CONTENTS

EDITORIAL

REVIEW ARTICLE
Point-of-care testing in the existing milieu of critical care: Miracle or mirage?
Dheeraj Kapoor, Pritam Singh, Meghana Srivastava, Manpreet Singh, Jasveer Singh
1-5

Seizure exacerbation by antiepileptic drugs: a less recognized entity
Chandrika Azad, Sukhwinder Singh
6-9

Managing epilepsy in pregnancy
Shikha Rani, Anju Huria, Poonam Goel
10-15

Pressure ulcers management and practice guidelines in critical care settings
Poonam Bhullar, Dheeraj Kapoor, Sudha Sharma, Manpreet Singh
16-20

BRIEF COMMUNICATION
Recent changes in regulation of clinical trials in India: an update for investigators
Jagjit Singh, Rajiv Kumar
21-23

CASE REPORT
Large cervical leiomyoma posing diagnostic dilemma
Sunita Arora, Rupneet Kaur, Poonam Goel, Rimpy Tondon, Sonil Prabhakar, Narinder Kaur
24-26

Initial management of complete laryngotracheal separation: a case report
Hitesh Verma, Arjun Dass, Surinder K Singhal, Nitin Gupta
27-30

Mucocoele of the junction of hard and soft palate - unusual location
Meenakshi Awana, Anand Gupta, Gurvanit Lehl, Varun Hatwal
31-33

Neglected post-burn severe joint contractures of hand - rehabilitation by surgical management - a case report
Ravi Gupta, Rajiv Gupta
34-37

Post human bite pinna reconstruction: a case report
Surinder K Singhal, Shashikant Pol
38-41

Mandibular reconstruction- a case report and rare experience
Hitesh Verma, Arjun Dass, Surinder K Singhal, Nitin Gupta, Mohit Bhattani
42-44

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Editorial

Point of Care testing-A new technology in modern era!

Point-of-care testing (POCT) is an important diagnostic tool used in various locations in the hospital, especially in intensive care unit (ICU), the operating room (OR), and emergency set-ups. Laboratory test results are often pivotal to fast decisions in majority of areas where patients are critical.

Testing provides doctors with valuable knowledge about the emergency in the patients so that appropriate therapeutic interventions can be made quickly. Huge efforts have been made to decentralize laboratory testing, especially point-of-care testing (POCT) in critical care settings (eg, ICU, OR), high dependency units and emergency settings where rapid therapeutic turnaround time is required. Point-of-care testing is a newer concept and is defined as “testing at or near the site of patient care whenever the medical care is needed.”\(^1\) the patient’s condition, so that this information can be integrated into appropriate treatment decisions that improve patient outcomes, that is, reduce patients’ criticality, morbidity, and mortality. Point-of-care testing can be performed in different environments, such as in the hospital, at home, or at other locations.

Point of care testing has been advantageous in diagnosis of various organ injuries, physiological and biochemical derangements at an earliest. It includes arterial blood gases, electrolytes, blood glucose testing, serum biomarkers for renal injuries, cardiac injuries, septicaemia, coagulation derangements etc. Point-of-care instruments differ by their method of testing. For example, whole-blood glucose meters are categorized as “electrochemical biosensor,” “reflectance photometry,” or “absorbance photometry.” These instruments are further differentiated by the type of chemistry used to measure the glucose: either glucose oxidase or glucose dehydrogenase enzymes. However, The purpose of POCT is to provide immediate information to physicians about some types of POCT may be controversial because of concern about the accuracy and performance of instruments when used with critically ill patients (ie, glucose meter testing). Point-of-care testing undoubtedly will take a more active role in upcoming critical care, emergency care and operative room settings in near future. It will be used more for on-site diagnostic testing and trend monitoring of patient conditions in various places of hospital settings. This is only because of shorter therapeutic turnaround time, and bidirectional connectivity of transportable, portable, and handheld devices.

While POCT may not necessarily replace centralized laboratory testing completely but it is definitely becoming an important modality for improving patient care and outcomes. To determine this, other factors must be considered, including advantages and disadvantages, analysis of test accuracy, clinical impact, and cost benefit ratio. A strong multidisciplinary and interdepartmental team approach is necessary to ensure cost effectiveness, better analytic performance, reliability, comparability, and quality control.\(^2\) Point of care testing requires some basic training to ensure a good quality service and this testing has shown to reduce hospital stay, improve adherence to treatment, and reduce complications. Although point of care testing is more expensive than laboratory testing, it produces wider economic benefits.

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Point-of-care testing in the existing milieu of critical care: Miracle or mirage?

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INTRODUCTION

Point-of-care testing (POCT) is one of the emerging concepts of decentralised, patient-centric healthcare delivery in the field of critical care medicine. Conceptually it is “testing at or near the site of patient care whenever the medical care is needed”.¹ The paradigm of POCT in the critical care has made the entire process of laboratory testing accessible at the bedside. Clinicians may thus have immediate access to battery of targeted laboratory parameters for diagnostic and therapeutic interventions and strategies as well as for predicting the morbidity and mortality in patients admitted in intensive care units (ICU) and hence benefitting the overall clinical outcome.

TYPES OF POINT-OF-CARE INSTRUMENTS

Point-of-care instruments can be classified as “transportable,” “portable,” or “handheld,” devices.¹ Transportable and portable devices, also called as table or bench-top instruments, are miniaturized forms of classical laboratory instruments. Many of them require unit use reagents, which mean that an individual portion of reagent is consume by a single measurement test.² Portable instruments are easily carry out with a built-in handle, while transportable equipment is usually carried on a cart.¹ Hand-held instruments utilize modern techniques of microfluidics and microsensors. They can determine one or several parameters quantitatively in different combinations. They have automatic calibration programs with a control system.²

Characteristics of contemporary point-of-care instruments²:

1. Able to measure more than 10 different tests per sample of blood.
2. Requires a small blood volume to perform a test (2.5 iL to 40 iL for single to multiple measurements).
3. Rapid analysis time (15 to 45 seconds for a single to multiple parameter test measurement).
4. Provides selective testing (operator may select the tests to be performed).
5. Advanced quality management features (automatic internal calibrations at assorted time intervals).
6. Safety features (like password option).

Areas for point of care testing

The POCT tests can be perform in numerous areas in hospitals and pre-hospital settings (Table 1).

Current POCT variables available in critical care settings¹-³,⁵

POCT variables comprise of blood glucose, electrolytes, hematocrit, renal function tests, blood gas analysis, lactate levels, urine ketones and others such as cardiac troponins, BNP/pro-BNP levels and d-dimer assays etc (Table 2). While it is perceived that use of POCT will help clinicians in better medical management of patients and cutting down the overall treatment costs, there are concerns regarding its reliability and reproducibility especially when performed by non-laboratory healthcare providers.

Point-of-Care Testing: is it a miracle or still a mirage?

Table 3 summarizes the potential advantages and disadvantages of modern POCT in the existing health care system. The aspect of POCT that has the most robust evidence in literature is its time effectiveness. Currently available POCT devices have analysis time as short as 45 seconds to 2 minutes. POCT is more time-effective in delivering test results when compared to conventional or central laboratory testing (CLT). POCT has a shorter therapeutic turnaround time (TATT) i.e. shorter time duration from ordering of blood
investigations to final therapeutic decision based on the interpretation of test results delivered. However, the evidence for the clinical impact of POCT has been less than unequivocal. Moreover, different studies have evaluated selective patient groups, limiting their extrapolation to routine patients in emergency or critical care settings. Studies performed on POCT-cardiac biomarkers in ER resulted in reduced length of hospital stay and reduced admission rates to cardiac care units. It also facilitated rapid transfer to step-down units among admitted patients.

Although, length of stay has other contingencies like availability of beds in step down units/wards, physicians practice style and dependency on other biochemical tests not being performed with POCT.

A potential advantage of POCT over CLT is reduction in pre-analytic and post-analytic errors (Table 4). The multi-staged procedure with CLT involving transport and

<table>
<thead>
<tr>
<th>Sites of use for point of care testing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>In-patient care</strong></td>
</tr>
<tr>
<td>- Intensive care units.</td>
</tr>
<tr>
<td>- Accident and emergency departments.</td>
</tr>
<tr>
<td>- Operating theatres and postoperative care unit.</td>
</tr>
<tr>
<td>- Renal dialysis units.</td>
</tr>
<tr>
<td>- Neonatal units.</td>
</tr>
<tr>
<td>- Special outpatient clinics.</td>
</tr>
<tr>
<td>- Research laboratories.</td>
</tr>
<tr>
<td>- General wards.</td>
</tr>
<tr>
<td>- Hospitals without central laboratories.</td>
</tr>
<tr>
<td><strong>Out-patient care</strong></td>
</tr>
<tr>
<td>- Ambulances.</td>
</tr>
<tr>
<td>- General practitioners, surgeries and primary health centers.</td>
</tr>
<tr>
<td>- House visits.</td>
</tr>
<tr>
<td>- Healthcare screening clinics.</td>
</tr>
<tr>
<td>- Out-patient nursing care.</td>
</tr>
<tr>
<td>- Pharmacies.</td>
</tr>
<tr>
<td>- Chronic care facilities.</td>
</tr>
<tr>
<td>- Old age homes.</td>
</tr>
<tr>
<td><strong>Special areas</strong></td>
</tr>
<tr>
<td>- Sports medicine.</td>
</tr>
<tr>
<td>- Military medical camps.</td>
</tr>
<tr>
<td><strong>Patient self monitoring</strong></td>
</tr>
<tr>
<td>- Glucose monitoring.</td>
</tr>
<tr>
<td>- Anticoagulant monitoring.</td>
</tr>
</tbody>
</table>

Table 2: Analytes that can be measure by point-of-care testing

<table>
<thead>
<tr>
<th>Analytes that can be measure by point-of-care testing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete blood count (CBC)</td>
</tr>
<tr>
<td>Three-part differential granulocytes, lymphocytes, monocytes, neutrophils, eosinophils and basophils.</td>
</tr>
<tr>
<td>Serum electrolytes (Calcium, magnesium, potassium, sodium, chloride, phosphate).</td>
</tr>
<tr>
<td>Arterial blood gas values and acid-base equilibrium (pCO2, pO2, SO2, pH, lactates).</td>
</tr>
<tr>
<td>Glucose, Blood Urea Nitrogen (BUN), Creatinine.</td>
</tr>
<tr>
<td>C-reactive protein (CRP) measurement before antibiotic treatment.</td>
</tr>
<tr>
<td>Glycosylated hemoglobin (HbA1c) determination in diabetics.</td>
</tr>
<tr>
<td>Cardiovascular diagnostic testing (cardiac troponins, D-dimers, natriuretic peptide (BNP/ NT pro-BNP)).</td>
</tr>
<tr>
<td>Serum albumin, alkaline phosphatase, amylase, bilirubin</td>
</tr>
<tr>
<td>Coagulation profile (Prothrombin Time: PT, Partial Tromboplastin Time: PTT, Activated Clotting Time: ACT)</td>
</tr>
</tbody>
</table>

Table 3: Advantages and disadvantages of Point-of-Care Testing

<table>
<thead>
<tr>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reduced therapeutic turnaround time (TATT) of diagnostic testing</td>
<td>Concerns about inaccuracy and imprecision</td>
</tr>
<tr>
<td>Rapid data availability</td>
<td>Quality of testing is operator-dependent: vulnerability of quality error if handled by poorly trained non-laboratorians</td>
</tr>
<tr>
<td>Reduced pre-analytic and post-analytic testing errors</td>
<td>Quality management/assurance issues and responsibilities not defined</td>
</tr>
<tr>
<td>Self-contained and user-friendly instruments</td>
<td>Concerns about cost effectiveness</td>
</tr>
<tr>
<td>Small sample volume for a large test menu</td>
<td>Lack of connectivity: difficulty in integrating test results with hospital information system (HIS) or laboratory information system (LIS)</td>
</tr>
<tr>
<td>Shorter patient length of stay</td>
<td>Narrower measuring range for some analytes</td>
</tr>
<tr>
<td>Ability to test many types of samples (capillary, saliva, urine etc)</td>
<td></td>
</tr>
</tbody>
</table>

(Also see appendix II and III)
Appendix I
Characteristics of Whole-Blood analyzers

