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RESEARCH ARTICLE

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COMPARISON OF PHYSICAL PARAMETERS AND THE PERCENTAGE PURITY OF DIFFERENT BRANDS OF DICLOFENAC SODIUM WITH THE LABELED CLAIM BY U.V METHOD

ABSTRACT

Now days so many pharmaceutical companies releasing the drugs for their commercial purpose with insufficient active ingredient in the dosage form as they claimed on the strip so to get awareness which pharmaceutical company gives appropriate active ingredient present in released dosage forms. The aim of this work is to compare Physical parameters and the Percentage purity of different brands of Diclofenac sodium with the labeled Claim by U.V Spectroscopy method.

KEYWORDS: Labeled Claim, U.V Method, Diclofenac, 220 nm,

INTRODUCTION

DICLOFENAC SODIUM

Diclofenac sodium is a strong anti inflammatory drug that exerts its action by inhibiting cyclooxygenase–I. Diclofenac sodium is white to slightly yellowish crystalline powder, slight hygroscopic. Peak plasma concentrations of Diclofenac sodium are achieved within one hour of oral administration. Diclofenac sodium has a half-life of 1.5-2.5 hours. It is rapidly absorbed when taken orally, dependent of food intake. Diclofenac sodium undergoes first-pass hepatic metabolism via CYP3A4 and CYP2C9 to its active metabolite, 4-hydroxydiclofenac,3-hydroxydiclofenac,5-hydroxydiclofenac, of which 4-hydroxy diclofenac is the major metabolite and is excreted through urine.

TABLETS[8-11]

Tablet is a solid dosage form each containing a unit dose of one or more medicaments. Tablets are flat or biconvex discs prepared by compressing a drug or a mixture of drugs with or without suitable excipients. Tablet may vary in shape and differ greatly in size and weight depending on the amount of medicinal substance and the intended mode of administration, with advancement in technology and increase in awareness towards modification in standard tablet to achieve better acceptability as well as bioavailability, newer and more efficient tablet dosage forms are being developed. The main reasons behind formulation of different types of tablets are to create a delivery system that is relatively simple and inexpensive to manufacture, provide the dosage form that is convenient from patient's perspective and utilize an approach that is unlikely to add complexity during regulatory approval process. To understand each dosage form, tablets here are classified by their route of administration and by the type of drug delivery system they represent within the route.

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1) **ORAL TABLETS FOR INGESTION** 1.1 Standard compressed tablets 1.2 Multiple compressed tablets I. Compression coated tablets II. Layered tablets III. Inlay tablets 1.3 Modified Release tablets 1.4 Delayed action tablets 1.5 Targeted tablets I. Floating tablets II. Colon targeting tablets 1.6 Chewable tablets 1.7 Dispersible tablets	2) **TABLETS USED IN THE ORAL CAVITY** 2.1 Lozenges and troches 2.2 Sublingual tablets 2.3 Buccal tablets 2.4 Dental cones 2.5 Mouth dissolved tablets
3) **TABLETS ADMINISTERED BY OTHER ROUTES** 3.1 Vaginal tablets 3.2 Implants	4) **TABLETS USED TO PREPARE SOLUTION** 4.1 Effervescent tablets 4.2 Hypodermic tablets 4.3 Soluble tablets

Table.1 VARIOUS TYPES OF TABLETS

Advantages and disadvantages of tablet as a dosage form:

The advantages are listed below:

I. Large scale manufacturing is feasible in comparison to other dosage forms. Therefore, economy can be achieved.

II. Accuracy of dose is maintained since tablet is a solid unit dosage form.

III. Tailor made release profile can be achieved.

IV. Longer expiry period and minimum microbial spillage owing to lower moisture content.

V. As tablet is not a sterile dosage form, stringent environmental conditions are not required in the tablet department.

VI. Ease of packaging (blister or strip) and easy handling over liquid dosage form.

VII. Easy to transport in bulk. Emergency supply supplies can be carried by patients.

VIII. Organoleptic properties (taste, appearance and odour) are best improved by coating of tablet.

IX. Product identification is easy and markings done with the help of grooved punches and printing with edible ink.

X. Different types of tablets are available like buccal, floating, colon targeting, effervescent, dispersible, soluble, and chewable, etc.

