hour Low sodium-containing solutions such as 4% glucose-0.18% sodium chloride or 5% glucose may worsen the brain edatemia and may exacerbate hyponateraemia. They should NOT be used with DCI unless the patient needs glucose for hypoglycaemia (e. g. IDDM). Urine collection should be introduced for 24-hour urinary volume, electrolyte and osmolality analysis. To reduce the inertance of DND, evidence of early use of lumbar lumbar discharge is emerging. See Appendix 3. Display level B Abnormalities between fluid and electrolyte, including hyponatremia Sodium disorders Sodium disorders are relatively common after subaracnoid bleeding frequently with DND. Their diagnosis and treatment can prove challenging. Hypernatramia Important causes to consider are (cranian) diabetes insipidus (DI) and iatrogenic. With DI, relative to complete absence of diuretic hormone leads to the loss of a large amount of diluted urine and subsequent hypernatremia and hypophalemia. The diagnosis is made by measuring high plasma osmolality in combination with low urine osmolality. The treatment is fluid retention (0.45% sodium chloride) and enterove and desmopressin (0.5-1.0 microgram bolus). Discussion with the endocrinology group may be useful for patient-specific treatment. Iatrogenic causes may be due to improper administration of diuretics and fluid restriction. Mild hypernatreamia (145-150 mmol/ l) may have a protective effect against vasogenic oedema. Hyponateremia is common after SAH. The causes of hyponatraemia can be classified by patient volume - hypovolaemia, normovolaemia or hypervolaemia SAH - In the case of inappropriate antidiuretic media syndrome (SIADH) and cerebral salt syndrome (CSWS), it is important to identify and treat. SIADH leads to an elevated circulatory volume - hypervolemia CSWS reduces circulatory volume - hypovolemia. It should be stressed that clinical syndromes can be complex with fluid therapy. In addition, the combination of the syndrome (SIADH and CSWS occur in the same patient) may further obscure the clinical picture. CSWS is seen more commonly than SIADH after SAH. Progressive symptomatic hyponaemia, which is not easy to correct with an extra sodium supplement, requires taking HDU in the CVP and monitoring of blood pressure inside the arteries. Consideration of hypertical sodium chloride determines the admission of HDU. The volume determined by clinical trial and CVP measurement suggests that a diagnosis and plasma and urine electrolyte levels confirm the diagnosis. SIADH SIADH is usually self-limiting after SAH. Normal treatment involves liquid restriction; subaracnoid is potentially dangerous because it carries a higher risk of stroke. If hyponatemia is severe (< 125 mmol/ l) and symptomatic, hypertical sodium chloride can be used with great caution. Drug-related causes of SIADH Antidepressants TCA, SSRIS, MAOI Epileptic Medication Karbamasepine, sodiumvalproate Antihypertensive drugs ACEi, ARB, amlodopine Psychotic medication Phenothiatsins, Butyrofenones PPIs Omeprazole, Lansoprazole Special attention should be paid to the neurological state of the patient when treating DI, CSW and SIADH, as rapid sodium changes can lead to reduced consciousness, neurological deficit or seizures. CSWS CSWS is treated according to volume and sodium substitution. The volume of the liquid and the sodium requirements for normalisation of intrasne volume and plasma sodium should be determined and appropriate fluid therapy (using 0.9% or 1.8% sodium chloride solution) should be determined and preserced. The aim is to keep sodium above 140mmol/l and below 150mmol/l. (see Appendix 2) Additional fluid therapy should be determined by assessing volume and sodium replacement needs over each 24-hour period. Changes should be made to fluid therapy based on 8-hour plasma and urinary electrolytes. Mineralocorticoid fludrocortisone can be used to help sodium and fluid accumulation, a typical starting dose of 50 micrograms of TDS. Sodium loss can also be compensated by oral supplements with slow sodium tablets or sodium chloride fluid 1mmol/ml (unlicensed medicine) (display level class II A/B) Display level B Special attention should be paid to the neurological state of the patient when treating DI, CSW and SIADH, as rapid sodium changes may lead to reduced consciousness, neurological deficiency or seizures. DI CSWS SIADH