<table>
<thead>
<tr>
<th>Instrument</th>
<th>Manufacturer</th>
<th>Type</th>
<th>Sample Volume (µL)</th>
<th>Analysis Time (seconds)</th>
<th>Test Analytes (measured)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I-STAT</td>
<td>Abbott Diagnostics, Abbott Park, IL</td>
<td>Handheld</td>
<td>65 or 95</td>
<td>90-140</td>
<td>(p_{O2}, p_{CO2}, pH, Na^+, K^+, Ca^{++}, Cl^-, Hct, urea nitrogen, glucose, lactate, creatinine)</td>
</tr>
<tr>
<td>AVL OMNI</td>
<td>AVL Scientific, Roswell, GA</td>
<td>Transportable</td>
<td>40-161</td>
<td>60-90</td>
<td>(p_{O2}, p_{CO2}, pH, Na^+, K^+, Ca^{++}, Cl^-, Hct, urea nitrogen, glucose, lactate, creatinine)</td>
</tr>
<tr>
<td>AVL OPTI</td>
<td>AVL Scientific, Roswell, GA</td>
<td>Portable</td>
<td>125</td>
<td>&lt;120</td>
<td>(p_{O2}, p_{CO2}, pH, Na^+, K^+, Ca^{++}, Cl^-, Hct, urea nitrogen, glucose, lactate, creatinine)</td>
</tr>
<tr>
<td>Rapid Lab 800 series</td>
<td>Bayer Diagnostics, Norwood, MA</td>
<td>Transportable</td>
<td>140-175</td>
<td>85</td>
<td>(p_{O2}, p_{CO2}, pH, Na^+, K^+, Ca^{++}, Cl^-, Hct, urea nitrogen, glucose, lactate, creatinine)</td>
</tr>
<tr>
<td>IRMA SL (series 2000)</td>
<td>Agilent Technologies, St Paul, MN</td>
<td>Portable</td>
<td>125</td>
<td>90</td>
<td>(p_{O2}, p_{CO2}, pH, Na^+, K^+, Ca^{++}, Cl^-, Hct)</td>
</tr>
<tr>
<td>HemoCue B-Hemoglobin</td>
<td>HemoCue, Mission Viejo, CA</td>
<td>Portable</td>
<td>10</td>
<td>45-60</td>
<td>Hb</td>
</tr>
<tr>
<td>Gem Premier 3000, 3015</td>
<td>Instrumentation Laboratory Lexington, MA</td>
<td>Portable</td>
<td>135</td>
<td>&lt;120</td>
<td>(p_{O2}, p_{CO2}, pH, Na^+, K^+, Ca^{++}, Cl^-, Hct, urea nitrogen, glucose, lactate, creatinine)</td>
</tr>
<tr>
<td>Stat profile pHOX, SO2%, Hct, Hb</td>
<td>Nova Biomedical, Waltham, MA</td>
<td>Transportable</td>
<td>85-190</td>
<td>78-108</td>
<td>(p_{O2}, p_{CO2}, pH, SO2%, Na^+, K^+, Ca^{++}, Mg^{++}, Cl^-, Hct, urea nitrogen, glucose, lactate, creatinine)</td>
</tr>
<tr>
<td>Stat Profile M/M7†</td>
<td>Nova Biomedical</td>
<td>Transportable</td>
<td>385</td>
<td>85</td>
<td>Na^+, K^+, Cl^-, Hct, TCO2, Hct, urea nitrogen, glucose, creatinine</td>
</tr>
<tr>
<td>ABL 700 series 1</td>
<td>Radiometer, Westlake, OH</td>
<td>Transportable</td>
<td>&lt;180</td>
<td>&lt;60</td>
<td>(p_{O2}, p_{CO2}, pH, Na^+, K^+, Ca^{++}, Cl^-, Hct)</td>
</tr>
<tr>
<td>ABL 70 Series</td>
<td>Radiometer</td>
<td>Portable</td>
<td>&lt;180</td>
<td>&lt;60</td>
<td>(p_{O2}, p_{CO2}, pH, Na^+, K^+, Ca^{++}, Cl^-, Hct)</td>
</tr>
<tr>
<td>YSI 2300 Stat Plus</td>
<td>Yellow Springs Instrument, Yellow Springs, OH</td>
<td>Portable</td>
<td>25</td>
<td>45</td>
<td>Glucose, lactate</td>
</tr>
</tbody>
</table>

\(p_{O2}\): blood oxygen tension; \(p_{CO2}\): blood carbon dioxide tension; SO2%: oxygen saturation; TCO2: total carbon dioxide in blood; Na+: sodium; K+: potassium; Ca++: ionized calcium; Mg++: magnesium; Cl-: chloride; Hct: hematocrit; Hb: hemoglobin.

Table 4
Pre-analytic and Post-analytic errors in laboratory testing

<table>
<thead>
<tr>
<th>Pre-analytic errors</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Mishandling and/or mislabeling of patient specimen</td>
<td></td>
</tr>
<tr>
<td>Contamination of specimen</td>
<td></td>
</tr>
<tr>
<td>Degradation of specimen due to delays in specimen</td>
<td></td>
</tr>
<tr>
<td>processing/testing</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Post-analytic errors</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Misreporting patient test results</td>
<td></td>
</tr>
<tr>
<td>Recording wrong patient test results</td>
<td></td>
</tr>
<tr>
<td>Lost data</td>
<td></td>
</tr>
<tr>
<td>Delayed reporting of critical results</td>
<td></td>
</tr>
</tbody>
</table>

Another benefit of POCT is small sample volume. Critically ill patients in ER or ICU settings often require repeated/sequential blood tests to closely monitor trends and may lead to substantial blood loss during hospital stay. POCT devices utilise as little as 40 µL of blood to deliver a battery of test results and thus help minimizing blood sample volumes.
Appendix II

Examples of improved clinical outcomes from using point of care testing

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Faster decision making</td>
<td>Chest pain, drug overdose</td>
</tr>
<tr>
<td>Starting treatment earlier</td>
<td>Drug overdose</td>
</tr>
<tr>
<td>Improved adherence to treatment</td>
<td>Diabetes</td>
</tr>
<tr>
<td>Reduced incidence of complications</td>
<td>Diabetes</td>
</tr>
<tr>
<td>Quicker optimization of treatment</td>
<td>Anticoagulation</td>
</tr>
<tr>
<td>Reduced reoperation or readmission rate</td>
<td>Parathyroidectomy</td>
</tr>
<tr>
<td>Patient satisfaction</td>
<td>Fewer journeys, ownership of disease</td>
</tr>
</tbody>
</table>

Appendix III

Examples of economic outcomes from use of point of care testing

<table>
<thead>
<tr>
<th>Reduced number of clinic visits</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Reduced length of hospital stay</td>
<td></td>
</tr>
<tr>
<td>Earlier discharge from hospital</td>
<td></td>
</tr>
<tr>
<td>Fewer unnecessary hospital admissions</td>
<td></td>
</tr>
<tr>
<td>Better optimized drug treatment</td>
<td></td>
</tr>
<tr>
<td>Less inappropriate use of drugs</td>
<td></td>
</tr>
<tr>
<td>Reduced use of blood products</td>
<td></td>
</tr>
<tr>
<td>Reduced use of staff, equipment, and estate</td>
<td></td>
</tr>
<tr>
<td>Improved quality of life</td>
<td></td>
</tr>
</tbody>
</table>

Table 5

Cost Factors in Point-of-Care Testing

| Supplies (eg, reagents, disposable cartridges, test strips) and equipment |                             |
| Training and retraining of instrument operators |                             |
| Maintenance of instruments, including replacing defective instruments |                             |
| Additional labor on the part of non-laboratorians (eg, nurses) to run patient tests |                             |
| New software that enables patient results to be entered into hospital/laboratory information systems (HIS/LIS) |                             |
| Troubleshooting instruments |                             |
| Consultation services for instrumentation problems |                             |
| Performing comparison studies of new instruments and methodologies with existing instruments |                             |
| Accreditation and proficiency testing fees |                             |
| Duplication, repeated tests, verification, and validation |                             |

There have been speculations regarding vulnerability of test results to quality errors in POCT, especially as it is performed beside by healthcare providers rather than trained technical laboratory staff. Most of these errors occurred in pre-analytic and analytic phases and were related to unwillingness or inability of operator to perform basic procedures for device priming or maintenance. Its negative impact would be as swift as the changes in management strategies based on test results and may override clinical assessment. Such limitations may be reduced with rigorous training of operators for internal quality control and provision for external quality assurance by trained laboratory staff on periodic basis and prospective availability user friendly devices.

There has been considerable debate over cost-effectiveness of POCT (Table 5). While prima facie it appears that procurement of new devices, training of operators, maintenance of quality control and a tendency for overzealous and unnecessary repeated testing with the use of POCT would increase the treatment costs, there are arguments that shorter diagnostic turn-around-time and reduced length of stay in hospital and emergency room may actually cut-down overall treatment costs. It seems likely that use of POCT will help minimizing inappropriate use of drugs or empiric therapeutic trials and may even avoid admissions because of diagnostic uncertainties.

CONCLUSION

Point-of-care testing is an attractive concept in critical care settings with prospective benefits of on-site diagnostic testing and trend monitoring of patient owing
to its the shorter therapeutic turn-around-time, reduced analytic errors and portability. While POCT may not completely replace centralized laboratory testing but may transform the clinical practice pattern and process of care of the critical carers. More comprehensive, rigorous and critical evaluations are required, to decisively state its role in contemporary practice of critical care medicine.

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Seizure exacerbation by antiepileptic drugs: a less recognized entity

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ABSTRACT

Antiepileptic drugs as per general notion should control seizures but in some instance they can produce or exacerbate seizures. The diagnosis is not always easy especially in patients with disabilities and epilepsy syndromes. Thus, it is essential for physicians dealing with seizure patients to be aware of this entity. It is prudent to avoid certain drugs notorious for consistently aggravating particular seizure types. In all cases of seizure aggravation, high degree of suspicion should be there for seizure aggravation by antiepileptic drugs especially in patients with risk factors like young age, epilepsy syndrome and polypharmacy.

Keywords: Antiepileptic drugs, Seizure exacerbation

Since their advent, antiepileptic drugs (AED) have been proven as a boon for millions of patients with seizure disorder, not only have these drugs helped in controlling seizures but improved quality of life of these patients. In some patients AEDs can cause opposite effects, not only can there be an increased frequency, strength and duration of established seizure type but also new types of attacks, and at worst status epilepticus can appear.¹,²,³ Both old and new AEDs can cause seizure aggravation.

Exact incidence of seizure exacerbation by AED is not known, most of the knowledge about it is from case series and case reports.

Often it is difficult to establish a causal relationship between seizure exacerbation and drug introduction so standard criteria for reporting these events should be there. According to Perruca et al one or more of following statements must be applicable to say AED induced seizure exacerbation: (1) An evident increase in seizure frequency after administration of the AED which is reversible on discontinuation/reduction of dose (2) A consistent adverse effect of specific AED in a specific seizure type or syndrome (3) Other known factor (eg. EEG features) predictive of AED induced seizure deterioration (4) Emergence of new seizure type (not known previously in that patient) showing a clear cut temporal association with the change in AED.⁴

Seizure exacerbation induced by AED can be caused by various mechanisms but generally four mechanisms are considered as most important: (i) Drug toxicity; (ii) Drug induced encephalopathy; (iii) Inappropriate choice of AED; and (iv) True paradoxical effect.⁵ In some instances clear categorization is not always feasible.

DRUG TOXICITY

It is usually seen in children with malignant epilepsy syndromes already on multiple AED. It can be due to paradoxical increase in drug levels or toxic doses. There can be nonspecific manifestations like sedation, irritability or sleep disturbances or/and increase in seizure frequency at toxic doses. Sedative AEDs like benzodiazepines and phenobarbitone are more likely to cause such effects. In children with malignant epilepsy syndromes, cognitive deficit along with frequent convulsive seizures and post-ictal states make AED-induced sedation difficult to diagnose so clinician must always be on alert for such adverse effects. It is essential to exclude drug toxicity in a heavily sedated patient with seizure aggravation.⁶ At toxic concentrations, phenytoin and carbamazepine can have true proconvulsant effect. Myoclonic status can be
induced by lamotrigine and tiagabine can induce non-convulsive status epilepticus in focal epilepsies and absence status in idiopathic generalized epilepsies (IGE) at high doses.\textsuperscript{3,7}

**DRUG INDUCED ENCEPHALOPATHY**

In AED-induced encephalopathy, cerebral function impairment occurs in the presence of nontoxic levels of the AED. The most common example is valproate. The encephalopathy can happen with valproate alone or during comedication with phenobarbitone, benzodiazepine, or topiramate. It can occur with or without altered hepatic function. Hyperammonemia with or without liver dysfunction has also been reported.\textsuperscript{8,9} Young children, particularly those under 2 years of age, are at high risk for this condition. Valproate induced hepatopathy/encephalopathy can develop within a week of introduction to several months later. In few patients, hepatopathy/encephalopathy may appear after an increase in the dose in patients established on valproate.\textsuperscript{10} Carbamazepine and vigabatrin have also been implicated in AED induced encephalopathy.\textsuperscript{9}

**INAPPROPRIATE CHOICE OF AED**

There is sufficient evidence that in certain seizure types or epilepsy syndromes certain AEDs can cause exacerbation of seizures i.e. these AEDs are inappropriate choice for them. Most widely recognised example is carbamazepine. In IGE and myoclonic epilepsies, carbamazepine can aggravate existing seizure types or induce new seizure types (absence, atonic, myoclonic, generalized tonic clonic seizures). Severe aggravation of seizures in IGE may result in absence or myoclonic status epilepticus, often with atypical clinical and EEG features.\textsuperscript{11,12} Oxcarbazepine has similar effects. Other examples include lamotrigine in severe myoclonic epilepsy in infancy, vigabatrin in myoclonic and partial epilepsies and gabapentin in absence and myoclonic seizures.\textsuperscript{13}