XI. In composition to parenterals dosage form, a doctor or a nurse is not required for administration. i.e. self administration is possible.

XII. In comparison to capsules, tablets are more tamper proof.

The disadvantages are listed below:

I. It is difficult to convert a high dose poorly compressible API into a tablet of suitable size for human use.

II. Difficult to formulate a drug with poor wettability, slow dissolution into a tablet.

III. Slow onset of action as compared to parenterals, liquid orals and capsules.

IV. The amount of liquid drug (e.g. Vitamin E, Simethicone) that can be trapped into a tablet is very less.

V. Difficult to swallow for kids, terminally ill and geriatric patients.

VI. Patients undergoing radiotherapy cannot swallow tablet.

INTRODUCTION TO INFLAMMATION:

Inflammation is part of the complex biological response of vascular tissues to harmful stimuli, such as pathogens, damaged cells, or irritants. Inflammation is a protective attempt by the organism to remove the injurious stimuli and to initiate the healing process. Although infection is caused by a microorganism, inflammation is one of the responses of the organism to the pathogen. However, inflammation is a stereotyped response, and therefore it is considered as a mechanism of innate immunity, as compared to adaptive immunity, which is specific for each pathogen. However, chronic inflammation can also lead to a host of diseases, such as hay fever, periodontitis, atherosclerosis, rheumatoid arthritis, and even cancer (e.g., gallbladder carcinoma).

Inflammation can be classified as either acute or chronic. Acute inflammation is the initial response of the body to harmful stimuli and is achieved by the increased movement of plasma and leukocytes (especially granulocytes) from the blood into the injured tissues. A cascade of biochemical events propagates and matures the inflammatory response, involving the local vascular system, the immune system, and various cells within the injured tissue. Prolonged inflammation, known as chronic inflammation, leads to a progressive shift in the type of cells present at the site of inflammation and is characterized by simultaneous destruction and healing of the tissue from the inflammatory process.

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TYPES OF INFLAMMATION[7]:

Acute inflammation:

Causatative agent: Bacterial Pathogens,

Major cells involved: Injured tissues Europhiles (primarily), Eosinophils and Basophils (response to helminthes worms and parasites), Mononuclear cells (monocytes, macrophages).

Primary mediaters: Vasoactive amines, Eicosanoids

Onset: Immediate **Duration:** Few days

Outcomes: Resolution, abscess formation, chronic inflammation.

Chronic inflammation:

Causative agent: Persistent acute inflammation due to non-degradable pathogens, viral infection, persistent foreign bodies, or autoimmune reactions.

Major cells involved: Mononuclear cells (monocytes, macrophages, lymphocytes, and plasma cells), fibroblasts.

Primary mediators: IFN-γ and other cytokines, growth factors, reactive oxygen species, hydrolytic enzymes.

Onset: Delayed **Duration:** Up to many months, or years

Outcomes: Tissue destruction, fibrosis, necrosis.

SIGNS AND SYMPTOMS

Acute inflammation is a short-term process, usually appearing within a few minutes or hours and ceasing upon the removal of the injurious stimulus. It is characterized by five cardinal signs. The traditional names for signs of inflammation come from Latin:

- Dolor(hyperglasia)
- Calor (increased heat)
- Rubour(redness)
- Tumour(swelling)
- loss of function

The first four (classical signs) were described by Celsus (ca 30 BC–38 AD), while loss of function was added later by Galen even though the attribution is disputed and the origination of the fifth sign has also been ascribed to Thomas Sydenham and Virchow.Redness and heat are due to increased blood flow at body core temperature to the inflamed site of swelling is caused by accumulation of fluid, pain is due to release of chemicals that stimulate nerve endings. Loss of function has multiple causes. These

five signs appear when acute inflammation occurs on the body's surface, whereas acute inflammation of internal organs may not result in the full set. Pain only happens where the appropriate sensory nerve endings exist in the inflamed area.