Plasma Sodium reduction Decreased urine Sodium excretation Plasma osmolality increased Normal/ increased urine osmolality Inappropriately decreased Normal/ high volume (clinical study/ CVP) Decreased normal/ increased Table 2: Summary phy diabetes insipidus (DI), brain salt wasting syndrome (CSWS) and inappropriate diuretic hormone secretion syndrome (SIADH) syndrome 7.2.1 Sodium monitoring in neurosurgical wards. It is recommended to remain vigilant about the critical treatment of sodium imbalance after discharge. All patients should have accurate cumulative fluid balance, including twice a week, and daily plasma sodium concentrations (you and Es) should be monitored for at least one week after discontinuation of critical therapy and for longer if concerns remain. If the plasma sodium concentration decreases by ≥5 mmol/l/day or the absolute value is less than 130 mmol/l, this should trigger an immediate assessment of the underlying cause (CSWS, SIADH or DI) and treatment of the suspected cause. Measurement of plasma and urine electrolytes and 24 hours start of collection shall begin with the initiation of a suspected diagnosis and rapid clinical clinical leading members of the neurosurgic and neurocritic treatment group have the right to decide on appropriate treatment and standard of care. The urine cooker should be located if it is not already on site. Hydrocephalus Hydrocephalus may occur acutely as rapid loss of consciousness after subaracnoid bleeding or insidiously for several days or weeks. It is characterized by: Lowering the level of consciousness Headache With or without intracnal pressure. The two main reasons for the hydrokephus after SAH. acute intraventricular bleeding may cause a blockage in the ventricular outflow, typically at the Saliva water supply. impaired absorption of intravasescence to arachnoid savages, leading to communication of hydrokefalus infection. 7.3.1 Hydrokefalus Treatment of acute hydrokephalus should be treated with external ventricular drain therapy (EVD) of csf. Remove evd as soon as possible when the patient is estimated to have restored normal brain line flow and absorption. A lye well may be selected at the brain and brain confluence point of the hydrokephus (see Appendix 3). Before removal, a 24-hour observation period shall be carried out, in which the sewer is on site but closed. Intra-vein sampling of the drainage system is not recommended unless there is clinical suspicion of intra-vein infection. Some patients remain dependent on emptying and require the insertion of an out-of-ventricular hetoneal (VP) shunt. Patients with insidious hydrokefalus should first be treated with a cerebral vein sin. In many cases, this can avoid the need for a permanent vice president sint. Repeated procedures are possible, but VP switching is inevitable in some patients. Display level B 7, 4 Breathing complications 7. Optimal treatment of NPO differs from standard hydrostatic pulmonary oedema and may be associated with a severe fluid load combined with ventilator supports with adequate/ high PEEP, vasopressor infusion, vasodilators and inodilators (dobutamine, adrenaline and phosphodiesterase inhibitors). Pulmonary arteries can be very beneficial in this patient population. Treatment decisions should be made on a case-by-case basis with senior nursing staff. 7. 4. 2 Infection and altelectase The risk of aspirantulation followed by pneumonia is higher when SAH(GCS<8) is worse. Prolonged ventilation puts the patient at risk of respiratory pneumonia (VAP). See the hyperlink below to examine and process VAP. Treatment of pneumonia associated with a ventilator Severe vasospasm, which requires long periods of vasopressor infusions and immobility, also increases the likelihood of developing altelectase and pneumonia; especially if variable GCSes are a problem. Regular physiotherapy can help control the altelectase. Early consideration of tracheostomia in this group helps to reduce sedation (reaching target levels for blood pressure), moistening, mobilization and physiotherapy +/- for mucous membranes 7.5 Heart complications 7.5.1 Cardiac ischemistry and myocardial infarction A sympathetic storm associated with SAH leads to significant cardiovascular changes and may even mimic acute myocardial infarction. ECG can indicate: Non-specific ST and T wave changes, especially in symmetric T-wave inversion Prolonged QT (>500ms) Repolarization abnormalities with simultaneous increases in cardiac troponins and CK-MB. Myocardial disorders appear to correlate more with neurological deficiency than with the severity of ECG abnormalities. ECG changes should never be ignored in these patients and discussion with senior colleagues and/ or cardiology is recommended. 