**TRUE PARADOXICAL EFFECT**

In this an AED appears to exacerbate a type of seizure against which it is usually effective, or when it leads to the onset of new types of seizures. It happens unpredictably at nontoxic drug levels, usually shortly after introduction of the AED. The distinction between paradoxical reaction and inappropriate AED choice cannot always be clearly established. In some cases, the type of seizure for which treatment has been chosen may have been incorrectly diagnosed (e.g., absence seizures treated with CBZ due to misdiagnosis as CPS).\textsuperscript{3,9} Carbamazepine is the AED of choice for partial epilepsies; yet in some partial epilepsies (Frontal lobe epilepsy, benign childhood epilepsy with Centro temporal spikes, Landau-Kleffner syndrome, benign epilepsy of childhood with occipital paroxysms, Angelman syndrome), its use is associated with the appearance of new seizure types like negative myoclonus and atypical absences.\textsuperscript{1} In Lennox-Gastaut syndrome, sometimes intravenous benzodiazepines can precipitate tonic status epilepticus.\textsuperscript{13}

**MECHANISM**

AEDs with single mode of action and narrower spectrum are more likely to cause seizure exacerbation than AEDs with multiple mechanisms of action. Certain patient groups like young children with high seizure load, epileptic encephalopathy, multiple seizure types (seizure syndromes), polytherapy are at higher risk although some benign syndromes can be affected.\textsuperscript{13} Cellular mechanisms involved in the paradoxical reaction of AEDs are different for GABAergic (Phenobarbitone, Barbiturates, Valproate, Gabapentin, Vigabatrine) and sodium channel blockers (Phenytoin, Carbamazepine, Oxcarbamazepine, Lamotrigine). In animal models, GABAergic drugs increase spike-wave discharges and clinical seizures probably related to GABA-induced hyperpolarization of thalamic neurons, enhancing oscillatory thalamocortical activity. Sodium channel blockers increase hypersynchronization of neuronal discharges in a thalamocortical loop because of enhanced membrane stabilization resulting in facilitation of generalized epileptogenesis.\textsuperscript{11} Pharmacogenomics is another interesting aspect which might explain why some children are more vulnerable.\textsuperscript{4}

**HOW TO MINIMIZE DRUG INDUCED SEIZURE EXACERBATION**

Most important point in prevention of these events is to make a correct diagnosis of seizure type. Many a times generalized tonic clonic seizures may present with lateralization i.e. versive seizures, these can be easily confused with focal onset seizures and mistakenly drugs suited for focal seizures e.g. carbamazepine is started and patient might develop absence or myoclonic seizures as a side effect. Likewise unilateral myoclonic jerks can be misinterpreted. Sometimes EEG changes can also be misleading; an EEG can be normal or can show lateralised discharges in generalised seizures or generalised discharges in benign Rolandoic epilepsy.\textsuperscript{13} A clinician
dealing in management of epilepsy should be well aware of seizure syndromes so that correct AED can be chosen according to the correct diagnosis of the seizure type and of the epilepsy type. Certain drugs should be avoided in specific epilepsy syndromes (Table 1). Drugs with several mechanisms of action are less likely to produce aggravation of seizures than narrower spectrum drugs. However exceptions can be there so possibility of aggravation should be considered in every patient, whenever an AED is prescribed.

**MANAGEMENT OF SUSPECTED SEIZURE AGGRAVATION BY AED**

Whenever one comes across a case of increase in seizures after introduction of a new AED or otherwise, one should rule out other potential causes of seizure exacerbation in patients with epilepsy, for example spontaneous increase in seizure tendency, progression of the epileptogenic lesion, intercurrent disease, seizure triggers such as stress, lack of sleep, etc., switching to a drug with poorer seizure-reducing effect, AED withdrawal, conditions giving altered efficacy of anticonvulsants (e.g. pregnancy, renal failure), drugs which may enhance the seizure tendency, including anticonvulsants etc. Unexpected increase in seizures especially in adolescence should raise the suspicion of drug default or pseudoseizures. Certain drugs can cause metabolic problems for example phenytoin can induce severe hyponatremia and valproate liver dysfunction and hyperammonemia. Appropriate investigations including video EEG recording, drug levels, serum electrolytes, liver function tests and repeat neuroimaging (especially in cases with expected deterioration e.g. tumour formation in tuberous sclerosis) should be done. In young children with idiopathic epilepsy, inborn errors of metabolism should be ruled out as they can be progressive and can worsen with certain drugs e.g. valproate. The incriminating drug should be withdrawn completely if newly introduced; replacement drug should be started along with it. If patient was on a drug for a long time and was partially controlled then withdrawal should be gradual. If cause of seizure aggravation was drug toxicity then drug dose should be reduced. Rechallenge with the suspected drug is usually not feasible as it will be unethical.

**CONCLUSION**

Seizure aggravation with AED should be considered in every patient with uncontrolled seizures. Some factors like young age, seizure syndromes, polypharmacy etc. make the patients particularly vulnerable for seizure aggravation by AED. Increased awareness of seizure syndromes and possible drug side effects is required to avoid such instances.

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Managing epilepsy in pregnancy

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ABSTRACT

Epilepsy is the most common neurological disorder encountered in pregnancy. Physiological changes of pregnancy make the management of this medical disorder very challenging. Antiepileptic drug with good seizure control and minimal adverse effect is ideally started in pre-conception period. Careful and critical monitoring during antenatal, intranatal and postnatal period results in good maternal and fetal outcome.

Keywords: Childhood, Seizures, Antiepileptic drugs

Epilepsy is repeated (at least 2) spontaneous, unprovoked seizure of any type. Seizures (convulsions) are episodes of disturbed brain function that cause changes in attention or behavior. Epilepsy can be caused by various known or idiopathic factors which directly or indirectly affect central nervous system. The most characteristic symptoms of epilepsy are seizures, which vary greatly in severity from petit mal, absence seizures, to grand mal, generalized violent tonic-clonic convulsions.

Epilepsy is suspected mainly based on clinical history. Electroencephalography and/or brain imaging should be done as supplementation to your suspected diagnosis, and neuroimaging should be done on any adult patient complaining of new-onset seizures to rule out structural causes.

With improvement in diagnosis and treatment of epilepsy increasing number of women with epilepsy (WWE) are becoming pregnant. In India there are about 2.73 million WWE and 52% of them are in the reproductive (15-49 years) age group. As compared to men, WWE experience more psychosocial problems like marital issues, social stigma and burden on society. We have to continue antiepileptic drug (AED) during pregnancy that makes management of epilepsy very challenging. As risk benefit assessment has to take into account the risk of seizures to mother and fetus also increased risk of major congenital malformation (MCM) and adverse effects on cognitive development after prenatal exposure.

Effect of pregnancy on epilepsy: In about two third of patients seizure activity may decrease or remain unchanged, during pregnancy. Also women who remain seizure free 9 months prior to conception are probably associated with a high likelihood (84%–92%) of remaining seizure free during pregnancy. Women with focal epilepsy or on polytherapy are at increased risk of seizures during pregnancy. Less than 1% of patients are affected by status epilepticus.

Physiological changes of pregnancy, has a direct or indirect effect on seizure activity. Altered AED level may occur due to decreased serum albumin levels, increased maternal blood volume, increased renal blood flow, increased hepatic metabolism as a result of increased hepatic blood flow and increased CYP 450 activity. Decreased gastrointestinal motility and nausea and vomiting which frequently occur in early pregnancy and labor leads to decreased or altered medication intake. Also sleep deprivation, stress and anxiety lowers the threshold for the seizures activity.

Pregnancy outcome

WWE are often considered at high risk in pregnancy, although most of these pregnancy proceed without any complication. It is still unclear whether there is an increased risk of complications in pregnancy or not. Some studies have reported an increased risk of complications like pre eclampsia, gestational hypertension, gestational diabetes, preterm birth and cesarean section in patients with epilepsy. But other studies have not found any increased risk of complications in WWE. American
academy of neurology (AAN) in 2009 has following recommendations for pregnancy outcome in WWE:7

- There is probably no substantially increased risk (greater than two times expected) of late pregnancy bleeding in WWE taking AEDs (Level B).
- There is probably no moderately increased risk (greater than 1.5 times expected) of premature contractions or premature labor and delivery for WWE taking AEDs (Level B).
- There is possibly a substantially increased risk of premature contractions and premature labor and delivery during pregnancy for WWE who smoke (Level C).

Few studies have explored complications in WWE during labor and delivery. Per say epilepsy is not an indication for induction of labor but induction of labor is more frequent in WWE. WWE are not at increased risk of instrumental deliveries.10, 16 Caesarean delivery frequency varies between cohorts of WWE, both increased (17.3% vs. 11.55%, p = 0.008) and no increase in caesarean delivery risk has been reported.10, 11 Multiple factors like increased risk of intrauterine growth restriction, hypertensive disorders of pregnancy and seizures in pregnancy can contribute to higher incidence of cesarean section in WWE. AAN recommends there is possibly a moderately increased risk (up to 1.5 times expected) of cesarean delivery for WWE taking AEDs (Level C).

**Effect of epilepsy on fetus:**
Women who are taking AEDs are twice likely to have intrauterine growth restriction in fetus. They are also at higher risk of low birth weight, small length and head circumference and low Apgar score.8, 17 Microcephaly and low birth weight occurred more significantly in children exposed to carbamazepine, valproate and poly therapy.18, 19 Causative factors could be the epilepsy, exposure to AEDs, seizures, genetic, any underlying condition and / or environmental factors. Various combinations of MCM, minor anomalies and aforementioned complication during pregnancy are known as fetal anticonvulsant syndrome.

Worldwide numerous studies have reported that prenatal exposure to AEDs increases risk of MCM from the background risk of 1-2% to 4-9%.1-20 Multiple factors cause MCM in WWE. These are genetic predisposition, direct or indirect (falls or injuries) effects of seizures, and teratogenic effects of AEDs. Seizures can cause reduced placental circulation and secondary ischemia in fetus. Increased oxidative stress after restoration of circulation could exert teratogenic effect.1 AEDs can cause MCM by various mechanisms like folate deficiency, increased oxidative stress due to AEDs metabolism, alteration in the homeobox genes, retinoic acid signaling pathways, histone deacetylators and polymorphisms involving AED transporters.21, 22 Various MCM caused by AEDs is shown in Table 1.

Risk of MCM increases, dose dependently with all most commonly used AEDs and is shown in Table 2.24 In utero exposure to AEDs is also associated with increased risk of impaired cognitive function at 3 years of age. As compared to other AEDs, in utero exposure to valproate is associated with poorer cognitive outcome.5 Increased risk of neonatal and perinatal mortality has been reported in previous studies, but a recent meta-analysis has not found any such difference in still birth and perinatal mortality rates.3, 25

No sufficient evidence to support or refute risk of hemorrhagic complication in newborn of WWE and any benefit by prenatal Vitamin K supplementation for

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Cases(n)</th>
<th>NTD</th>
<th>Facial cleft</th>
<th>Cardiac</th>
<th>Hypospadias</th>
<th>Skeletal</th>
<th>Others</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carbamazepine</td>
<td>900</td>
<td>2(0.2)</td>
<td>4(0.4)</td>
<td>6(0.7)</td>
<td>2(0.2)</td>
<td>3(0.3)</td>
<td>1(0.1)</td>
</tr>
<tr>
<td>Valproic acid</td>
<td>715</td>
<td>7(1)</td>
<td>11(1.5)</td>
<td>5(0.7)</td>
<td>9(1.3)</td>
<td>2(0.3)</td>
<td>2(0.3)</td>
</tr>
<tr>
<td>Lamotrigine</td>
<td>647</td>
<td>1(0.2)</td>
<td>1(0.2)</td>
<td>4(0.6)</td>
<td>6(0.9)</td>
<td>2(0.3)</td>
<td>4(0.6)</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>82</td>
<td>0</td>
<td>1(1.2)</td>
<td>1(1.2)</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

Adapted from Morrow et al (23)
reducing hemorrhagic complication of newborn. But as a routine intramuscular vitamin K is administered to neonate at birth.

**Treatment of epilepsy**

Medical management is always first line of treatment. However, initial drug of choice depends on severity and type of seizure but always monotherapy is preferred. Polytherapy is reserved for patients with persistent seizure or unacceptable side effects. Epilepsy surgery is for resistant cases. After initiating the therapy, further dose adjustment depends on the seizure frequency and side effects. AEDs levels are routinely not done in pregnancy. If at all they are measured, free drug levels should be measured, because of changes in protein binding during pregnancy.

