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Contents

Original Article

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Hyperpara Thyroidism and its Management

Simrat Kaur[1], S.P. Subashini[2], Pooja Jain[3]

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ABSTRACT

Hyperparathyroidism is an increase in parathyroid hormone (PTH) levels in the blood. This occurs from a disorder either within the parathyroid glands (primary hyperparathyroidism) or as response to external stimuli (secondary hyperparathyroidism). Symptoms of hyperparathyroidism are caused by inappropriately normal or elevated blood calcium leaving the bones and flowing into the blood stream in response to increased production of parathyroid hormone. In healthy people, when blood calcium levels are high, parathyroid hormone levels should be low. With long-standing hyperparathyroidism, the most common symptom is kidney stones. Other symptoms may include bone pain, weakness, depression, confusion, and increased urination. Both primary and secondary may result in osteoporosis (weakening of the bones). In 80% of cases, primary hyperparathyroidism is due to a single benign tumor known as a parathyroid adenoma. Most of the remainder are due to several of these adenomas. Very rarely it may be due to parathyroid cancer. Secondary hyperparathyroidism typically occurs due to vitamin D deficiency, chronic kidney disease, or other causes of low blood calcium. The diagnosis of primary hyperparathyroidism is made by finding elevated calcium and PTH in the blood.

Keywords: Vitamin D; Osteoporosis; Parathoromone; Renal Rickets.

INTRODUCTION

It is caused by overproduction of parathoromone by the parathyroid glands and is characterized by bone decalcification and development of renal calculi containing calcium. Primary Hyperparathyroidism occurs two or 4 times more often in women than in men. Rare in children younger than 15 years of age, but it increases 10 fold between the ages of 15 and 65 years. It is caused by over production of parathoromone by the parathyroid glands and is characterized by bone decalcification and development of renal calculi containing calcium.

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TYPES OF HYPERPARATHYRIODISM

Primary Hyperparathyroidism occurs two or 4 times more often in women than in men. Rare in children younger than 15 years of age, but it increases 10 fold between the ages of 15 and 65 years. Half of the people diagnosed with hyperparathyroidism do not have symptoms. Secondary hyperparathyroidism, with manifestation similar to those of primary hyperparathyroidism occurs in patients who have

chronic renal failure and so called ***renal rickets*** as a result of phosphorous retention, increased stimulation of the parathyroid glands and increased parathyroid hormone secretion.

Clinical Manifestations

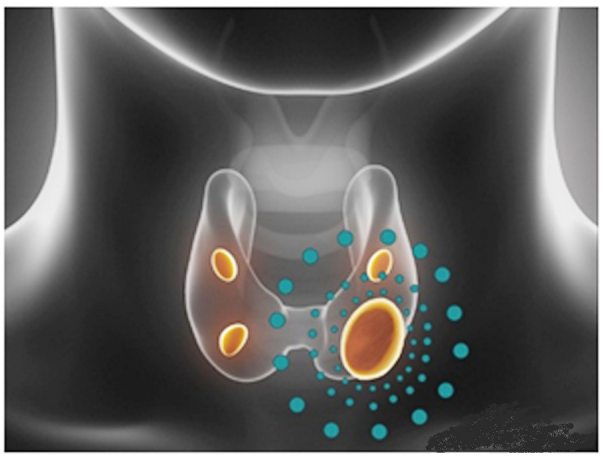

Fig. 1: Showing enlarged parathyroid gland (***Source:*** https://www.hyperparathyroidmd.com/hyperparathyroidism/

- Patient may have no symptoms or may experience signs and symptoms resulting from involvement of several body systems.
- Apathy, fatigue, muscle weakness, nausea, vomiting, constipation, hypertension and cardiac dysrhythmias may occur.
- All these signs are attributable to the increased concentration of calcium in the blood.
- Psychological effects may vary from irritability and neurosis to psychoses caused by the direct action of calcium on the brain and nervous system.
- An increase in calcium produces a decrease in the excitation potential of nerve and muscle tissue.
- The formation of stones in one or both kidneys related to the increased urinary excretion of calcium and phosphorous is one of the important complications of hyperparathyroidism and occurs in 55% of patients with primary hyper parathyroidism.
- Renal damage results from the precipitation of calcium phosphate in the renal pelvis and parenchyma which causes renal calculi, obstruction, pyelonephritis and renal failure.
- Musculoskeletal symptoms accompanying hyperparathyroidism may be caused by demineralization of the bones or by bone tumors composed of benign giant cells resulting from over growth of osteoclasts.
- The patient may develop skeletal pain and tenderness especially of the back and joints, pain on weight bearing, pathological fracture, deformities and shortening of body stature.
- Bone loss attributable to hyperparathyroidism increase the risk of fracture.
- The incidence of peptic ulcer and pancreatitis is increased with hyperparathyroidism and may be responsible for many of the GI symptoms that occur.
- Due to increased serum calcium levels which leads to increased serum levels of gastrin and acetylcholine. These changes result in stimulation of gastric acid secretion and peptic ulcer disease.

ASSESSMENT & DIAGNOSTIC FINDINGS

Primary hyperparathyroidism is diagnosed by persistent elevation of serum calcium levels and an elevated concentration of parathoromone. An elevated serum calcium level alone is a non-specific finding because serum levels may be altered by diet, medications and renal and bone scans in advanced disease. The double antibody parathyroid hormone test is used to distinguish between primary hyperparathyroidism and malignancy as a cause of hypercalcemia. Ultrasound, MRI, thallium scan and fine needle biopsy have been used to evaluate the function of the parathyroid and to localize parathyroid cysts, adenomas or hyperplasia.

Thallium Scan

- A radioactive substance, Thallium is injected at peak exercise (or when typical chest pain develops). Exercise is continued for one minute after injection to ensure maximum myocardial extraction.
- The patient is scanned within 10 minutes.
- The ECG is monitored for the 12 minutes of the procedure.