Eg: Acute inflammation of the lung (pneumonia) does not cause pain unless the inflammation involves the parietal pleura, which does have pain-sensitive nerve endings.

CAUSES: Inflammation can be caused by a number of factors that can damage cells but is broadly divided into :

1. Physical – can be mechanical as in a car accident injury or assault or environmental like severe cold and heat (burns).
2. Chemical – for example: Acid 'burns', drugs, venom.
3. Infection – bacteria, viruses, fungi and other parasites.
4. Ischemia – lack of or restricted blood supply which may eventually lead to death of tissue (necrosis) known as an infarct.
5. Immune – autoimmune conditions and allergy.

PATHOPHYSIOLOGY

The process of acute inflammation is initiated by cells already present in all tissues, mainly Resident macrophages, Dendritic cells, Histiocytes, Kupffer cells and Mastocytes. These cells present on their surfaces certain receptors named pattern recognition receptors (PRRs), which recognize molecules that are broadly shared by pathogens but distinguishable from host molecules, collectively referred to as pathogen-associated molecular patterns (PAMPs). At the onset of an infection, burn, or other injuries, these cells undergo activation (one of their PRRs recognize a PAMP) and release inflammation mediators responsible for the clinical signs of inflammation. Vasodilation and its resulting increased blood flow causes the redness (rubor) and increased heat (calor). Increased permeability of the blood vessels results in an exudation(leakage) of plasma proteins and fluid into the tissue (edema), which manifests itself as swelling (tumor). Some of the released mediators such as bradykinin increase the sensitivity to pain (hyperalgesia, dolor). The mediator molecules also alter the blood vessels to permit the migration of leukocytes, mainly neutrophils outside of the blood vessels (extravasation) into the tissue. The neutrophils migrate along a chemotactic gradient created by the local cells to reach the site of injury. The loss of function (functio laesa) is probably the result of a neurological reflex in response to pain. In addition to cell-derived mediators, several a cellular biochemical cascade systems consisting of preformed plasma proteins act in parallel to initiate and propagate the inflammatory response. These include the complement system activated by bacteria, and the coagulation and fibrinolysis systems activated by necrosis, e.g. a burn or a trauma. The acute inflammatory response requires constant stimulation to be sustained. Inflammatory mediators have short half lives and are quickly degraded in the tissue. Hence, acute inflammation ceases once the stimulus has been removed.

TREATMENT

Inflammation is usually a short term process intended to protect the body. Once the cause of the injury has been neutralized or the integrity of the living tissue has been secured, inflammation gradually subsides. Inflammation is a process and not a disease, however it may require treatment if it continues to persist or is causing significant discomfort in the lack of any threat to tissue integrity. In chronic conditions, inflammation is usually persistent but low grade, with occasional acute aggravations.

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Non-steroidal anti-inflammatory drugs:

These drugs, like paracetamol, usually act by inhibiting the cells from producing prostaglandins, the main chemical mediator of inflammation.

Corticosteroids

'Steroids' also inhibit prostaglandin formation by the cells as well as inhibiting the function of white blood cells which play an essential role in the inflammatory process.

Anti-histamines

Histamine is a chemical produced by the white blood cells like basophils and mast cells and is usually secreted in allergic responses. Histamine causes local inflammation and an anti-histamine drug blocks basophils and mast cells from producing and secreting histamine.