AEDs like phenytoin, carbamazepine, phenobarbital and valproate are category D drugs. New AEDs like gabapentin, lamotrigine, topiramate and levetiracetam are categorized as category C drugs but it’s too early to draw any conclusion about safety of these drugs. Side effects of these AEDs in mother are categorized into common, rare and unique. Common side effects are dose related, nonspecific and affect central nervous system. Rare side effects are unpredictable and not dose dependent like skin rashes, leukopenia, thrombocytopenia, and hepatic problems. Unique adverse effects are specific to drugs. Phenytoin can cause gingival edema and bleeding and valproate can cause alopecia and weight gain in WWE. Worsening of seizure is more commonly seen in women on lamotrigine as compared to other monotherapy (carbamazepine, valproate and phenobarbitone). Also higher proportion of pregnancies on lamotrigine requires dose increments or addition of another AED.

**Antenatal care:**

In addition to routine obstetric care following care is to be given to WWE:

- Adequate rest and sleep
- Continuation and optimization of pre-pregnancy AED, including maintaining free drug levels within the woman’s range of optimal therapeutic and symptomatic efficacy
- Avoidance of hazardous activities such as driving, swimming
- Prenatal testing for neural tube defects and other structural anomalies
- Detail anatomy scan at 16-20 weeks and fetal echocardiography at 22-24 weeks
- Folic acid supplementation (4mg/day)
- Antepartum fetal surveillance is done for standard obstetrics indication only

**Intrapartum care**

Care during labor and delivery does not differ significantly in WWE. But intrapartum period is crucial as these women are at risk of suffering from seizure because of potential disruption of medication schedule, sleep deprivation, pain and/or co medication. Oxygen, equipment, and expertise to protect the maternal airway, and intravenous benzodiazepines (lorazepam) should be readily available in case of any tonic clonic seizure. During

<table>
<thead>
<tr>
<th>Antiepileptic drug(mg per day)</th>
<th>Cases</th>
<th>CM up to Birth to 2 months</th>
<th>CM up to 1 year</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Carbamazepine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;400</td>
<td>148</td>
<td>2 (1·3%, 0·16-4·80)</td>
<td>5 (3·4%, 1·11-7·71)</td>
</tr>
<tr>
<td>≤400 to &lt;1000</td>
<td>1047</td>
<td>34 (3·2%, 2·26-4·51)</td>
<td>56 (5·3%, 4·07-6·89)</td>
</tr>
<tr>
<td>≥1000</td>
<td>207</td>
<td>16 (7·7%, 4·48-12·25)</td>
<td>18 (8·7%, 5·24-13·39)</td>
</tr>
<tr>
<td><strong>Lamotrigine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;300</td>
<td>836</td>
<td>14 (1·7%, 0·92-2·79)</td>
<td>17 (2·0%, 1·19-3·24)</td>
</tr>
<tr>
<td>≥300</td>
<td>444</td>
<td>16 (3·6%, 2·07-5·79)</td>
<td>20 (4·5%, 2·77-6·87)</td>
</tr>
<tr>
<td><strong>Phenobarbital</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;150</td>
<td>166</td>
<td>7 (4·2%, 1·71-8·50)</td>
<td>9 (5·4%, 2·51-10·04)</td>
</tr>
<tr>
<td>≥150</td>
<td>51</td>
<td>7 (13·7%, 5·70-26·26)</td>
<td>7 (13·7%, 5·70-26·26)</td>
</tr>
<tr>
<td><strong>Valproate</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;700</td>
<td>431</td>
<td>18 (4·2%, 2·49-6·52)</td>
<td>24 (5·6%, 3·60-8·17)</td>
</tr>
</tbody>
</table>
labor, seizures or pre-seizure aura may require intravenous administration of AEDs. AEDs which are available as parenteral administration are levetiracetam, fosphenytoin, and phenytoin. Carbamazepine has no parenteral preparation so alternative AED should be available for women on carbamazepine.29

The National Institute for Health and Clinical Excellence in the United Kingdom and AAN recommend vaginal delivery in WWE, with the exception of women with frequent seizures.7,30 Induction of labor is performed only for medical and obstetrics indication including worsening of seizures. Labor pain management is to be provided and there is no contraindication to commonly used pain management including epidural anesthesia.29

**Postpartum care**

After delivery, as the physiologic changes of pregnancy start normalizing. So, return to pre-pregnancy AED dose is required to prevent drug toxicity.31 Adequate rest and support is particular important to prevent seizure precipitation due to stress and sleep disturbances. Concentration of AEDs in breast milk is significantly low. Also, there is no evidence to determine whether this indirect exposure of newborn has any clinical consequence. As the benefits of breast milk are well established AAN recommends benefits of breast feeding outweigh the risks.32 However, breast feeding mothers are advised to report immediately if there is any acute change in newborn behavior like lethargy.

General precautions are to be taken by WWE while caring their newborn. Like never bathing the child alone, never carrying the child in a carrier attached to the mother’s body, avoiding the use of high changing tables or placing infant at higher than ground level.

**Contraception**

Contraception advice should a part of routine postpartum care. Table 3 describes different contraceptive options available for WWE.

Hepatic microsomal enzyme inducing AEDs are phenytoin, carbamazepine, barbiturates, primidone, topiramate, oxcarbazepine. These drugs cause decrease in estrogen concentration of combined contraceptive methods (CCM) thus compromises their efficacy. Nonenzyme-inducing AEDs (valproate, benzodiazepines, ethosuximide, and levetiracetam) do not show any interactions with the combined oral contraceptive pill, but are relatively less commonly used in pregnancy and post-delivery. Lamotrigine levels of decrease significantly during CCM use and increase significantly during the pill-free interval, so to be cautiously used in WWE on CCM. Depo medroxy progesterone acetate (DMPA) appears to be effective in WWE but then it is to be administered at every 10 weeks rather than 12 weeks. Implants are contraindicated due unacceptably high failure rates. Levonorgestrel intraterine device (LNG IUD) use is safe as it acts locally on uterus. Emergency contraceptive pill can be used in WWE.32,33

**Preconception counselling:**

Pre conception care is very critical with the goal to improve maternal fetal outcome. Aim of counselling:

---

**Table 3**

WHO category of different contraceptive methods in epileptic woman on treatment

<table>
<thead>
<tr>
<th>Drug</th>
<th>COC/P/R</th>
<th>CIC</th>
<th>POP</th>
<th>DMPA/NET-EN</th>
<th>LNG/ETG</th>
<th>Cu IUD IUD</th>
<th>LNG-IUD IUD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatic microsomal inducing AEDs</td>
<td>3</td>
<td>2</td>
<td>3</td>
<td>DMPA 1</td>
<td>NET-EN 2</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Nonenzyme inducing AEDs</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Lamotrigine</td>
<td>3</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

COC/P/R- Low dose combined oral contraceptive pill/ patch/ring , CIC- Combined injectable contraception , POP- Progesterone only pill, DMPA- Depo medroxy progesterone acetate , NET-EN- Norethindrone acetate , Cu IUD -Copper containing intrauterine device, LNG IUD Levonorgestrel intrauterine device
* Assessment of current seizure control including type and time of last seizure

* Current AED regimen

* Risk to woman and fetus during pregnancy

If a woman is seizure free for several years, she can be weaned off her AED over several months. But there is 50% risk of seizure recurrence. Ideally conception should be deferred till seizures are well controlled, preferably seizure 9-12 months before pregnancy. Woman should be put on relatively safer AEDs, monotherapy where possible and avoid valproate. Pre-conception 4mg/day folic acid supplementation should be started at least 1 month before pregnancy. Fetus is two to three fold increased risk of MCM compared to general population and possibly at risk of cognitive impairment. Majority of women can be reassured that there is very low risk of passing epilepsy to their children. For woman with idiopathic generalized epilepsy (IGE) there is 9-12% risk of having an affected child. Risk of developing IGE is 5-20% and >25% if one first degree and two second degree relatives are affected. Most of women have successful pregnancy outcome.

CONCLUSION

Epilepsy is common neurological disorder complicating pregnancy. A team of obstetrician, neurologist and perinatologist should manage these pregnancies thereby optimizing maternal and fetal safety throughout pregnancy.

REFERENCES


Pressure ulcers management and practice guidelines in critical care settings

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ABSTRACT

Pressure ulcers tend to have colossal effect on the general management, clinical outcomes and period of hospital stay of patients admitted in critical care units. Multidisciplinary team approach is required to follow appropriate protocols and guidelines for acquiring the desired results. The critical carers should formulate appropriate strategies and incorporate active participation of paramedical staff including nurses, physiotherapists and dieticians for its successful management. This review article focuses on its prevention, risk assessment, early detection and subsequent effectual treatment by following current evidence based practices to achieve the optimum clinical outcome in existing critical care settings.

Key words: Pressure ulcers; debridement; repositioning techniques; critical care; polyurethane dressing.

INTRODUCTION

One of the most overlooked clinical entities in any intensive care unit (ICU) is pressure ulcers (bed sores), having profound effect on patient’s overall clinical outcome and subsequent hospital stay period. Pressure ulcers require a multidisciplinary approach for its management. The focus of its management is to prevent the development of pressure ulcers at the early stages of its development. The critical carers should formulate strategies and incorporate nursing and other paramedic’s staff including physiotherapists and dieticians for its successful management. Strict implementation of management protocols and dissipation of awareness amongst the caregivers by clinical updates and feedbacks should be the foremost concern.

DEFINITION

Pressure ulcers are soft tissue necrosis of localized area that occurs because of soft tissue compression between bony prominences and external surface for a prolonged period of time.

CLASSIFICATION

There are four stages in the development of full blown pressure ulcers.

Stage 1
There is non-blanchable patch of skin area (appears reddened or bluish/purplish cast in dark coloured people) which may be itchy, warm, painful, spongy or firm when touched. It heals quickly when pressure is relieved.

Stage 2
In this stage, there is a loss of skin (epidermis and/or dermis) with red or purple surrounding area, similar to a blister or abrasion formation.

Stage 3
The skin loss involves all layers or entire thickness of skin, sparing the underlying muscles and bones. At this stage, the wound deepens and appears like a crater.

Stage 4
This stage is most critical. There is severe damage of skin with extensive tissue necrosis. The muscles, tendons and bone are generally affected. It may progress to severe life-threatening infections if not managed timely.

Sites of pressure ulcers development

In ICU set ups, the patients are inevitably confined to beds (non-ambulatory) and hence the pressure prone dependent areas may differ in line with the patient position (Table 1).
Table 1
Pressure prone areas in line with the patient position

<table>
<thead>
<tr>
<th>Patient Position</th>
<th>Pressure prone areas affected</th>
</tr>
</thead>
<tbody>
<tr>
<td>Supine</td>
<td>Occiput, Scapula, Sacrum, Heels, Elbows</td>
</tr>
<tr>
<td>Lateral</td>
<td>Ear lobes, Acromion process, Ribs, Greater trochanter, Medial and lateral condyle, Malleolus</td>
</tr>
<tr>
<td>Prone</td>
<td>Cheek and ear, Acromion process, Breast(women), Genitalia(men), Knees, Toes</td>
</tr>
</tbody>
</table>

PREDISPOSING FACTORS IN THE DEVELOPMENT OF PRESSURE ULCERS\(^2,3\)

The prime risk factor involve in the development of pressure ulcers is the prolong immobilization of the patient position. The critical carer's should ensure that the position of the patient should be periodically changed wherever possible. There are numerous other predisposing factors in critically ill patients leading to pressure ulcers development such as spinal cord injury, extremes of ages, poor nutritional status, persistent infections, faecal/urinary incontinence and heat.\(^2,3\)

GENERAL GUIDELINES FOR PRESSURE ULCER PREVENTION\(^4\)

Pressure ulcers are best managed by preventive measures. Proper implementation of institutional protocols and team management plays a vital role. Nursing care plans formulated after thorough patient assessment and its devotional execution can lead to better clinical outcomes. A three-pronged comprehensive approach is carried out in critical care units to achieve the desired result which includes(i) high risk identification, (ii)preventive measures implementation and (iii) subsequent follow-ups after discharge form ICU.\(^4\)

1. **Risk identification:** Several scales have been design for risk assessment of every admitted patient in ICU. Most popular among them are Braden and Norton scales.\(^5,6\) Many studies have shown these scales to be most sensitive and accurate in approach. In general, every patient must be assessed on admission in ICU, using these specified scales to identify high risk patients.

The common parameters included in the risk identification scales are:

* General physical assessment
* Nutritional status
* Psychological status
* Acute medical conditions affecting skin and peripheral circulation
* Age
* General health status
* Sensory perception
* Conditions contributing to moisture
* External friction and shear forces

Considering these parameters, nursing care plan must be prepared and focused on prevention of pressure ulcers and if, already received with one, its early management followed by complete cure.

2. **Preventive measures:** Current up-to-date knowledge and continuing education of health care professionals, especially nursing staff, is the primary...