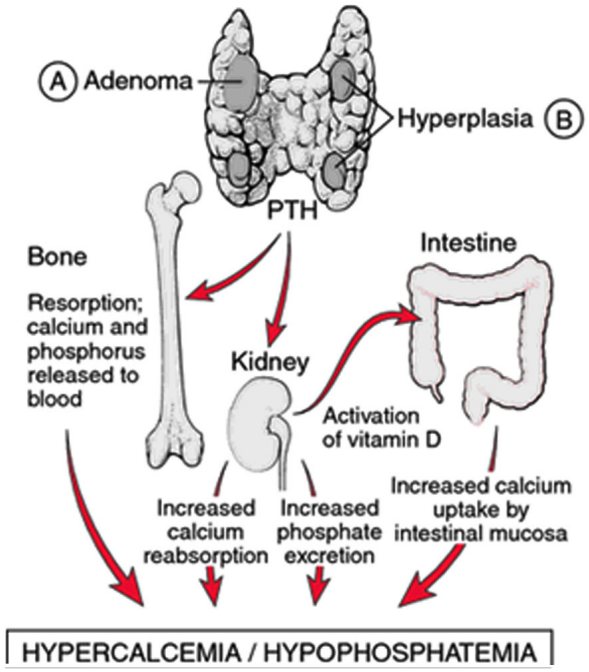

Fig. 2: Showing process of hypercalcemia and hypophosphatemia

SURGICAL MANAGEMENT

Recommended treatment is the surgical removal of abnormal parathyroid tissue. Previously, standard. Parathyroidectomy involved a bilateral neck exploration under general anesthesia. Nowadays, minimally invasive parathyroidectomy techniques allow for unilateral neck exploration using local anesthesia, these are performed on an outpatient basis. In some cases only the removal of a single diseased gland is necessary, reducing morbidity rates associated with surgery. For asymptomatic patients who have only mildly elevated serum calcium concentrations and normal renal function, surgery may be delayed and the patient monitored closely for worsening of hypercalcemia, bone deterioration, renal impairment or the development of kidney stones. Surgery is recommended for asymptomatic patients who meet the following criteria:

1. Younger than 50 years of age
2. Unable or unlikely to participate in follow up care
3. Serum calcium levels more than 1.0 mg/dl (0.25mmol/L) above normal reference range.
4. Urinary calcium levels greater than 400 mg/day
5. A 30% or greater decrease in renal function
6. With the complaints of primary hyper parathyroidism including nephrocalcinosis, osteoporosis or severe psychoneurologic disorder.

HYDRATION THERAPY

- Because kidney involvement is possible, patients with hyper parathyroidism are at risk for renal calculi.
- Therefore a daily fluid intake of 2000 mL or more is encouraged to help prevent calculus formation.
- Cranberry juice is suggested because it may lower the urinary pH.
- Cranberry extract tablets are an alternative to reduce urinary pH
- Patient is instructed to report other manifestations of renal calculi, such as abdominal pain and hematuria.
- Thiazide diuretics are avoided, because they decrease the renal excretion of calcium and further elevate serum calcium levels.
- Because of the risk of hypercalcemic crisis, the patient is instructed to avoid dehydration and to seek immediate health care if conditions that commonly produce dehydration (e.g. vomiting, diarrhea) occur.

Mobility

Mobility of the patient, with walking or use of a rocking chair for those with limited mobility, is encouraged as much as possible because bones that are subjected to normal stress give up less calcium. Bed rest increases calcium excretion and the risk for renal calculi. Oral phosphates lower the serum calcium level in some patients, long term use is not recommended because of the risk of ectopic calcium phosphate deposition in soft tissues.

Diet and Medications

Nutritional needs are met, but the patient is advised to avoid a diet with restricted or excess calcium. If the patient has a coexisting peptic ulcer, prescribed antacids are necessary. Because anorexia is common, efforts are made to improve the appetite. Stool softeners and physical activity, along with increased fluid intake, help offset constipation which is common post-operatively.

Complications: Hypercalcemic Crisis

Acute hypercalcemic crisis can occur with extreme elevation of serum calcium levels. Serum calcium levels greater than 15 mg/dl result in neurologic, cardiovascular and renal symptoms that can be life threatening. Treatment includes rehydration with large volumes of IV fluids, diuretics agents to promote renal excretion of excess calcium, and phosphate therapy to correct hypo phosphatemia and decrease serum calcium levels by promoting calcium deposition in bone and reducing gastrointestinal absorption of calcium. Cytotoxic agents (e.g. mithramycin), calcitonin and dialysis may be used in emergency situations to decrease serum calcium levels quickly. A combination of calcitonin and corticosteroids has been administered in emergencies to reduce the serum calcium level by increasing calcium deposition in bone. Other agents may be administered to decrease serum calcium levels include bisphosphonates.

CONCLUSION

Hyperparathyroidism is a condition in which one or more of the parathyroid glands is overactive and makes excess parathyroid hormone, regardless of the level of calcium in the body, which this hormone normally regulates. In other words, the parathyroid glands continue to make large amounts of parathyroid hormone even when the calcium level is normal and they should not be making hormone at all. Over production of parathyroid hormone by overactive parathyroid glands can rob you of your health. It can make you feel run down and tired and cause osteoporosis, kidney stones, and many other serious problems. Hyper parathyroidism can be fixed in most people with newer minimally invasive surgery techniques in less than 20 minutes.