7. 5. 2 Takotsubo Cardiomyopathy (Clover LV ballast syndrome) Well-described cardiomyopathy associated with intra-brain haemorrhage and aneurysm SAH, characterised by the expansion and ballooning of the LV epic and the associated systolic dysfunction with preserved (and even hyperkinetic) dysplexia. It represents the extreme form of myocardular dysfunction caused by calycoliamine. The treatment supports inotropic and vasopressors, which are controlled by echocardiography or pulmonary arterieskateter placement. 7. 5. ECG abnormalities of cardiac arrhythmia have been reported in 25-100% of cases. These are mainly prolonged QT, widened QRS complexes and takyarrhytmias. There is no evidence that these arrhythmias require a different treatment than if they occurred in the non-SAH population. Hyperglycaemia/ glycaemia management hyperglycaemia is a common phenomenon after SAH and is associated with increased mortality and morbidity. Hyperglycaemia causes secondary injury after SAH, which causes ischemia and neuronal death in the brain by increasing the production of brain lactic acid. Glycemic control – keep between 5-10 mmol/L - prevents secondary offending. This should be balanced with the adverse effects of hypoglycaemia. . (Display level II(b/B) Guidelines for the treatment of hyperglycaemia have been adapted from the treatment pathway of the intensive care unit: Glycaemic monitoring guideline for critical therapy (below) It should be recalled that both hyper/ hypoglycaemia and natremia have adverse effects on the damaged brain through osmotic transmission and increased lactic acid production. Both sodium and glucose levels should be monitored regularly to prevent the above abnormalities. Rebleeding is common and has the highest in visibility immediately after first bleeding (5-10% in the first 72 hours). Early rebledation, within hours of the first bleeding, occurs in at least 15% of patients. Risk factors include poor quality of SAH, higher aneurysms and sentinel bloodsped. At the moment it is practically impossible to prevent this from happening, but medical surgical procedure can prevent repeated bleeding later. The rebleeds released in the first few days are thought to be related the static nature of aneurysmical thrombus, unlike the lulysis of the clot sitting on top of the tear point. Clinical factors that increase the likelihood of rebleeding include extreme hypertension, anxiety, agitation and convulsions, so monitoring and rapid treatment of these factors can reduce re-exposure rates. Some authorities recommend the use of antifibrinolytic substances such as tranexamic acid to be admitted until the aneurysm is ensured to reduce rebledation. This is not universally accepted and exposes the patient to a higher risk of thromboembobolic complications. Back to the top DVT/PE thromboprophylaxia In patients with aneurysmal SAH, there is a moderate risk (10-40%) DVT development. SAH itself causes a prothrombotic state. In addition, patients are often elderly, immobile and initially dehydrated. The risk of DVT in all patients should be assessed using local approved tools. VTE Propylaxy in neurological surgery patients Thromboprophylax should be used routinely in patients undergoing major neurosurgery. Adequate hydration and early mobilisation are important preventive factors For intermittent pneumatic calf compression with or without elastic socks, it is recommended that it be introduced and should be continued for up to two weeks after bleeding. Pharmacological prophylaxitic prevention should be considered 24 hours after the procedure (surgery or interventional terminology) unless it is contingent for other reasons. Prophylaxia with low molecular heparine is the recommended pharmacological method. A combination of mechanical and pharmacological prevention is recommended for high-risk neurosurgery patients. Back to the top Analgesia SAH can lead to significant pain, which can be difficult to treat. Remember that photophobia is best