Table 2

Repositioning techniques

| Pressure must be released or redistributed on surface area. |
| Repositioning should be alternated (every 1 to 2 hour) with side lying, (right side, on back, left side) or prone, if medical conditions allows. |
| Head-end elevation not more than 30 degrees is advised but is an area of dispute in ICU patients as head-end elevation of 45 degrees is advised to prevent complications leading to ventilator associated pneumonia (VAP). Many studies have carried out in this regard which conclude that positioning should be versatile and according to the patients need and medical condition. |
| Efforts must be taken to make sure that patients are not repositioned on already reddened skin area. |
| While repositioning, the patient must be lifted with the help of sheets and not at all dragged on bed to avoid the shear forces to mark their impact. |
| Avoid the use of cut out rings, synthetic sheep skin pads or doughnut shaped devices and water filled gloves. |
| Proper and regular skin inspection of the heels is important. Heels must not touch the bed surface. Friction must be avoided. They are needed to be completely off the bed surface and in slight flexion. A pillow can be kept under the whole length of both calves to keep heels in air. |

objective for prevention of pressure ulcers, in critical care settings.7

A. Repositioning techniques: Proper repositioning techniques (Table 2) to relieve pressure on pressure points are highly beneficial in patient who cannot move or shift by themselves.2, 4, 8, 9

B. Documentation: It is mandatory to record the frequency, position applied each time, skin assessment and outcome of regime employed.

C. Special devices application: Air and water mattresses and specialised beds such as kinetic beds and oscillatory beds, which can turn the patients 360 degrees, are very helpful. Frequent use of pillows, cushions to protect pressure points can immensely reduce the risk.2, 8

D. Skin care: Skin needs special attention. Do not massage vigorously for prevention of pressure ulcers as it may worsen the situation. Skin may damage with vigorous massage, particularly in high risk patient. Use skin emollients to prevent dryness and hence reducing the risk of damage.

E. Protection from heat: High temperature also contributes to formation of pressure ulcers. So body temperature must be maintained. Repositioning and air current flow between skin and bed surface helps to reduce high temperature and hence decreases the risk of pressure ulcer formation with tissue destruction because of high heat.

F. Protection from moisture: Skin needs protection from moisture as well, as it can lead to skin excoriation over time. Patients in critical units are normally catheterised (urinary catheters) because of their medical conditions and hence need regular check-ups for any leakages. Non-catheterised patients must be checked for incontinence and further managed. Patients for bowel incontinence, as in case of spinal cord injury with complete paralysis may be considered for surgical colostomy and skin barrier products must be used to prevent skin excoriation.

G. Nutrition: Diet should incorporate optimal amounts of proteins, fats and carbohydrates along with vitamins, iron and zinc.10 A Team approach is required here by integrating dieticians in management of patient. All patients admitted in ICU under high risk of pressure ulcer development and compromised nutritional levels, must be provided a minimum of 30-35 kcal/kg of body weight per day with 1.25-1.5gm/kg/day of proteins and 1ml of fluid intake /kcal/day.4

3. Follow-ups after discharge form ICU: Regular follow up of all high risk patients is highly important. Daily assessment of skin and pressure points is recommended along with proper documentation. It helps identifying early development of pressure ulcers and hence can be managed in early stages.

TREATMENT OF PRESSURE ULCERS

Pressure ulcers demand a compassionate team approach regarding its treatment. Multidisciplinary team actions do a lot in early healing and prevention from worsening the case. Pressure ulcer can be managed according to its
stages of development:

1. **First stage**: Here routine assessment plays a vital role. Patients must be managed by increasing the frequency of turning and repositioning. It must not be positioned on already insulted skin area. More padding, pillows and other equipment should be incorporated in care. Specialised transparent polyurethane dressing can be applied on area to prevent further friction with bed linen surface and to avoid skin peeling. It can be applied for a period of 1 to 10 days.

2. **Second stage**: In this stage partial skin loss is present which can be taken care by applying transparent polyurethane dressing’s. These, as described earlier may help in reducing friction and direct contact with bed surface. Wound needs to be kept clean and moist in order to promote growth of healthy tissue. It can be achieved by applying occlusive dressings such as hydrocolloid dressings, hydrogels, bio-membranes and alignates (highly absorbent complex polysaccharide).

3. **Third and Fourth stage**: These stages involve full length tissue loss along with or without muscles, tendons and bones involvement. Treatment follows more comprehensive multidisciplinary management and protocols. As these stages involves necrotic tissue which delays healing process and acts as optimal medium of bacterial growth, debridement is of utmost importance. There are many methods to achieve healthy tissue without necrotic tissue and debris:

   a) **Surgical debridement**: Surgical intervention happens to be the treatment of choice which may be easy, painless and provides opportunity to control bleeding effectively.

   b) **Sharp debridement**: It can be effectively done on bedside with appropriate instruments.

   c) **Chemical debridement**: Use of enzymatic agents used on the necrotic tissue only, can help in its removal but should be used with caution and should not to be applied on healthy viable tissue.

   d) **Mechanical debridement**: This is achieved by applying wet-to-dry dressing on the necrotic tissue.

   e) **Autolytic debridement**: Body’s self defence mechanism activates and tends to remove the necrotic tissue by itself when occlusive dressing is applied.

   f) **Maggot therapy**: This is an alternative therapy which may be helpful in removal of dead tissue by laboratory grown maggots. They feed on necrotic tissue and help in epithelial growth.

   g) **Ultrasound therapy**: Ultrasound waves are sent through a saline bath on dead tissue which eventually separates it from healthy tissue.

   h) **Whirlpool bath therapy**: It may be used in patients whose condition allows sitting in water bath where whirlpools are generated to remove dead tissue.

   i) **Vacuum Assisted Wound Therapy (V.A.C)**: Recently, V.A.C is extensively used to treat acute, chronic and non-healing wounds. It comprises a closed wound system acquired by using a specialised open cell foam dressing, an evacuating tube, a collection canister, vacuum generating device and a transparent film to be applied on wound after the application of foam dressing and tubing (placed parallel to skin and wound). Pressure may be adjusted from -50 to -200 mm of mercury. This therapy helps to achieve healing of wound bed by clearing the secretions, decreasing the bacterial load, stimulating granulation tissue.

   j) **Wound cleansing**: wound cleansing needs to be done with non-cytotoxic fluids like normal saline, ringer lactate, sterile water. Skin cleansers like povidine- iodine, hydrogen per oxide, acetic acid should not be used on the ulcers. Wound needs to be kept moist at all times to facilitate healing process. Wet to dry dressing helps to remove loose necrotic tissue and may be changed every 8 hourly. Dressings that absorb more exudates may be used in these stages. Hydrocolloids and hydrogels are very effective in wound healing.

   k) **Low intensity electric current therapy** for the non-healing wound is under clinical trials and happens to be effective in chronic wounds.

   l) **Growth factors in the form of topical agents** are also under trials and are giving positive results in treating stage 3 and 4 ulcers.

**CONCLUSION**

The common complications associated with chronic, non-healing pressure ulcers are sepsis and osteomyelitis. Hence, the important role of critical carers is its prevention, early diagnosis and treatment to minimise the morbidity and mortality related to these ulcers.
Multidisciplinary team approach with comprehensive management protocols may be the best tools against pressure ulcers and may lead to better clinical outcomes. To manage such patients guidelines based approach is also beneficial for outcome patients. Periodic feedback from attending physicians and empathetic attitude of caregivers are equally important to device and implement current practices for its successful management. Continuing education regarding new approaches being used worldwide and may help in implementing best quality of care to these patients.

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Recent changes in regulation of clinical trials in India: an update for investigators

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The year 2013 has been a year of trials and tribulations for the clinical trials and clinical research in India. It could well prove to be the maker or breaker of the clinical trial industry of the country which, until last year, was poised as one of the most rapidly emerging global player. What has led to the drastic decrease in the number of global clinical trials in India are a set of three successive notifications of the Government of India regarding changes in the regulation of clinical trials under the Schedule Y of the Drugs and Cosmetics Rules, 1945 (D& C Rules).

Here we take a brief look at these new amendments to understand the current regulations for conduct of clinical trials in India. An intense debate has started in the country on these new regulations with a major stakeholder, the pharmaceutical industry citing it as particularly harsh. It is almost sure now that some changes will follow in near future in these newly laid new rules. Nonetheless, it is of utmost importance to take a grasp of the current situation and understand the current laws governing clinical trials.

1. AMENDMENT VIDE G.S.R. 53(E) DATED 30-01-2013.1

Compensation in case of injury or death during clinical trial- Rule 122 DAB of Schedule Y.

It further entails the following provisions:

A) Provisions for payment of compensation

B) Expansion of responsibilities of Investigator, Sponsor and Ethics Committee (EC)

C) Amendment in Informed Consent Document (ICD)

D) Analysis of Serious Adverse Events (SAEs)

As defined in Schedule Y of D& C Rules, 1945, Serious Adverse Events (SAE) is an untoward medical occurrence during clinical trial that is associated with death, in-patient hospitalization, prolongation of hospitalization, persistent or significant disability or incapacity, a congenital anomaly or birth defect or is otherwise life threatening.2

A) Provisions for payment of compensation.1

In case of any AE (adverse event), subjects need to be provided free medical management as long as required and the expenses have to be borne by the sponsor of the study. The sponsor or its representative needs to compensate subjects financially for any injury or death occurring in clinical trial due to following reasons:

a. adverse effect of investigational product(s),

b. violation of the approved protocol, scientific misconduct or negligence by the sponsor or his representative or by investigator,

c. failure of investigational product to provide intended therapeutic effect,

d. use of placebo in a placebo controlled trial,

e. adverse effects due to concomitant medications,

f. injury to a child in-utero due to participation of parents,

g. any clinical trial procedures involved in the study.1

Recently, the special committee set by the DCGI office has come up with the final formula for calculation of compensation in case of SAE of death during a clinical trial. According to this formula the
compensation for a SAE of death may range from somewhere around rupees 4 lac to 70 lacs. This new formula for compensation will be very useful for the Ethics Committee to recommend the amount of compensation to be paid by the sponsor.³

B) Expansion of responsibilities of Investigator, Sponsor and Ethics Committee (EC)  

For all serious and unexpected adverse events the time required for reporting by principal investigator is now within 24 hours of occurrence of event. The PI has to report to the Licensing Authority ie DCGI, the Sponsor and the EC. The detailed report of SAEs, after due analysis, should be forwarded by the investigator and sponsor to Chairman of the EC, Licensing Authority and the Head of the Institution within ten calendar days of occurrence of the SAEs. The report of SAEs of death, in addition, also needs to be forwarded to the Chairman of Expert Committee appointed by Licensing Authority (DCGI). Similarly the sponsor of the clinical trial is now supposed to report a SAE of death to the Chairman of the EC, Chairman of Expert committee with a copy to Licensing Authority (DCGI) and the Head of the Institution within ten calendar days of occurrence of the SAEs. The Ethics Committee, on its part, is also expected to report SAE of death after due analysis and its opinion on amount of compensation to the Expert Committee with a copy to the DCGI within twenty one days of the event.

C) Changes in the Informed Consent Document (ICD).¹

As per Appendix V of the Schedule Y, now the Informed Consent Documents should clearly state that the subject is entitled to free medical management as long as required in case of injury, and financial compensation in case of clinical trial related injury or death. The investigator will have to clearly inform the subject about his right to claim compensation in case of trial related injury or death, and to contact the sponsor / representative directly for any claim related queries. The contact details of sponsor or it’s representative should be provided in the ICD. The ICD now needs to contain details about the subject like qualification, occupation, annual income, address, and contact details of the nominee and his/her relation with the subject in order to aid the calculation of compensation amount. A copy of ICD has to be provided to subject and same should be mentioned in the ICD document. The latest requirement is to get an audio-visual recording of the informed consent by the PI and to keep it for documentation to the Licensing authority, if and when desired.⁴

D) Analysis of Serious Adverse Events (SAEs).¹

The new amendments have added a new appendix ie, Appendix XII under the rule 122 DAB. Appendix XII elaborates the procedure for examination of SAE. The Licensing Authority (DCGI) is now the final authority for determination of causality of SAE of death as also quantum of compensation to be awarded. It will do so after due recommendations from the specially formulated Expert Committee. Now onwards, the sponsor is bound to pay compensation due to injury or death related to clinical trial within thirty days of order from the Licensing Authority, failing which necessary action may be initiated against it, as per rules.

II. Amendment vide G.S.R. 63 (E) dated 01-02-2013.⁵

Permission to Conduct Clinical Trials : Rule 122 DAC of Schedule Y. ⁵

Rule 122 DAC lays down the rules about the compliance to regulatory and ethical guidelines for data submitted for clinical trials, and actions taken in case of noncompliance. All clinical trials should be conducted in compliance with the approved protocols, requirements of Schedule Y, Good Clinical Practice Guidelines for conduct of clinical trials in India and other applicable regulations. The DCGI may inspect the clinical trial sites and the sponsors and investigators or others employees of the sponsors to verify the compliance to the rules. The DCGI may reject or cancel studies and debar investigators and sponsors and their employees for conduct of any clinical trials in future. The above rule also mandates registration of clinical trials in the Clinical Trial Registry Of India (CTRI).³

III. Amendment vide G.S.R. 72(E) dated 08-02-2013.⁶

Registration of Ethics Committees: Rule 122DD of Schedule Y.⁶

This new notification makes it binding for all Ethics Committees, whether Institutional or otherwise to be registered in the office of the DCGI. Once an Ethics Committee is registered it will remain operational for three years. The EC is also supposed to analyse and report the SAE occurring during the clinical trials to the concerned authorities, as per the newly added Appendix XII of the
Schedule of the D& C Rules, 1945. Requisite guidelines have been made available on the CDSCO website regarding documents required for registration of ECs. 
Till date about six hundred ECs have been registered with the DCGI.