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Sheer Sinisterness of Uncontrolled Diabetes Mellitus: Parapharyngeal Abscess

Apoorva P[1], Rajesh Radhakrishna Havaldar[2], Shilpa Mallapur[3], Redkar Aditya Achyut[4], Neema K[5]

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ABSTRACT

Diabetes mellitus is a common disease in developing countries and the complications are feared by many physicians and surgeons. One such rare and dangerous complication is parapharygeal abscess which can spread via its intricate anatomical site to other deep neck spaces. We present the case of a young male who is a known uncontrolled diabetic presented to us with neck swelling and toxic symptoms and managed successfully with timely surgical intervention by exploring all neck planes to evacuate purulent material thus speeding up the recovery process.

Keywords: Parapharyngeal space; Diabetes Mellitus; Klebsiella; Abscess; DNSI.

INTRODUCTION

Deep neck space infections involve fascial planes and spaces of the head and neck. Because of complications related to the compression of upper airway, sepsis or septic shock or acute respiratory syndrome they can be life-threatening.[1] Incidence of deep neck space infections are higher in cases of immunocompromised individuals and diabetics.

This is a case report of a patient with uncontrolled Diabetes Mellitus presenting as a case of deep neck space infection.

CASE REPORT

A 28 year old Male patient, belonging to socio-economic Class I as per Modified BG Prasad's Classification May 2022[2] presented to ENT OPD with swelling over right lateral aspect of the neck since 5 days and pain while swallowing accompanied by fever with chills. There is no history suggestive of any dental infection. Patient is a known diabetic since 5 years, not on regular medications. His Random Blood Sugar level was 352 mg/dl at presentation.

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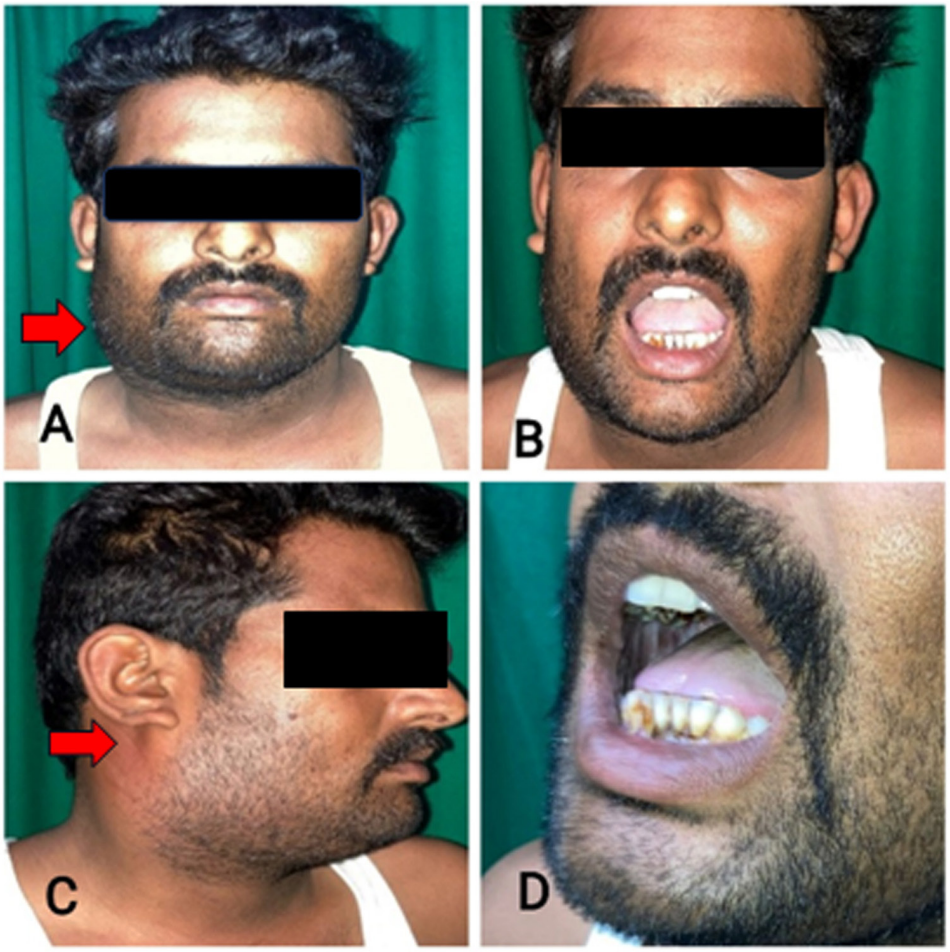

Fig. 1: Pre-operative image

On detailed clinical examination, a diffuse swelling measuring 3x5 cm was noticed at right lateral aspect of the neck in the upper cervical region pushing the lobule of the pinna superiorly and anteriorly (Fig. 1A, 1C). Skin over the swelling was warm to touch, tender, tense and reddish in colour (Fig. 1C). Patient's mouth opening was restricted to two fingers (Fig. 1B) and had a foul smelling breath and a poor oral hygiene. Teeth were stained because of chronic tobacco consumption (Fig. 1D). On oropharyngeal examination, the anterior tonsillar pillar of the right side along with the right tonsillar fossa and lateral pharyngeal wall was pushed medially. Posterior pharyngeal wall was congested.

All routine blood investigations were sent and patient was started on intravenous broad spectrum antibiotics as empirical therapy covering wide range of Gram positive and Gram negative organism.