Hot & Cold Therapy

Certain applications may also assist with inflammation due to their physical effects on living tissue. Cold applications cause constriction (narrowing) of the blood vessels thereby inhibiting inflammation. Cold also assists with the signs and symptoms of inflammation by causing 'numbing' of the area thereby blocking pain and 'cooling' the area. Hot applications usually aggravate (worsen) inflammation but may help with easing the cause of inflammation like spasms or cramping of muscles. Hot and cold therapy should be used cautiously under the supervision of a medical practitioner to prevent tissue death (necrosis**)**

Drug profile

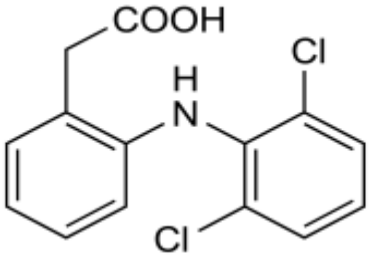

Figure No 1: Structure of Diclofenac sodium

IUPAC NAME: **Sodium2-[(2,6-dichlorophenyl)-amino] phenylacetate.**

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EMPIRICAL FORMULA	$C_{14}H_{10}Cl_2NNaO_2$
MOLECULAR WEIGHT	318.13
APPEARANCE	White to slightly yellowish crystalline powder, slightly hygroscopic in nature.
SOLUBILITY	It is freely soluble in methanol, soluble in ethanol, sparingly soluble in water.
METABOLITES FORMED	Aromatic hydroxylation takes place and the metabolites formed are 5-hydroxydiclofenac,4-hydroxydiclofenac.
DOSAGE FORMS AVAILABLE	It is available as tablets for oral administration containing either 25 mg, 50 mg and 100 mg and also available as gel
BRANDS AVAILABLE	REACTIN-50,100mg. NOVATAB 50,100mg. VOVERAN-50,100mg

Table.2

MECHANISM OF ACTION

- Inhibition of Arachidonic cyclooxygenase system, and decreasing the Prostaglandin production and Thromboxanes.
- Inhibition of arachidonic acid release and stimulation of its re up take.
- Inhibition of lipooxygenase pathway resulting in decreased production of Leukotrienes.

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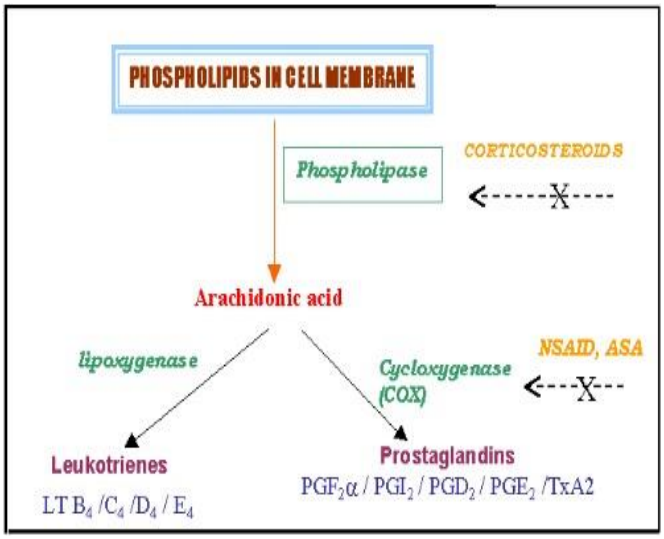

Figure 2: MECHANISM OF INHIBITION OF CYCLOOXYGENASE

INDICATIONS:

Indicated for patients in the conditions like

- Rheumatoid arthritis
- Juvenile idiopathic arthritis
- Ankolysing spondylitis
- Renal colic
- Dysmennorhea
- Migraine

DOSAGE AND ADMINISTRATION:

Diclofenac sodium delayed-release (enteric-coated) and extended-release tablets are not recommended for relief of acute pain or primary dysmenorrhea because of slow onset of action in.Adults.