Tribulations posed by the new rules on conduct of clinical trials:

The industry sponsored clinical trials

These new regulations created intense debates especially regarding the new compensation rules, including the provision of payment of compensation by the sponsor in case of
- failure of investigational product to provide intended therapeutic effect,
- use of placebo in a placebo controlled trial intense

The very short time period for reporting of SAEs, by the PI, ie within 24 hrs of occurrence, rather than within 24 hours of their being reported to the PI is also being looked upon as a major deterrent in compliance.

Investigator -initiated clinical trials and academic research

The fate of academic research also hangs in balance now, as to how can the investigators who initiate trials be capable of paying the huge compensation occurring due to the new clauses in the Rule 122DAB , especially the two points noted above.

A Ray of Hope for the future

Regarding the concerns of the various stakeholders after the notification of these three new rules, a high level committee headed by Prof. Ranjit Roy Chaudhary was constituted by the Ministry of Health and Family Welfare for preparing policy and guidelines for clinical trials and new drugs. The committee has submitted its reports and is expected that it will address the major concerns of various stakeholders to enable the derailed clinical trial express come back on track. Besides many other suggested actions, some of the most important actions to be taken soon in response to the above mentioned committee’s recommendations are as follows:

- Accreditation of Ethics Committee, Investigators and the clinical trial sites.
- Procedure for review of applications of clinical trials and new drugs.
- Computerized database and selection of experts
- Informed Consent process
- Compensation in case of clinical trial related injury or death.

It is hoped that the timely enactment of the said laws and their amendments, as proposed by the Prof. Roy Chaudhary Committee will ensure the right atmosphere for the safe and pragmatic conduct of clinical trials in India in the very near future and that it will bring back the trust of the general public back in this vital aspect of medical care.

REFERENCES

Large cervical leiomyoma posing diagnostic dilemma

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ABSTRACT
Leiomyomas are the commonest uterine and pelvic tumours. The usual anatomical location is the body of the uterus. Cervical leiomyomas are uncommon and rarely present as a huge abdominal mass. We report a case of a 46-year old female who presented with abdominal distension for one year. Abdominal examination revealed a huge mass of 32-34 week size pregnant uterus filling the whole abdomen with restricted mobility, non tender and solid consistency with a possible diagnosis of retroperitoneal mass or ovarian neoplasm. On ultrasound and MRI there was a strong suspicion for retroperitoneal mass. On exploratory laparotomy, there was a large cervical fibroid visualized on laparotomy that was successfully excised. Histopathological examination confirmed the diagnosis of leiomyoma. The patient had an uneventful postoperative recovery.

Keywords: Cervical leiomyoma, Abdominal mass

INTRODUCTION
Leiomyoma is the commonest of all pelvic tumors, being responsible for about 1/3rd of hospital admissions to gynaecology department. Most leiomyomas arise in the body of the uterus. Cervix is involved in 1 to 2 percent of cases and usually it involves supravaginal portion, in the wall of cervix.¹ A cervical leiomyoma is commonly either interstitial or subserous. Usually it presents as an isolated entity. Cervical leiomyoma are known for varied presentations. These tumors can present with frequency or retention of urine, urinary frequency, constipation, sensation of mass coming down, foul smelling discharge per vaginam, menstrual abnormalities, dyspareunia, and sometimes post coital bleeding.² It rarely becomes sub mucous and polypoidal.³ Incarcerated procidentia is also one of the rare presentations of cervical fibroid.⁴ They can change the shape of the cervix or may lengthen it. If cervical fibroid get bigger, it may even push the uterus upwards. In some cases, cervical fibroid may grow rapidly and can obstruct the cervix. Large cervical fibroids are difficult to handle and need an expert hand to operate these cases.⁵

However, to the best of our knowledge, there has been less number of case reports of such a large cervical fibroid in the literature. This case is reported for its preoperative diagnostic dilemma.

CASE REPORT
A 46 years old patient came with the history of gradual abdominal distension for past one year. She had associated symptoms of bloating sensation, dyspepsia and off and on discharge per vaginam. She was married and had 2 living issues. Her general physical examination was normal and abdominal examination revealed an abdomino-pelvic mass corresponding to 32-34 weeks gravid uterus with restricted mobility. The mass was non tender, firm to hard in consistency. Local examination of perineum was normal. Per speculum examination revealed high cervix and cervix looked healthy in appearance. Per vaginam examination confirmed the abdominal pelvic

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Fig 1. Maximum dimensions of the mass
mass arising from pelvis; uterus couldn’t be felt separate from the mass. Per rectal examination suggested firm to hard mass deeply impacted in pelvis with healthy rectal mucosa. Her preoperative investigations were within normal limits.

The patient was evaluated for the same complaints. Abdominal ultrasonography showed large heterogeneous solid pelvic abdominal mass arising from pelvis extending bilaterally up to lumbar region respectively & superiorly up to epigastric region. Uterus & ovaries couldn’t be demarcated separately from the mass. MRI report revealed large heterogeneous mass measuring 22 x 15 x 13 cm, arising from pelvis and extending into upper abdomen (Fig. 1). Mass was arising from the pelvis at the level of lower border of rectum. The rectal shadow showed compression and displacement posteriorly and laterally (Fig. 2).

The uterus outline was seen clearly with central endometrial lining. Uterus was compressed and displaced anteriorly. The mass was abutting the posterior wall of uterus but not infiltrating into it. Both ovaries could not be visualized. The mass showed hypointensity on both T1 and T2 sequences and also showed linear hyperintensities in it. Posteriorly the mass extended up to the presacral area. There was no associated destruction of sacrum. Superiorly the mass extended up to retroperitoneal level. There was no associated lymphadenopathy. MRI report concluded the mass to be retroperitoneal pelvic mass, likely retroperitoneal sarcoma arising from rectum/ vaginal wall. MRI images were re-reviewed with two different radiologists but the conclusive diagnosis remained the same. Cervical cytology showed normal report. FNAC report showed spindle cells. Tumor marker CA 125 levels were 38.8 U/ml (normal range 0.0 - 35.0 U/ml).

Exploratory laparotomy under general anaesthesia revealed a large cervical fibroid 35 x 26 x 22 cm arising from posterior wall of the cervix. The uterus was normal in size sitting on top and both fallopian tubes and ovaries were normal. Total abdominal hysterectomy with bilateral salpingo-oophorectomy was performed (Fig. 4). Cut section showed whorled appearance suggestive of leiomyoma. Histopathological examination confirmed the diagnosis of cervical leiomyoma. Her postoperative recovery was uneventful and was discharged by day 7.

**DISCUSSION**

Cervical fibroids can have different presentation. Supravaginal fibroids can be central surrounding the entire cervical canal and lying centrally in the pelvis displacing the uterus superiorly. They can also be unilateral or bilateral, can be intramural or subserosal, and can be lying in the pelvis. Presence of isolated leiomyoma in cervix with intact uterus is infrequent. Cervical fibroids are uncommonly seen with excessive growth. Treatment of cervical fibroid is either hysterectomy or myomectomy. They may give rise to greater surgical difficulty by virtue of relative inaccessibility and close proximity to bladder and ureter.

There were various management dilemmas in this case i.e:

1. Whether the working diagnosis was leiomyoma, ovarian mass or retroperitoneal mass?
2. How can the definitive diagnosis we made?

Cases are being reported with abdominopelvic masses mimicking ovarian tumor which were diagnosed...
to be huge cervical fibroid, intra-operatively.\(^7,8\) Similar to the case reports, our case had an abdomino-pelvic mass with gradual increase in size. There were no-bladder symptoms. Imaging didn't reveal the mass to be fibroid uterus, but, instead, prime suspicion was of retroperitoneal mass. Ovaries were also not visualized even on MRI.

Unlike the fibroids with excessive growth which are known to cause pressure symptoms, our patient had gradual abdominal distension, bloating sensation, dyspepsia and off and on discharge per vaginam. There was high suspicion of retroperitoneal sarcoma in the present case and thus FNAC was done to reach at diagnosis.

Final plan for the patient was exploratory laparotomy and proceed. Intra-operatively the dilemma was cleared and we performed total hysterectomy with bilateral salpingo-oophorectomy.

This case report exemplifies that though the new diagnostic modalities like ultrasound and MRI have improved the accuracy of pre operative diagnosis, the final diagnosis can only be made at laparotomy.

REFERENCES

Case Report

Initial management of complete laryngotracheal separation: a case report

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ABSTRACT

Laryngotracheal trauma can be an immediately life-threatening injury if diagnosis or treatment is delayed. Failure to recognize acute injuries or to observe the principles of management can lead to laryngotracheal stenosis. Emergency room physicians, trauma surgeons, anesthesiologists, and especially otolaryngologists should maintain a high level of awareness of and suspicion for laryngotracheal trauma whenever a patient presents with multiple injuries in general or with cervical trauma in particular. Treatment in experienced hands will usually result in a favorable outcome. A case of laryngotracheal trauma was brought to emergency. After securing airway, complete laryngotracheal separation with tear in anterior wall of oesophagus was found. Laryngotracheal anastomosis with closure of oesophageal perforation was performed. A glove finger filled with two merocel was used as a stent for support.

Key words: Laryngotracheal Trauma, Penetrating Injury

INTRODUCTION

Laryngotracheal trauma is a rare injury, accounting less than one percent of trauma cases seen in most major centers.¹² This is because of the anatomy and location of larynx.³ The larynx and cervical trachea protected inferiorly by sternum, superiorly by mandible, posteriorly by cervical spine and sternocleidomastoid muscle on both side. The laryngeal framework supported by muscles in all direction except posteriorly.⁴ The infrequency of laryngeal injuries in survival trauma cases, larynx and trachea trauma may too often be ignored or overlooked. The blunt and penetrating laryngotracheal injuries can cause airway obstruction and death at scene of an accident. It is second to only intracranial injury as the most common cause of death among patient of head and neck trauma.⁵ Initial intubation or tracheostomy in patients with respiratory problem may introduce risk of injuries.

In recent years, advances in emergency services including well equipped ambulance services with rapid transport to hospital, skilled staff with high standard facilities, have improved survival rate.

CASE REPORT

Sixty five years male patient was referred to our department with one day history of head and neck injury following household assault. There was bleeding from wound site. The bleeding was minimal in amount and

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was stopped by pressure only. He had history of loss of voice and difficulty in swallowing, which was sudden in onset. The patient was also complaint of respiratory difficulty in upright position. There was oral and nasal bleeding. On examination of the neck revealed multiple penetrating wound seen in the right side with subcutaneous emphysema (Fig. 1). On further careful evaluation, one wound extending deep and was moved with respiration. After basic investigations and informed consent, the patient was taken up for further assessment and management under general anesthesia. The low tracheostomy was performed by separate incision. The direct laryngoscopy showed laryngotracheal separation four cm below vocal cords. The trachea was found divided in two parts with multiple pieces of anterior tracheal wall cartilages (Fig. 2). The oesophagoscopy showed 1 cm rent in the anterior wall and was three cm below cricopharynx. The oesophageal rent was repaired by interrupted suturing with 3-0 vicryl. After carefull debridement of lacerated tissue the cartilage pieces were sutured together with 4-0 prolene and were stabilized by a stent prepared from finger glove filled with two merocel (Fig. 3). This stent was fixed in position with cervical skin by through and through suturing with 3-0 silk on both side (Fig. 4). The wound was closed in layers. The patient was kept on I.V antibiotics, analgesic, antireflux therapy and Ryle’s tube feeding. The stent of finger glove and Ryle’s tube was removed on 7th postoperative day. The check direct laryngoscopy was done after 6 weeks. The subglottic and trachea was normal with minimal narrowing at wound side. The patient was decannulated in ward after four weeks. The patient was symptom free after six months.

Fig 2. Fractured cartilage of trachea, endotracheal tube in situ with separate incision.

Fig 3. Laryngeal stent (prepared with finger glove and merocel)

Fig 4. Repaired trachea with finger coot in situ and secured with suture throw trachea and stoma.
DISCUSSION

Upper airway injuries are relatively rare, mainly because the larynx and trachea are protected by their position relative to the bony protection of the mandible, sternum and cervical spine. A high index of suspicion is required to make the diagnosis, as they are often associated with other more obvious injuries such as closed head injuries, cervical spine injuries, facial trauma and chest trauma. The common causes for blunt and penetrating laryngotracheal trauma include road side accident, gunshot and stab injuries. Estimates of incidence varies from 1:660-65 to 1:125 trauma admissions. The cricoid cartilage and cricothyroid membrane involved in 50% of cervical injuries. Complete laryngotracheal separation has been reported to occur in as many as 63% of patients with blunt airway injuries and cricoids cartilage injuries are mostly associated with it. In such kind of injuries, the airway held in close approximation by peritracheal connective tissue, soft tissue of neck and mediastinum. The major signs and symptoms for laryngotracheal trauma includes subcutaneous emphysema, dyspnoea, stridor and inability to tolerate upright position.