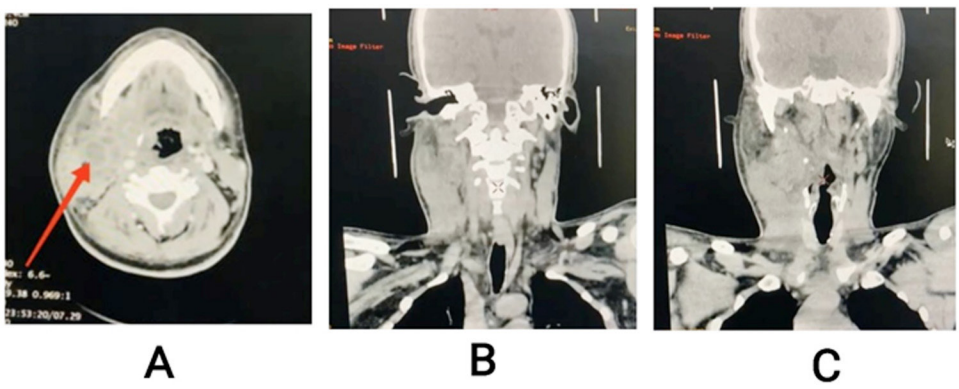

Fig. 2: Contrast enhanced Computed Tomography of neck – Axial and coronal views

A contrast enhanced CT scan of the neck was performed, showing conglomerated lymph node mass with areas of necrosis in the right level 2 region, medially extending into parapharyngeal space, laterally till sternocleidomastoid muscle with multiple sub-centimetric nodes involvement of all levels, encasement of carotid vessels on right side, compressing the right internal jugular vein.

Insulin was started in view of raised Glycosylated Haemoglobin (HbA1C – 12%) and due to presence of ketone bodies in the urine. Ophthalmology reference was given and Diabetic Retinopathy was ruled out.

Patient was shifted to the operation theatre and under general anaesthesia, Incision and Drainage of the abscess was done after making an incision over the area of maximal fluctuance. Following evacuation of the abscess contents, the abscess wall was curetted, and loculations between adjacent neck spaces were broken down by digital dissection. Pus was sent for culture and sensitivity testing and histopathological evaluation and result was awaited. The cavity was packed by medicated ribbon gauze. Intraorally, the tonsil that was pushed medially returned to it's normal position. Hence, intraoral exploration was not done.

Daily dressings of the cavity were done with mixture of Metronidazole Infusion IP (0.5%w/v) and Povidone Iodine Solution IP (10%w/v)wash and ribbon gauze soaked with Povidone Iodine Solution IP (10%w/v) was packed into the cavity. The edges of the cavity were digitally manipulated to drain out any residual pus. On post operative day 3 through microbiological reports we learnt that Gram staining of the pus showed plenty pus cells with Gram positive Bacilli and Gram negative Bacilli and Gram positive cocci.

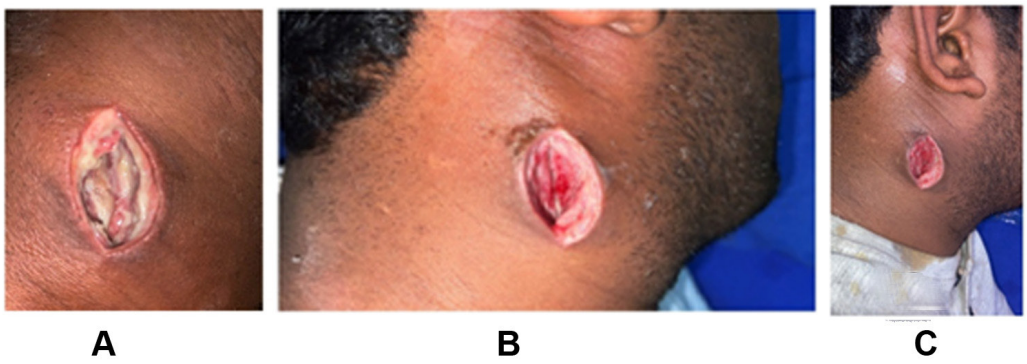

Fig. 3A: Wound image on post-operative Day 7. **B:** Wound image on Post-operative day 12. **C:** Wound image on Post-Operative Day 15.

On post-operative day 7, report for culture and sensitivity was available according to which the isolated microorganism was Klebsiella pneumoniae and was sensitive to most of the preferred antibiotics.

Regular dressings were done for a period of 28 days till the borders of the cavity naturally approximated following which secondary suturing was done. Raised blood sugar levels were managed by physicians throughout the hospital stay using fixed dose insulin and oral hypoglycemic agents. Patient was discharged on oral antibiotics on post operative day 29 and came for follow up on post operative day 40 showing drastic clinical and symptomatic improvement. (Fig. 4)

Fig. 4: Follow up clinical photograph on Post-Operative Day 40.

DISCUSSION

A combination of fever, enlargement or tenderness of the neck mass, and overlying erythema indicates that the mass belongs to an inflammatory etiology like deep neck space infections. The most prevalent cause of DNSI happens to be tonsillitis among children whereas Dental infections in adults.[1,9] DNSI often starts as cellulitis of soft tissue involving an isolated region adjacent to the main source of infection, however the further spread is kept in check by body's natural defence system and the neck's fascial layers. However, in cases of diabetic individuals DNSI has extensive inflammation and tend to spread in the neck with alarming seriousness.[3] In uncontrolled diabetics there is decreased bactericidal action due to declined neutrophil function leading to impaired phagocytosis. Systemic hyperglycemia tends to hamper the functioning of normal immune system such as neutrophil function, cellular immunity, complement fixation which plays a major role to keep the spread of infections in control.[4] According to the study by Gino Marioni *et al*, in a series of 233 cases of DNSI most commonly isolated organism was found to be Gram positive anaerobic cocci followed by Steptococcus group of pathogens.[5] According to a case series of 54 patients with DNSI done in New Delhi by R Meher, A Jain *et al* most positive cultures were polymicrobial and the most common single isolated species were found to be of Staphylococcus Aureus.[6] The organism isolated in this case was Klebsiella pneumoniae which also according to Huang TT *et al* who performed his study in Taiwan, becomes the most common bacteria isolated in DNSI in diabetics.[7] CT Scan is the most preferred radiological investigation for deep neck space infections. MRI are generally not done as they are costlier and take more time to finish the scan[8] According to Case series on 173 patients done by Bakir S *et al*, as per radiological corelation the most common involved site was the submandibular space followed by the peritonsillar space and the third commonly involved being the parapharyngeal space.[9] In this case, the space involved is the parapharyngeal space. Contrast enhanced CT (CECT) scan is sensitive (91%) for the identification of extent of the deep neck infections and to distinguish cellulitis from abscess. Contrast enhanced CT helps to decide whether surgical intervention is needed or not as cases with findings of cellulitis respond to medical treatment, whereas those with abscesses are aggressive, and may require surgical treatment.[9] In deep neck space infections penicillin with a b-lactamase inhibitor (eg. amoxicillin with clavulanic acid) or a b-lactamase resistant antibiotic (such as cefuroxime, imipenem, or meropenem) in combination with a drug that is highly effective against most anaerobes (such as metronidazole) is recommended for optimal empirical coverage.[10] Combined intra oral drainage was not done in this case fearing the complication of fistula which can form, hence only external drainage was preferred in this case. Also the return of the tonsil to it's normal position further gave us an indication that all pockets were drained adequately. Parhiscar A, Har-El recommend early surgical drainage as the standard treatment choice for all cases of DNSI[11] however, many authors like Eftekharian A, Roozbahany NA *et al* believe that surgical treatment is needed in only a few cases where as many of the cases are treated with intravenous antibiotic coverage.[12] Based upon this study and through previous article search on DNSI we propose that geographical location does play a role in deciding the prognosis of the problem and also in terms of the expected microbiological variant. The duration of hospital stay also corresponds to one's pre-disease glycaemic control. Patients of uncontrolled diabetes mellitus with higher glycaemic index tend to have a longer hospital stay, like this patient in the study who had a total hospital stay of 29 days.