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Inflammatory diseases:

Osteoarthritis:

May change dosage to 50 or 75 mg twice daily in patients who do not tolerate usual dosage. however, these dosages may be less effective in preventing NSAID-induced ulcers.

Preparation	**Dosage**
Diclofenac potassium conventional tablets	100–150 mg daily, given as 50 mg 2 or 3 times daily
Diclofenac sodium delayed-release tablets	100–150 mg daily, given as 50 mg 2 or 3 times daily or 75 mg twice daily
Diclofenac sodium extended-release tablets	100 mg once daily
Diclofenac sodium (in fixed combination with misoprostol)	50 mg 3 times daily

Table.3

Topical (gel)

For lower extremity (i.e., knees, ankles, feet) joint pain, massage 4 g of diclofenac sodium 1% gel into the affected joint 4 times daily.

For upper extremity (i.e., elbows, wrists, hands) joint pain, massage 2 g of diclofenac sodium 1% gel into the affected joint 4 times daily. If multiple joints are treated, total daily dose applied to all joints should be ≤32 g of gel daily.

Rheumatoid arthritis:

Oral

May change dosage to 50 or 75 mg twice daily in patients who do not tolerate usual dosage; however, these dosages may be less effective in preventing NSAID-induced ulcers.

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Preparation	Dosage
Diclofenac potassium conventional tablets	150–200 mg daily, given as 50 mg 3 or 4 times daily
Diclofenac sodium delayed-release tablets	150–200 mg daily, given as 50 mg 3 or 4 times daily or 75 mg twice daily
Diclofenac sodium extended-release tablets	100 mg once daily; may increase to 100 mg twice daily
Diclofenac sodium (in fixed combination with misoprostol)	50 mg 3 or 4 times daily

Table.4

Ankylosing Spondylitis*:*
Oral

100–125 mg daily (as diclofenac sodium delayed-release tablets); administer as 25 mg 4 times daily, with 5th dose at bedtime as needed.

Pain

Oral

50 mg 3 times daily (as Diclofenac potassium conventional tablets). Some patients may benefit from initial dose of 100 mg (followed by 50-mg doses).

Topical (transdermal system):

Apply 1 transdermal system (diclofenac epolamine 1.3%) twice daily.

Dysmenorrhea:

Oral

50 mg 3 times daily (as diclofenac potassium conventional tablets). Some patients may benefit from initial dose of 100 mg (followed by 50-mg doses).

PHARMACOKINETICS:

Diclofenac sodium well absorbed orally, rectal suppositories, intramuscular injection. It undergoes first hepatic metabolism and only 50% reaches systemic circulation. It is converted into5-hydroxydiclofenac,4-hydroxydiclofenac, 4,5-dihydroxydiclofenac, by aromatic hydroxylation. The metabolites excreted in form of glucoronide, sulfate conjugate, mainly in urine, bile.

HALF LIFE:

Half- life of oral preparations Diclofenac sodium is 1 to 2 hours.

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BIOAVAILABILITY:

Rapidly absorbed an oral administration, with peak plasma levels being reached within 1.5 to 2.5 hours. Highly bound to serum proteins, primarily albumin. Only 50 to 60% of an oral dose is bioavalable because of extensions of hepatic metabolism.

SIDE EFFECTS:

Cardiovascularrisk, Renaldamage, Hepatitis, Gastrointestina lbleeding, Diahorrea, Epigastricpain, Colonic ulceration, Convulsions, Aplastic anaemia, Thrombocytopenia, Neutropenia, Corneal toxicity, Rashes.

DRUG INTERACTIONS[7,13]:

a) Diclofenac when administered with ACE inhibitors, Cyclosporins, Tacrolimus it results in Nephrotoxicity.
b) Antihypertensive effects of ACE inhibitors, β-blockers, diuretics are decreased when given with Diclofenac sodium.
c) GI Bleeding increases with Corticosteroids**,** SSRI'S, Clopidogrel, and Ticlopidine.
d) Increased haematotoxicity with Zidovudine.
e) Detoriation of renal function with Diclofenac with Clopidogrel, increased concentration of Diclofenac.
f) Detoriation of renal function when used with Triameterine.
g) Decreased concentration of Diclofenac, when given with Sucralfate.