The minor signs and symptoms include tenderness, local swelling, hoarseness, dysphagia and haemoptysis. The investigation battery includes X ray neck lateral view that may reveal subcutaneous emphysema and cervical spine injuries. The X ray chest may reveal pneumothorax and pneumo-mediastinum and CT scan is done to assess the integrity of laryngeal skeleton. The indirect laryngoscopy and flexible nasolaryngoscopy allows assessment of vocal cord mobility, patency of airway above trachea and integrity of laryngeal mucosa. A suspected oesophageal injury can also be evaluated with a water soluble gastrograffin swallow study. Laryngotracheal trauma graded depending on the severity of injury as per Schaefer’s classification.

i. Minor endolaryngeal hematomas or lacerations, without detectable laryngeal fractures.

ii. Laryngeal oedema or hematoma, or minor mucosal disruption without exposed cartilage

iii. Massive oedema, large mucosal lacerations, exposed cartilage, displaced fractures, vocal cord immobility.

iv. As per group iii, but with comminuted or unstable fractures.

The treatment options for stable airway include observation in monitored setting, humidified inspired air, reverse tredlenberg position, broad spectrum antibiotics, steroids and antireflux therapy. The management options for unstable airway includes intubation, cricothyroidotomy or tracheostomy in local or general anesthesia. The panendoscopy to see the site and area involved in trauma, distance from vocal cord. The open surgical repair indicated laryngotracheal separation, fracture of cartilages in blunt and penetrating trauma with stent to support the fracture segment and to prevent future stenosis by healing as we have done in our case. The oesophageal injury was repaired during the same operating sitting. The ultimate goal for long term treatment to provide adequate airway, prevent aspiration and to restore ventilation, deglutition and phonation to pretrauma quality.

ACKNOWLEDGMENT

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Mucocele of the junction of hard and soft palate - unusual location

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ABSTRACT

Mucoceles are common minor salivary gland lesions clinically characterized by single or multiple, spherical, fluctuant nodules which are generally asymptomatic. The most common locations of mucoceles are lower labial mucosa, floor of mouth, ventral tongue, buccal mucosa, retromolar area and palate. The incidence of mucocele on the palate is reported to be low (0.7- 4.3%). This paper presents a case report of mucocele of an unusual location at junction of hard and soft palate. A 57 year old woman presented with a painless swelling on the left side of palate posterior to the last molar tooth that increased in size gradually in six months. The histopathological report confirmed the diagnosis of mucocele. Postoperatively, the operated site healed completely with no recurrence.

Key words: Mucocele, palate, junction, soft palate.

INTRODUCTION

Mucoceles are common minor salivary gland lesions clinically characterized by single or multiple, spherical, fluctuant nodules which are generally asymptomatic.¹,² Currently, the term “mucocele” (also known as mucus extravasation phenomenon or mucus escape reaction) is used for lesions resulting from mucin spillage from a ruptured salivary duct and they do not exhibit a true cystic epithelial lining, although a ruptured salivary duct occasionally may be found adjacent to a mucocele. In contrast, the term “salivary duct cyst” (also known as the “mucus retention cyst,” “sialocyst,” or “mucus duct cyst”) is used for cysts lined by salivary ductal epithelium.³ In this report, the terms “mucocele” is used for “mucous extravasation phenomenon” as per these definition.

The mucous extravasation phenomenon is more common and occurs most frequently on the lower lip. This possibly relates to a higher incidence of mechanical trauma to the salivary duct, such as from biting, although the lesion also can occur on the floor of the mouth, cheek, upper lip, tongue, retromolar fossa, and palate.⁴,⁵

The most common locations of mucoceles are lower labial mucosa, floor of mouth, ventral tongue, and buccal mucosa; and infrequent sites included the palate and retromolar area. The incidence of mucocele on the palate is reported to be low.³ The English literature was searched and very few case reports are published for the mucocele of palate. Only one case has been reported for the mucocele on the junction of hard and soft palate.⁴ This paper presents a case report of solitary swelling found on the junction of hard and soft palate which was confirmed to be mucocele on histopathological examination.

CASE REPORT

A 57 year old woman presented to our department with a painless swelling on the left side of palate posterior to the last molar tooth for six months. The swelling increased in size gradually with no history of trauma or injury in that specific area. She had a habit of smoking for 10 years. Her medical history included diabetes mellitus and hypertension, both controlled by medication.

Intraoral examination showed bluish coloured spherical swelling on the left side of the palate at the junction of hard and soft palate, approximately 1 cm medial to left maxillary molars which measured about 1.5 cm x 1.5 cm and no discharge was noticed (Fig 1). It was firm in consistency, non tender, smooth surfaced, and there was no pulsation or bruit on palpation. A panoramic and intraoral radiograph revealed no involvement of bone.
Excisional biopsy was done under general anaesthesia. The lesion was dissected gently from the surrounding tissue and in-toto excision was done. Along with lesion adjacent minor salivary glands were also removed. After excision the junction of hard and soft palate was visible at the base together with the surrounding palatine muscles. Haemostasis was achieved and Surgicel® was packed before closure.

The histopathological report showed collagenic wall and indistinct lining. Other areas showed lobules of seromucinous glands and cystically dilated ducts containing mucinous secretions [Fig 2 (a), (b), (c)], so a diagnosis of mucocele was confirmed. Postoperatively, the operated site completely healed in 3 months. After one year follow up no recurrence is detected (Fig 3).
DISCUSSION

Mucoceles of the minor oral salivary glands are common lesions that result from damage to the excretory duct that leads to pooling of mucus into the connective tissue. No significant gender predilection is seen and most of the cases arise in children or young adults. Mucoceles clinically present as small, soft, discrete swellings of the mucosa and range from normal pink to deep blue in color. The most common locations of mucoceles are lower labial mucosa (81.9%), floor of mouth (5.8%), ventral tongue (5.0%), and buccal mucosa (4.8%); and infrequent sites included the palate (1.3%) and retromolar area (0.5%).

The incidence of mucocele on palate reported in the English literature is low ranging from 0.7-4.5%. In long case series by various authors, the reported incidence of mucocele on palate by Cataldo et al is 4.5%, Chi et al is 1.3% and Re Cecconi et al is 0.7%. The Medline literature was searched for the mucocele of the junction of hard and soft palate. To the best of our knowledge, only one case has been reported in the study of Cohen on 80 patients of mucocele in the English literature for the presentation of mucocele at this unusual location.

Cohen has reported a review of 80 cases of mucocele. They were located on the lower lip in 65%, floor of mouth in 20%, cheek in 10%, retromolar fossa in 3%, tongue in 1% and junction of hard and soft palates in 1%. Surgical excision with removal of the involved accessory salivary gland has been suggested as the treatment. Marsupilization usually result in reoccurrence. Large lesions are best treated with an unroofing procedure (marsupilization). If the fibrous wall is thick, moderate-sized lesions may be treated by dissection. If this surgical approach is used, the adjacent minor salivary glands must be removed. Care should be taken to avoid the injury to any marginal glands and ducts which may lead to reoccurrence of the lesion. The excised tissue should always be submitted to the pathological investigations to confirm the diagnosis and rule out the salivary gland tumors. Laser ablation, cryosurgery, and electrocautery are approaches that have also been used for the treatment of the mucoceles with variable success. The incidence of mucocele at this unusual location is rarely reported. So there is importance of considering this lesion in the differential diagnosis of lesions of the junction of hard and soft palate.

Conflict of Interest Statement : There is no conflict of interest related to financial or personal relationships with any people or organisation.

REFERENCES

Neglected post-burn severe joint contractures of hand - rehabilitation by surgical management - a case report

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ABSTRACTS

Burns have always been a grievous injury and restoration of pre-injury status becomes a challenge for the treating orthopaedician. A healed burn patient may be left with many scars that have varying degrees of functional and aesthetic component. We hereby present a case of neglected joint contracture in hand of a young female who develop the disorder following a burn injury treated by soft tissue release and joint stabilisation.

Keywords : Burns, Contracture, Surgery

INTRODUCTION

An extensive burn is one of those devastating injury which a patient can hope to survive. In case of burns, the restoration to pre-injury status becomes most important for the treating team. A healed burn patient may be left with scars that have varying degrees of functional and aesthetic components.

Post-burn contractures are distressingly common and severe in developing nations. Failure to seek medical help, inadequate medical care, and inadequate post-healing care are common causes of burn contractures. In case of neglected burn patients, scar contractures adjacent to or across the joints lead to disabling deformities. Contractures can begin as a slight puckering of scar tissue but over time they can worsen, becoming thick bands of hypertrophic scars. These tight bands of scar tissue can restrict joint movement, lead to the loss of joint mobility, and permanently impair normal joint function. The bones may be deformed and joints can get dislocated, especially in growing children. We report a case of neglected joint contracture in hand of a young female who developed the disorder following a burn injury treated by soft tissue release and joint stabilisation with excellent results.

CASE PRESENTATION

A 11-year-old right-hand-dominant female, a school student, presented with ten year old history of burns to the wrist of her left hand due to scalds by hot water which was treated in the local hospital by daily dressing. The burn wound healed in approximately 15 days. Six months after the burn injury, she was unable to extend her left thumb. The condition was progressive and deformity worsened with time (Fig.1A-B). The patient neglected the deformity and did not receive any form of treatment before coming to our hospital.

Physical examination revealed a thick and rigid post-burn scar over volar aspect of left wrist extending up to dorsal aspect of first web space healed with secondary intention. Left thumb had 'Z' shaped deformity with flexion of metacarpal, hyperextension of proximal phalynx and flexion of distal phalynx. Thumb was completely stiff and non-functional and due to this, patient was unable to perform majority of active movements with her left hand. Movements of rest of the fingers of left hand were within normal limit (Fig.2A-B).

X-rays of the left hand revealed that there was palmar flexion of first metacarpal with subluxation of first capo-metacarpal joint, dorsal dislocation of first metacarpo-phalangeal joint along with flexion at interphalangeal joint. Arthritic changes were not present at any joint (Fig. 3A-B).

TREATMENT

The patient was taken for reconstructive surgery of thumb and by using microsurgical techniques and magnifying loop, multiple 'Z'-plasties for soft tissue release and K-wire fixation of joints after open reduction were done in a single sitting (Fig. 4A-B).
Ravi Gupta et al: Neglected post-burn severe joint contractures of hand - rehabilitation by surgical management - a case report

Fig 1A-B: Showing post-burn scar and deformity of thumb of left hand.

Fig 2A-B: showing grossly deformed thumb with stiffness of movements in all planes.

Fig 3A-B: Postero-anterior and lateral X-rays of left hand showing dorsal dislocation of metacarpo-phalangeal joint and flexion contracture of interphalangeal joint of thumb.
After the surgery, hand was immobilised in cast for 4 months. After 4 months, cast and K-wires were removed and gentle physiotherapy and exercises were started. At the end of 8 months post-operatively, patient's hand deformity was fully corrected and she was able to do every movement of thumb including extension and opposition which were not possible before (Fig. 5A-D). Hand of the patient also got an aesthetic and normal look.
which is of significant psychological and social importance.

**DISCUSSION**

Burn contractures of the hand can produce a significant impact on quality of life by reducing a patient's ability to perform activities of daily living. Appropriately timed and selected operations will achieve optimal functional benefits while keeping morbidity to a minimum. The range of possible procedures available for the reconstruction of the neglected post-burn joint contractures in hand has increased greatly in the past 15 years. The choice of the correct procedure and technique suitable to the contracture must be well considered to achieve optimal results. Inappropriate techniques and procedure may result in injury to neurovascular bundles in hand leading to necrosis, gangrene and inferior results. Microsurgical techniques and instruments should be used to avoid such dreadly complications while dealing with severe deformities in hand. Caution should be taken when reconstructing such severe cases without the availability of hand surgeon as this is likely to limit the possible outcomes.

Although such facilities may mean travelling significant distances for these patients, it is still preferable for them to have a single hospital admission, the aim of which is to return them to a financially productive level of function for the rest of their life.

**REFERENCES**

Post human bite pinna reconstruction: a case report

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ABSTRACT

Traumatic amputation of pinna following a human bite is uncommon though not rare. These are usually the result of domestic violence and usually inflicted by closed relatives and siblings. The resulting cosmetic deformity is distressing for the patient and reconstruction of the defect is immediately warranted. Here we present a case of partial amputation of pinna by human bite which was repaired in two stages with a six weeks gap. Patient had a good cosmetic outcome after the repair and is well till the last follow up which was one year after the procedure.