CONCLUSION

Deep Neck Space Infections is a serious problem which requires immediate surgical intervention. Proper surgical drainage and removing all loculi completely ensures faster recovery and healing of the wound. Radiological imaging is helpful as in this case and thus the approach was planned better and the potential complication can be averted. Good glycaemic control is needed for optimum control in known diabetics. Oral hygiene is of utmost importance to prevent such an issue. Proper antibiotic coverage for both aerobic and anaerobic organisms is needed during the course of hospital stay and also after discharge. Standard dressing protocols must be followed while addressing the wound. Regular dressing must be done under all aseptic precautions.

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Management of Lysol Burn in Tertiary care Hospital: Our Experience

Yadunand Sriram[1], Ravi Kumar Chittoria[2], Barath Kumar Singh P[3]

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ABSTRACT

Lysol is a derivative of phenol and is alkaline in nature. Lysol produces liquefactive necrosis after contact with skin by producing protein denaturation and saponification of fats. In this article we are sharing our experience of managing a case of Lysol burns with various regenerative therapies.

Keywords: Lysol burn; Management; Alkali burns; Face.

INTRODUCTION

Phenol (carbolic acid) is one of the antiseptic agents. Currently it is used as a disinfectant, chemical intermediate and nail cauterizer. Phenol is a general protoplasmic poison (denatured protein) with corrosive local effects. Phenol derivates are less toxic than pure phenol. The lethal dose is between 3 to 30 g, but may be as little as 1 g. Lysol is a derivative of phenol with 50% solution of cresol (3-methyl phenol) in saponified vegetable oil. It is commonly used as a disinfectant or toilet cleaner. It has toxicity both in ingestion as well as topical exposure.[1] In this case report, we share our experience of managing the Lysol burns in our patient.

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MATERIALS AND METHODS

This study was conducted in the Department of Plastic Surgery in a tertiary care center in South India after obtaining the departmental ethical committee approval. Informed written consent was taken from the patient. 23-year-old female Patient had alkali burn injury when accidental splash of disinfectant in restroom wherein she sustained injuries to face, eyes and chest. (Fig. 1)

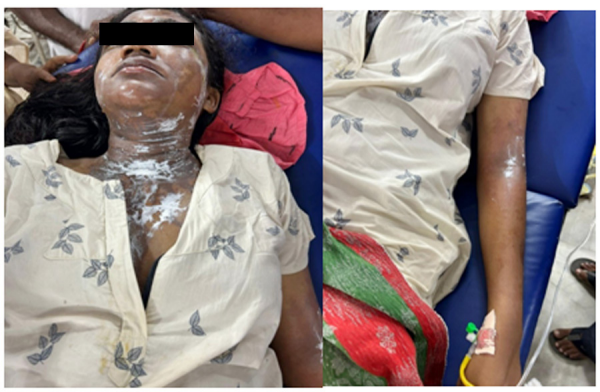

Fig. 1: Alkali Chemical burns at presentation

Patient was admitted with the above symptoms and managed according to WHO burn protocol. She underwent Wound debridement and hydrotherapy. The wounds were managed with Regenerative therapies like collagen scaffold application, feracrylum, , Low Level Laser Therapy Autologous Platelet Rich Plasma, Regenerative Scaffold, Negative Pressure wound therapy (Fig. 2-7).