MATERIALS AND METHODS

I) **DRUG**: Diclofenac sodium a gift sample from srinija perantrals located at Peracharla,Guntur.
II) **SOLVENT USED** : Acetonitrile is a sample used from laboratory manufactured by merk laboratories, Mumbai.
III) **EQUIPMENTS USED**:

S.No	TEST	EQUIPMENT
1.	Weight variation	High Precision Balance
2	Hardness	Monsanto Hardness Test
3.	Friability	Roche Friabilator
4	Assay	EID Double beam UV-Visible Spectrophotometer model no: 1372

Table. 5

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Diclofenac sodium tablets of 50 mg of three different brands are coded as follows:

BRAND NAME	DOSE(mg)	CODE
Reactin	50	S1
Voveran	50	S2
Novatab	50	S3

Table. 6

Were kindly provided by Maruthi pharmacy store located at Sattenapalli. The 50mg tablets have expiry date till jan-2014, july-2014, and july-2014 respectively.

EXPERIMENTAL WORK

1)**Physical parameters of Diclofenac sodium :**

Weight variation:

With a tablet designed to contain a specific amount of drug in a specific amount of tablet formula, the weight of tablet being made is routinely measured to help ensure that a tablet contains the proper amount of drug.

Individual weights of twenty tablet were taken and the average weight was calculated and weight variation was calculated by using the following formula.

$$\text{Weight variation} = \frac{\text{weight of tablet} - \text{average weight}}{\text{Average weight of tablet}} \times 100$$

Weight variation should not be more than 7.5%.

The tablets meet the USP test if no more than two tablets are outside the percentage limit and if no tablet refers by more than two times the percentage limit.

Hardness:

Tablet hardness is defined as the force required to break a tablet diametrically. A tablet is placed between two anvils, force is applied to the anvils and the crushing strength that causes the tablet to break is recorded. Hardness is termed also termed as Tablet crushing strength. Devices used to measure the hardness Monsanto tester, strong Cobb tester, Pfizer tester, Erweka tester, Schleuniger tester. Hardness of the tablets was observed by the use of Monsanto hardness tester. Monsanto tester consists of a barrel containing a compressible spring held between two plungers. The lower plunger is placed in contact with the tablet, and a zero reading is taken. The upper plunger is then forced against a spring by turning a threaded bolt until the tablet fractures. As the spring is compressed, a pointer rides along a gauge in the barrel to indicate the force. The force of fracture is recorded, and zero force reading is deduced from it.

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Desired hardness was 2 to 4 kg/square cm.

Figure .3 Monsanto hardness tester

Friability:

The laboratory friability tester subjects a number of tablets to the combined effects of abrasion and shock by utilizing a plastic chamber that revolves at 25 rpm, dropping the tablets a distance of 6 inches with each revolution. Normally, a pre weighed tablet is placed in the friabilator, which is then operated for 100 revolutions. The tablets are then dusted and reweighed. Tablets that lose less than 0.5 to1% their weight of generally considered acceptable. When capping is observed on friability testing, the tablet should not be considered for commercial use, regardless of the percentage of lose seen. Friability of tablets was calculated by the use of Roche friabilator. Friability should be less than 1%.

$$\%\text{Friability} = (1 - W_0 / W) \times 100$$

W_0 = weight of the tablet before test

W= weight of the tablet after test

Figure.4 Roche friabilator

Thickness:

Tablet thickness, at a constant compressive load, varies with changes in die fill, with particle size distribution and packing of the particle mix being compressed, and with tablet weight, while with a constant die fill, thickness varies with variations in compressive

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load. Tablet thickness is consistent batch to batch or within a batch only if the tablet granulation or powder blend is adequately consistent in particle size and size distribution, if the punch tooling is of consistent length, and if the tablet press is clean and in good working order.