treated with subdued lighting and a quiet environment. Many commonly used analgesics have side effects that make them relatively counterintent after SAH. It must assess the nature of the pain and ensure that it does not exceed what is expected of a baseline disease condition, which may indicate a worsening of the underlying pathology. Pharmacological treatment Unless the counterindicated initiates paracetamol 1g (if more than 50 kg) orally, rectangatively or parenterally. If the pain persists, paracetamol should be given regularly (qds) and more potent substances should be added. [Step up to the next 'rung' of the painkiller ladder]. Patients <50 kg - IV dose is 15mg/ kg The second line of analgesics are opiates. Codeine or dihydrocodine is usually administered at doses of 30-60 mg. If the efficacy of codeine for oral codeine (morphine sulphate) appears to be limited. Ask your anaesthetic and pharmacy staff for advice. The use of morphine PCAS is controversial, but may play a role in the pain stairs of subaracnoid bleeding. As above, ask Tramadol is equally potent for dihydrocodine; all opioids have potential lower the scene threshold. More of it has been observed with Tramadol, so optimize alternative pain relief before starting this. Acute pain guidelines The use of NSAIDs is controversial and recent studies have shown that they add little to post-op-craniotomy pain relief. Their use shall be considered in an individual risk-benefit assessment. NSAIDs are not recommended if patients have stents and are taking dual vaccine therapy to prevent thrombosis. (NSAIDs increase the risk of bleeding, especially with Prasugrel (approximately 70% platelet blocking) If NSAIDs are selected, taking into account the broader class and cox-2 selective inhibitors and only for a short period of time. Back to the top Of infection prevention and control See high-impact intervention 1 and normal infection control precautions policy and LTHT hand hygiene policy. Hand hygienic policy Standard precautions 10. For Level 2 patients, if surgical surgery or interventional radiological retraining is likely, decolonisation should be initiated from the start of treatment. The risk of MRSA in all patients should continue to be assessed. Treatment includes (after checking the state of allergy, especially for nuts); Baktroban® (Mupirocin 2%), nasal ointment three times a day for 5 days. Chlorhexide is washed once a day for 5 days, and two hair washes during these 5 days. The first hair wash should be given before surgical procedure. MRSA Policy 10.2 PERIPHERAL VENOUS ACCESS Cannula Guidelines for peripheral kidney 10.3 URINARYKATER GUIDELINES In September 2012, new measures were introduced to reduce the risk of hospital infections in patients with central charge control devices. This consists of daily antiseptic washes with 4% chlorhexide and twice-weekly hair washes with 4% chlorhexide. Octenisan should be used if the patient is allergic to chlorhexide. Control of the arterial line Central Venous Catheter Back to top evidence base: ReferencesAdams JP, Bell D, McKinlay. Neurocritic therapy - A guide to practical management. Springer 2010 Adrogue HJ, Madias NE. Hyponatemia. N Engl J Med 2000; 342(21): 1581-1589 Agnelli G et al. Enoxamarine plus compression stockings compared to compression stockings alone in the prevention of venous thromboembolism after electron surgery. NEJM 1998; 339:80-5 Albanese A, Hindmarsh P, Stanhope R. Control of hyponatremia in patients with acute brain violations. 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(in the case of non-national guidelines or widespread disagreement on the recommendation at level C or in the case of conflicts with national guidance documents) Trust Clinical Guidelines GroupLHP version 1.1 WFNS (World Federation of Neurosurgeons Scale)NB CSWS is clinically more likely than SIADH after ASAH. The aim is to increase the [Na] level by up to 0.5 mmol/h to a maximum of 10 mmol per day. Avoid hypovolemia. Reliable diagnosis of SIADH excludes the causes of the body, thyroid gland, pituitary gland, liver and heart. Monitor [Na] levels at 6-8 hours per hour until normalized and stable. Total weight (litres) = 0.6 x weight (men) = 0.5 x weight (women) = 0.5 x weight (men >65 years) = 0.45 x weight (women>65 years)Estimates the effect of 1 liter of sodium chloride (308 mmol/l) at 1 litre. Sharing the desired change in serum Na with the formula result determines the volume and rate of administration of the necessary infusate.60-year-old male, 100 kg. serum Na 114. The desired Na 130 mmol/l. 1 liter of 1.8% sodium chloride (308 mmol/l) would change serum Na (308-114) / [(100 x 0.6) + 1] = 3. 