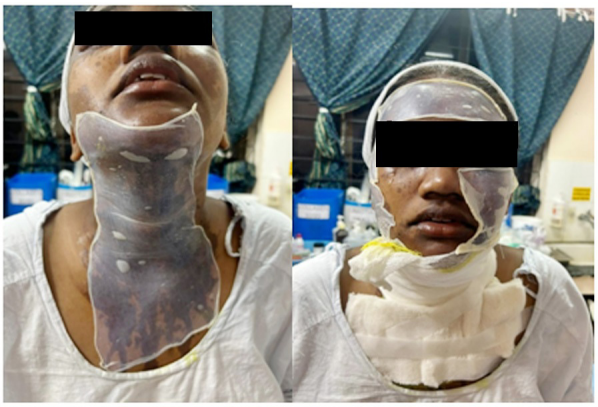

Fig. 2: Collagen Application

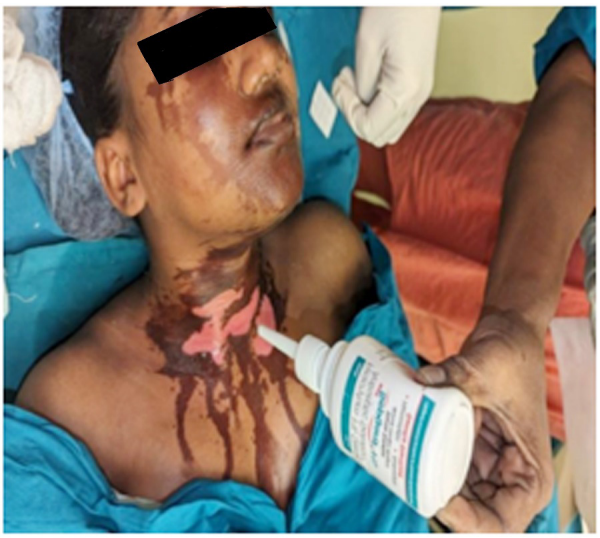

Fig. 3: Feracrylum 1% in Burn wounds

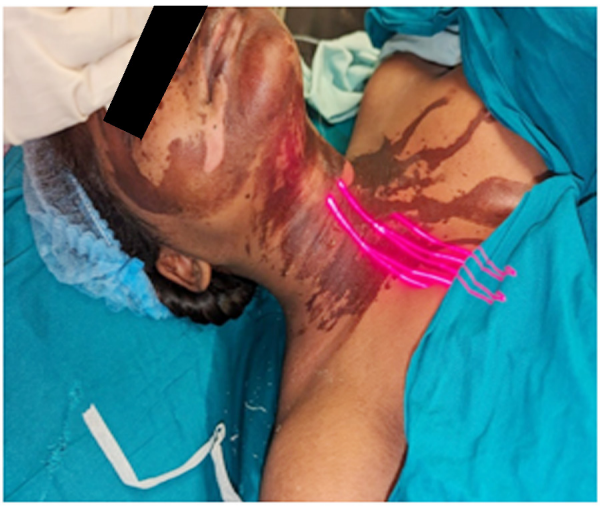

Fig. 4: Low level laser therapy for the burn wound

Fig. 5: Autologous platelet rich plasma therapy

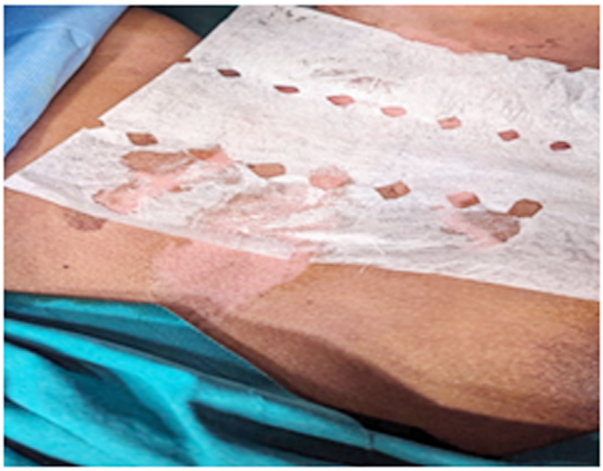

Fig. 6: Collagen application

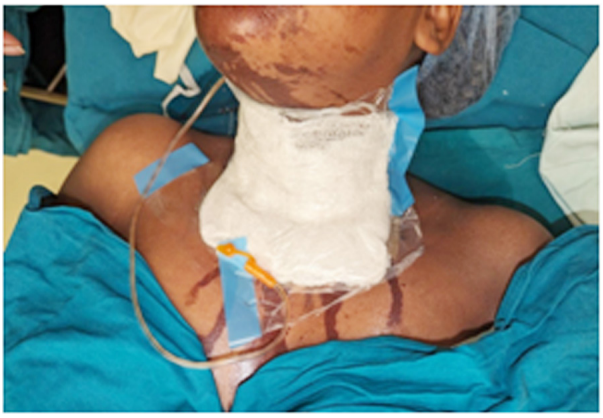

Fig. 7: Negative Pressure Wound Therapy

Once wound showed promising signs of healing and epithelization was complete, we applied Silicone gel and silicon sheet (Fig. 8 and 9) over the healed wound to prevent the abnormal scarring. We also used low-level laser therapy and autologous platelet rich plasma for scar management. (Fig. 10 and 11)