Keywords: Pinna, Human bite, Pinna Reconstruction

INTRODUCTION

Traumatic auricular amputation due to human bite is not a common event. The traumatic loss of an ear constitutes a great aesthetic deformity and considerably affects the patient’s psychology. In addition, the severed ear constitutes a major challenge for the head and neck or plastic surgeon particularly when a human bite is the cause, taking into account the high possibility of severe contamination by the bacteria of oral flora. The difficulty of reconstruction is mainly related to the unique anatomical structure of the auricle, with fine skin covering, a thin and elastic cartilage, and small size vessels responsible for its perfusion.¹,²

Different operative techniques can be used for its repair. The extent of injury and the defect is the deciding factor for surgery. There may be complete or partial loss of the auricle. The partial loss may be of upper, middle or lower auricle transversely and may include loss of cartilage. Vertical loss may be of helix only or includes antihelical structures. Direct closure may be attempted when the loss is small i.e., less than 1-2 cm but larger defects require planned repair and reconstruction.³,⁴ Helical defects can be corrected by advancement of auricular skin and cartilage, composite grafts, free skin graft or post auricular skin flaps.⁵

This article presents a case of 38 year old male with amputation of helix of left pinna due to human bite that was reconstructed with the post aural skin under local anaesthesia in staged manner.

CASE REPORT

Thirty eight year old male presented with chief complaints of injury to left pinna since 14 days after fight with his brother. During the fight his left pinna was bitten off that was causing pain and profuse bleeding. The avulsed part of pinna was brought to a local hospital for its repair. He was told that it cannot be repaired and was discarded. Injectable medication and pressure dressing was applied. Thereafter, regular daily dressing was done from the local hospital. He received 3 injections of immunoglobulin antirabies. There was no history of decreased hearing, facial asymmetry, abnormal sounds in ear, rotatory sensation of moving around, nausea/vomiting. The past history, personal history, family history were not significant. His general physical examination and systemic examination were unremarkable. Examination of left pinna revealed a laceration 4 cm in size in left helix, with complete loss of skin and partial loss of cartilage (Fig.1). Rest of the ENT examination was normal.

His routine blood investigations were within normal range. We planned reconstruction of left pinna under local anesthesia in a staged manner. In first stage, margins of lacerated part of pinna were freshened and anterior margin undermined (Fig. 2A). Separate incision was made in post aural region parallel to left helix which was equal to the length of laceration and then undermined. Anterior
margin of laceration was sutured with posterior margin of post auricular skin with 3, O silk. (Fig. 2 B)

After 6 weeks second stage reconstruction was done wherein incision was made parallel to helix with one cm margin (Fig. 2C) and then pinna was released from post auricular area (Fig. 2D). Then posterior skin was mobilized anteriorly and primary suturing was done (Fig. 2E). Corrugated drain was fixed which was removed on third post-operative day. The sutures were removed on 7th post-operative day and the healing was satisfactory (Fig. 2F).

**FOLLOW UP:**

There was no complication in post operative period. Patient was subjectively and cosmetically satisfied till last follow up of one year (Fig. 3).

**DISCUSSION**

Human bites are the third most common bites, with an
overall infection rate of 18%. Most of the victims are young male adults. The difference may reflect social differences and the fact that most of the injuries are sustained during fight. It is interesting to observe that close relatives, including brothers and nephews, as well as friends and sexual rivals, are the usual assailants.6

Various types of pinna injuries can be skin avulsion larger than 5 mm, severe crush injuries, complete or nearly complete avulsions or amputations, auricular hematoma, cartilage defects larger than 5 mm, obvious devitalization, and total ear avulsion. Ear and nose are common target areas for human bites. Most bites occur secondarily to aggressive activities. The risk of infection from human bites depends on the area bitten and how quickly and adequately irrigation and debridement are undertaken. Despite human saliva containing $10^8$ bacteria per ml the incidence of infections is less in facial trauma caused by human. The high vascularity of the head and neck region makes infection less likely following a human bite than other areas which are prone to such injury, e.g. the hand in the clenched fist, punch injury.7

From medico legal point of view, forensic investigation of human and victims demands careful photography of wounds. DNA profiling and matching of dental impressions, bite marks can be done for prosecution of human aggressors.8

The most appropriate management of facial primary versus delayed repair and the necessity for intravenous antibiotics remains unanswered, as only a few studies in the literature have addressed these issues. The data that are available in the literature have focused mainly on extremities. Favorable factors in management of facial wounds are rich vascularity, appropriate choice of antibiotics and thorough debridement.7 Facial bites can be closed primarily, since bleeding is profuse and wounds are easily cleaned. In established wound infection, sutured wounds should be opened and drained.9

Partial traumatic amputation of the part of pinna is a rare occurrence; many treatment modalities have been used up to date.10 However, none of them have solved the problem in a definite manner. The simple reattachment of the avulsed part of pinna as a compound graft usually cause necrosis and can lead to total loss of the organ.10 To solve this problem, many techniques have been advocated in order to enhance the take up of a replanted part of pinna. Some authors have suggested the removal of the skin from the cartilage followed by burial of the cartilage alone under the postauricular skin or at a distance, and reconstruction of the ear in staged fashion.11 However, the cartilage, denuded of its dermal coverage, becomes distorted due to scarring and the end result after these procedures is not that satisfactory.1

In 1971, Mladick et al. made use of the retroauricular pocket, for non microsurgical ear reattachment. This method involved deepithilization of the amputated part followed by anatomic reattachment to the amputated stump and then burial in a retroauricular pocket.11 In this way, a larger area of inset and greater surface of contact with the vascular bed was provided for the graft, thus allowing for better composite graft take.

Park et al. described another technique for amputated auricular cartilage burial, by removing all skin from the graft except over the helix area. The denuded cartilage is then sandwiched between a retroauricular flap anteriorly and a facial flap posteriorly. However, the unburied helical skin can undergo necrosis, while three stages are required to achieve a satisfactory result.12 A similar technique has been proposed by Destro and Speranzini, in which all the skin is removed from the graft except over the concha. Multiple small perforations are made in the cartilage which is then covered with a postauricular flap. A second operation is required for elevation of the ear.13

**CONCLUSION**

Pinna reconstrutution is a procedure that does not require microsurgery. It is a simple technique that produces excellent aesthetic results, while preserving all neighbouring tissues.

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Mandibular reconstruction- a case report and rare experience

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INTRODUCTION

Mandibular deformities and defects may result from trauma, infections, prior radiation exposure, neoplasms, and congenital defects. Most mandibular deformities result from ablative surgery for neoplasms. The mandible plays a major role in airway protection, support of the tongue, lower dentition and the muscles of the floor of the mouth permitting mastication, articulation, deglutition, and respiration. It also defines the contour of the lower third of the face. Interruption of mandibular continuity, therefore, produces both a cosmetic and functional deformity. The resulting dysfunction after loss of part of the mandible varies from minimal to major. Loss of mandibular continuity results in deviation of the mandible toward the resected side due to the unopposed pull of the remaining muscles of mastication and soft tissue contracture and scar formation. When undertaking mandibular reconstruction, the restoration of bony continuity alone should not be considered the measure of success. The functions of chewing, swallowing, speech articulation, and oral competence must also be addressed. The ultimate goal of mandibular reconstruction is to return the patient to their previous state of function. In order to achieve this goal, the reconstructive surgeon must attempt to restore bony continuity and facial contour, maintain tongue mobility, and attempt to restore sensation to the denervated areas. Oral rehabilitation postoperatively is important to improve the patients ability to manipulate the food bolus, swallow, and articulate speech. We are presenting our rare experience of mandibular reconstruction.

CASE REPORT

Seventy year old female patient presented with history of ulcer in right side of oral cavity for the last three months. She also had complaint of bleeding from ulcer and difficulty in chewing along with pain. There was no history of neck swelling or any addiction. On examination, an ulcer was present, involving right side of mandible extending anteriorly from lateral incisor till 1st molar, medially one cm from alveolar margin and laterally involving lower gingivobuccal sulcus. The pre-operative biopsy report was squamous cell carcinoma and orthopantogram showed lytic lesion involving right side of mandible (Fig.1). After medical fitness, the patient was

Fig 1. Pre-operative orthopantogram showing lytic lesion involving right side of mandible

Fig 2. A superiorly based sternocleidomastoid muscle pedicle flap, used to cover the defect
subjected for wide local excision, supraomohyoid neck dissection along with segmental mandibulectomy under general anaesthesia. The reconstruction was done with iliac crest graft along with titanium plate and screws. A superiorly based sternocleidomastoid muscle pedicle flap was used to cover the defect (Fig. 2). The patient was kept in ICU for one day. Broad spectrum intravenous antibiotic was given in the post-operative period for ten days. She was kept on Ryles tube feeding for three weeks. The post-operative orthopantogram showed well placed graft and titanium plate (Fig. 3). The final biopsy report showed disease free margin with two metastatic lymph node out of 11 nodes. The patient received curative dose of radiotherapy. She was disease free at the time of last follow up.

**DISCUSSION**

With increasing extent of bony loss, severe functional and cosmetic deformities result which necessitate reconstruction in order to restore quality of life. Defects which are lateral and limited to the mandibular body often cause only minimal cosmetic and functional deformity. When evaluating defects that involve the mandibular ramus, it is important to note if the patient has a proximal segment of bone, a functioning temporomandibular joint, or a condylar neck to which the graft may be secured. Radiographic analysis of the bony mandibular anatomy can be very helpful when formulating a plan for oromandibular reconstruction.

Mandibular reconstruction can be done by non-vascularized or vascularized bone graft. Free bone grafting was the first method of reconstructing mandibular defects and was initially reported by Bardenheuer in 1881. With the advent of pedicle flap procedures and free tissue transfer, combined with the advent of steel and titanium reconstruction plates in the 70's and 80's, the reconstructive options using these materials increased. In previous decades, delayed reconstruction of mandibular defects was favored over primary reconstruction secondary to the belief that primary reconstruction could potentially mask tumor recurrence. Improved treatment modalities and the advent of microvascular free tissue transfer techniques have resulted in the resurgence of primary oromandibular reconstruction. More effective imaging and more effective adjuvant therapies have improved mapping and control of primary tumors. Non-vascularized autogenous bone grafts can be used for reconstruction of small to medium size mandibular defects. These can be harvested from the patients calvarium, rib, ilium, tibia, fibula, scapula, humerus, radius, and metatarsus and provide viable and immunocompatible osteoblastic cells. When considering the use of nonvascularized bone grafts, the ideal soft tissue bed would have enough bulk, vascularity, and cellularity in order to incorporate the bone graft. However, tissue loss, scar contracture, and prior irradiation often make secondary reconstruction difficult and decrease the chances of success. The availability of large vascularized segments of bone which can be transferred have allowed wider resections with oncologically sound margin size reducing the tendency to conserve tissue at the expense.

![Fig 3. Post-operative orthopantogram showing well placed graft and titanium plate](image3)

![Fig 4. Post-operative picture showing the disease free status at the time of last follow up](image4)
of adequate margins. Microvascular reconstruction of bone, soft tissue, and skin can now be used for primary reconstruction with a high rate of success. The ability to restore sensation to flaps has further enabled patients to achieve marked improvements in oral competence, speech, and swallowing. In the case due to non-availability of plastic surgeon, iliac crest and rib graft were the only option left for reconstruction of mandible. We chose iliac crest graft because ENT surgeons are familiar with this, causing less complications, away from field of work will reduce surgery time, curved piece of bone of up to 16 cm in length, ideal for receiving osseointegrated implants.

Mandibular reconstruction plates and screws are the most widely used alloplastic devices for mandibular reconstruction.\textsuperscript{7,8} The most common metals used in the fabrication of these plates are stainless steel, vitallium, and titanium. Vitallium is an alloy of cobalt, chromium, and molybdenum. This type of plate initially seemed to be ideal, however, the low malleability can make application difficult. Stainless steel and titanium reconstruction plates were developed in an attempt to find a mandibular reconstructive option that was fast, single-staged, and reliable while maintaining oral function and form. These plates have been used with varying rates of success. The development of the titanium hollow osseointegrated reconstruction plate (THORP) was an attempt to address the failures of the older plating systems.\textsuperscript{9} This plate has a hollow screw made of titanium with perforations along the screw body which allow bone ingrowth and result in increased plate stability at the bone-screw interface. An expansion bolt within the screw head allows the plate to be anchored to the interosseous screw instead of being compressed to the underlying mandible. This prevents pressure necrosis of the underlying bone decreasing the potential for plate failure at the screw-bone interface. Placement of mandibular reconstruction plates does not contraindicate the use of post-operative radiation therapy.

Bone graft healing occurs in two phases. Initially, new osteoid is deposited by osteoblastic cells which survive the transplantation process. The second phase contributes very little to the new bone mass. It begins about two weeks after implantation and continues indefinitely. It involves the revascularization, remodeling, and reorganization of the previously formed bone by osteoblasts and osteoclasts. Cancellous bone grafts, consisting of medullary bone and bone marrow, contain the highest percentage of viable osteoblasts. These grafts become revascularized rapidly due to their particulate structure and large surface area. This results in a higher percentage of surviving cells after transplantation. In contrast, cortical grafts consisting of lamellar bone struts contain large numbers of osteoclasts.

**ACKNOWLEDGEMENT**

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**REFERENCES**

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- **Discussion:** 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. What this study adds to the available evidence, effects on patient care and health policy, controversies raised by this study; an future research directions.

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