2 mmolIf the desired remedy is 130-114 = 16 mmol Then the volume of the liquid is 16 / 3.2 = 5 liters 1.8 liters 1.8% sodium chloride (5000ml) Correction in 32 hours (0.5 mmol/h) the repair rate is 5000 / 32 = 156 ml/ h Without hydrocephalus Ii class evidence of early snow use, the entry into the drainage to reduce the intrenity of delayed coronary neurological deficiency (DIND) and to improve early clinical outcome in the World Federation of Neurological Surgeons (WFNS) class III in patients with aneurysmal subaracnoid haemorrhage should be directed as early as possible after an aneurysmical diagnosis and treatment (or placed under general anaesthesia for aneurysm treatment). The addition should not be made no later than four days after bleeding. There is no evidence that the addition of a lying well reverses established DIND unless the hydrocephalus element is believed to contribute to a neurological deficit (in which case DIND should not be diagnosed until the hydrocephalus is excluded). THNNE TUBE DISCHARGE IS COUNTERINVESTIGATION WITH CLOTTING ABNORMALITIES, INTRA-BRAIN DEPLETION AND/OR SUBDURAL HAEMATOMA. The connection must be carried out aseptically with a suit and gloves and a complete sterile field created in the appropriate lumbar space (L3-5). Local anaesthes are applied to the skin and under subcutaneous tissue and allowed to work (3 minutes) before attempting to install the drain. Once the drain is inserted, it must be fixed with a stitch and dressed. The dressing must not be removed unless there are concerns about the operation of the drain or the integrity of the exit point. The Becker collection system shall be set to the same level as the exit point of the drain and set the feeling for hours with the aim of 5-10 ml of csf drainage per hour. The sewer must remain in place until the cerebrospinal fluid (CSF) is visibly clear or xanthochrome or until the sewer has been present for eight days. Csf sampling is not routinely required if the drain is less than 4 days old and must be carried out every 48 hours thereafter (using aseptic technique). When the drain needs to be removed, it must be removed immediately without prior attachment and a non-silk suture placed on top of the exit point. Hydrokephalus Patients with aSAH and consciousness (GCS <13-14) may have symptomatic hydrokephalus. If this is thought to require treatment, a lying water drain can be selected to treat this through an external chamber drain (EVD). If the patient does not follow the commands, it should be advisable to set evd instead of a thingal tube. If the patient has improved to at least 13 GCSes after hydrokefalus therapy, the above lye tube removal treatment protocol may be applied. The only difference from the above is that when the drain is removed, it has been tightened 24 hours before removal to assess whether the patient is dependent on drainage of the brain veins. If so, it may be necessary to use a VP replacement (or if it's still a heavy blood load), a replacement thena plug if the original is over 10 days old. Additional dose : Patients currently taking phenyenyinThe technical phenythyin level (last 24-48 hours) and correct it with the above formula in hypoalbuminemia patients before the additional dose is calculated. Additional dose of phenytoin sodium (mg) = [20 – (phenytoin level (mg/l)] x 0.7 x body weight(kg) Saturation dose: Patients NOT taking phenytoin prior to treatment Taking level 6 to hours after loading the dosePPM declared level EI EI phenyenyin level if albumin <35 g/l. In critically ill patients, this calculation serves as a guideline in the clinical state of patients due to rapidly changing albumin levels. From oral care (Weaning begins when the first dose of oral care is provided)Leeds Teaching Hospitals NHS Trust → is committed to ensuring that the way we provide services and the way we recruit and treat staff reflects individual needs, promotes equality and does not unfairly discriminate against any particular person or group. Group.

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