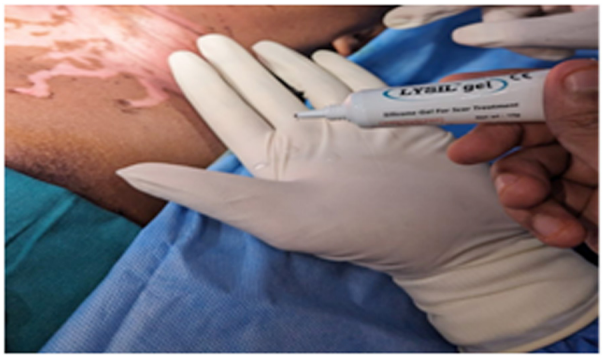

Fig. 8: Silicone gel application for scar

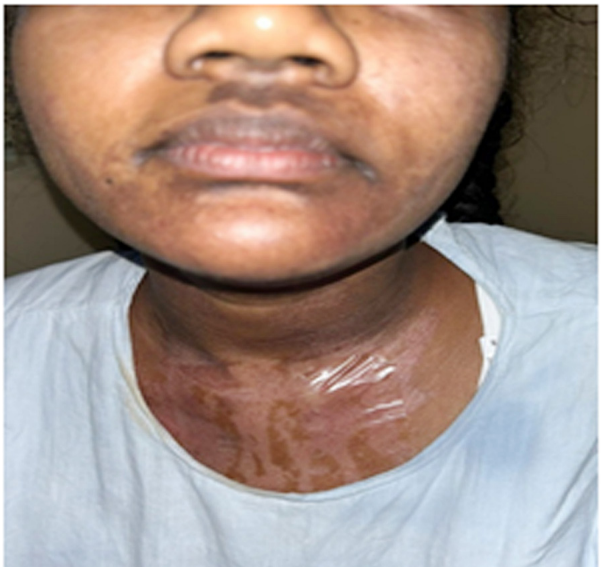

Fig. 9: Silicone sheet application

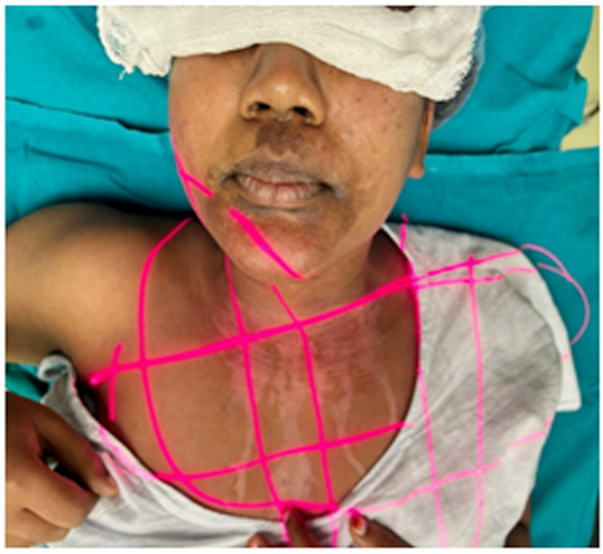

Fig. 10: Low-Level laser therapy for scar

Fig. 11: Autologous platelet rich plasma for scar

RESULTS

Patient burn wounds healed and there was minimal abnormal scarring and no hypertrophic scarring at the time of discharge. (Fig. 12). Vancouver Scar Scale - 5/13 at the time of discharge.

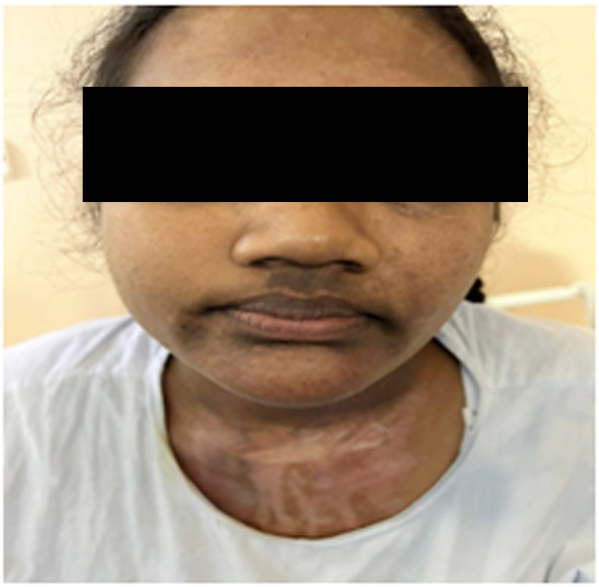

Fig. 12: Healed burn wound at discharge

DISCUSSION

Lysol is derivative of phenol and is used frequently in industries and as disinfectants in households. Instead of causing a hyperthermic injury, most chemical agents harm the skin by causing a chemical reaction. Even though some chemicals cause significant heat when they come into contact with water due to an exothermic reaction, the majority of skin damage is caused by chemicals due to their direct chemical alterations on the skin. Depending on the agent acids, alkalis, corrosives, oxidizing and reducing agents, desiccants, vesicants, and protoplasmic poisons different chemical changes may occur. Dermal exposure produces lesions which are initially painless white patches and later turn erythematous and finally brown. Phenol produces mucosal burns and coagulum. They cause eye irritation and corneal damage. When ingested, it causes extensive local corrosions, pain, nausea, vomiting, sweating, and diarrhea. Severe gastrointestinal burns are uncommon and strictures are rare. Inhalation produces respiratory tract irritation and pneumonia. Systemic manifestations develop after 5 to 30 minutes post ingestion or post dermal application, and may produce nausea, vomiting, lethargy or coma, hypotension, tachycardia or bradycardia, dysrhythmias, seizures, acidosis, hemolysis, methemoglobinemia, and shock.[2] The degree of skin degradation is mostly determined by the toxic agent's concentration and length of contact.[3] Lysol is alkaline in nature and produces liquefactive necrosis on contact with skin causing protein denaturation and saponification of fats.[4] Autologous platelet rich plasma is an upcoming and proven treatment modality for patients with burn injuries wherein concentrated platelet preparations are used which enhance body regenerative process. Several kinds of bioactive mediators, including immunological mediators, clotting factors, chemokines, integral membrane proteins, adhesion proteins, growth factors, and clotting factors, are stored and prepared to react to tissue injury inside the cytoplasmic granules of platelets. In wounds treated with PRP, the bioactive mediators have a favorable impact on cellular development, proliferation, differentiation, and re-epithelialization by promoting angiogenesis, mitogenesis, and controlling the endogenous inflammatory process.[5]

The advantage of allogeneic PRP is that it can be obtained from willing blood donors, and its derivatives can be used right away without the requirement for clinicians to get a patient sample. In some clinical circumstances, such as those involving acute burns when patients may be fluid depleted and thrombocytopenic, this may be helpful. Other conditions that preclude the creation of PRP include hemophilia, sepsis, or infection; related contraindications include the use of NSAIDs or corticosteroids, tobacco usage, malignancies, and anemia.

LLLT, which can trigger photochemical reactions in tissue and cells, is sometimes referred to as biological stimulation or photobiological regulation. Previous research has demonstrated that LLLT affects the photoreceptors on mitochondria, stimulates the electron transport chain of produced energy, enhances mitochondrial respiration, and boosts the synthesis of adenosine triphosphate (ATP). As a result, LLLT has the ability to change the cellular redox state and to trigger the activation of signaling pathways that drive transcription factors involved in proliferation, tissue repair, and regeneration.[6]

The various dressings and tissue-engineered constructions used in burn therapy depend heavily on biomaterials. The major goal of employing them is to mimic the skin's ECM, which is composed of laminin, elastin, collagen, and proteoglycans. Laminin gives the skin strength, while proteoglycans give it moisture and viscosity. Biomaterials of diverse origins are employed in skin grafts and substitutes, and the decision made during scaffold manufacturing is crucial because it can affect in situ regeneration. These materials' characteristics control cell behavior and facilitate the development of new tissue. Biodegradability, momentary mechanical support, and permeability are the primary needs. Scaffolds can be either with or without cells, and the latter can be further broken down into dermal, epidermal, and epidermal dermal composites depending on the methodology.[7] According to theory, negative pressure might generate an interstitial gradient shift that can reduce oedema and, as a side effect, promote cutaneous perfusion, facilitating the evacuation of blood or serous fluid. Additionally, it is hypothesized that NPWT's capacity to generate a mechanical stress or force that directly influences cellular activity, particularly the growth of new blood vessels, may help slow the advancement of burn wounds. Additionally, it may be desirable to maintain a wet environment that offers ideal circumstances for epithelialization and prevents tissue desiccation.[8]

The skin surface temperature of hypertrophic

burn scars under SGS is increased by 1.7°C, and temperature increases of this magnitude can significantly increase collagenase activity and could affect scarring. As a result, it is possible that an increase in skin surface temperature is involved in the mechanism of action of silicone based products for scar management. Because it has been suggested that the negative static electric field produced by friction between SGS and the skin may cause collagen realignment and lead to the involution of scars, the development of a static electric field may also be implicated.[9]

The way feracrylum works is by building water insoluble multi-complexes with different proteins, including those found in blood. The hemostatic effect of feracrylum is given through the creation of a synthetic complex on the wound surface that consists of its adduct with plasma proteins, primarily albumin. The in-vitro mixture of feracrylum and serum albumin results in a substantial rubbery clot. The feracrylum albumin combination degrades over time like all other biodegradable polymers. After that, these subunits are ejected. The benefit of feracrylum is that it combines antibacterial activity with little local toxicity or irritation, making it useful in preventing acute, chronic, and hospital infections, especially in post-operative wounds, and facilitating wound healing.[10]

CONCLUSION

With advent of newer technologies, treatment of chemical burn wounds has been much more streamlined and produce better results in patients. In our experience we have seen better wound healing in patient with chemical burns with minimal scarring. However large randomized control trials are necessary to establish association between the same.

Conflicts of interest: None

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The second page should carry the full title of the manuscript and an abstract (of no more than 150 words for case reports, brief reports and 250 words for original articles). The abstract should be structured and state the Context (Background), Aims, Settings and Design, Methods and Materials, Statistical analysis used, Results and Conclusions. Below the abstract should provide 3 to 10 keywords.

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State the background of the study and purpose of the study and summarize the rationale for the study or observation.

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Include summary of key findings (primary outcome measures, secondary outcome measures, results as they relate to a prior hypothesis); Strengths and limitations of the study (study question, study design, data collection, analysis and interpretation); Interpretation and implications in the context of the totality of evidence (is there a systematic review to refer to, if not, could one be reasonably done here and now?, What this study adds to the available evidence, effects on patient care and health policy, possible mechanisms)? Controversies raised by this study; and Future research directions (for this particular research collaboration, underlying mechanisms, clinical research). Do not repeat in detail data or other material given in the Introduction or the Results section.

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Standard journal article

[1] Flink H, Tegelberg Å, Thörn M, Lagerlöf F. Effect of oral iron supplementation on unstimulated salivary flow rate: A randomized, double-blind, placebo-controlled trial. J Oral Pathol Med 2006; 35: 540-7.

[2] Twetman S, Axelsson S, Dahlgren H, Holm AK, Källestål C, Lagerlöf F, *et al*. Caries-preventive effect of fluoride toothpaste: A systematic review. Acta Odontol Scand 2003; 61: 347-55.

Article in supplement or special issue

[3] Fleischer W, Reimer K. Povidone iodine antisepsis. State of the art. Dermatology 1997; 195 Suppl 2: 3-9.

Corporate (collective) author

[4] American Academy of Periodontology. Sonic and ultrasonic scalers in periodontics. J Periodontol 2000; 71: 1792-801.

Unpublished article

[5] Garoushi S, Lassila LV, Tezvergil A, Vallittu PK. Static and fatigue compression test for particulate filler composite resin with fiber-reinforced composite substructure. Dent Mater 2006.

Personal author(s)

[6] Hosmer D, Lemeshow S. Applied logistic regression, 2nd edn. New York: Wiley-Interscience; 2000.

Chapter in book

[7] Nauntofte B, Tenovuo J, Lagerlöf F. Secretion and composition of saliva. In: Fejerskov O,

Kidd EAM, editors. Dental caries: The disease and its clinical management. Oxford: Blackwell Munksgaard; 2003. p. 7-27.

No author given

[8] World Health Organization. Oral health surveys - basic methods, 4th edn. Geneva: World Health Organization; 1997.

Reference from electronic media

[9] National Statistics Online—Trends in suicide by method in England and Wales, 1979-2001. www.statistics.gov.uk/downloads/theme_health/HSQ20.pdf (accessed Jan 24, 2005): 7-18. Only verified references against the original documents should be cited. Authors are responsible for the accuracy and completeness of their references and for correct text citation. The number of reference should be kept limited to 20 in case of major communications and 10 for short communications.

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