The crown thickness of individual tablets may be measured with a micrometer, which permits accurate measurements and provides information on the variation between tablets. Another technique involves placing 5 or 10 tablets in a holding tray, where their total crown thickness may be measured with a sliding caliper scale.

Tablet thickness should be controlled within a $\pm$ 5% variation of a standard value. Any variation in tablet thickness within a particular lot of tablets or between manufacturers' should not be apparent to the unaided eye for consumer acceptance of the product. In addition, thickness must be controlled to facilitate packaging. Difficulties may be encountered in the use of unit dose and other types of packaging equipment if the volume of the material being packaged is not consistent. A secondary packaging problem with tablets of variable thickness relates to consistent fill levels of the same product container with a given number of dosage units.

Thickness of the tablets was calculated by the use of Vernier calipers. Desired thickness was 3.5 to 4 mm.

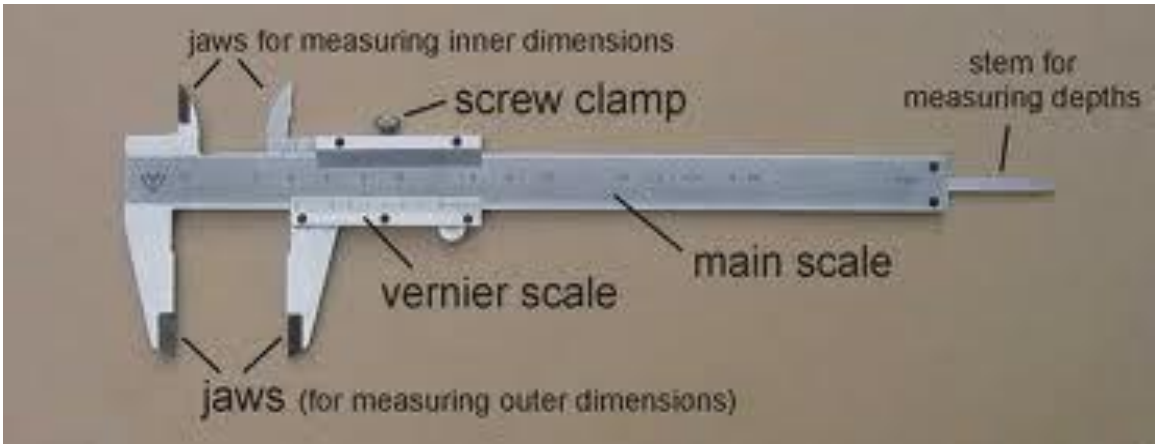

Figure.5 Vernier calipers

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S. no	Physical parameter	S1	S2	S3
1.	Weight variation	± 0.92	± 0.89	± 0.99
2.	Hardness	3.4kg/sq.cm	3.6kg/sq.cm	3.4kg/sq.cm
3.	Friability	0.46%	0.46%	0.7%
4.	Thickness	3.7	3.8	3.7

Table.7

2) **PREPARATION OF STANDARD SOLUTION OF DICLOFENAC SODIUM:**

Diclofenac sodium standard solution was prepared by accurately weighing 100mg of Diclofenac sodium in to 100ml volumetric flask and adding 60 ml Acetonitrile. The flask was shaken for 10 minutes, then final volume is make up to100ml with Acentonitrile.

3) **THE DETERMINATION OF λ_{max} OF DICLOFENAC SODIUM**:

The UV spectrums of Diclofenac sodium in solvent like Acetonitrile were recorded at the concentration of 6µg/ml. The spectrum of Diclofenac sodium was found to have good spectrum pattern and maximum absorbance was obtained. The maximum absorbance was found to be 220 nm for the Diclofenac sodium.

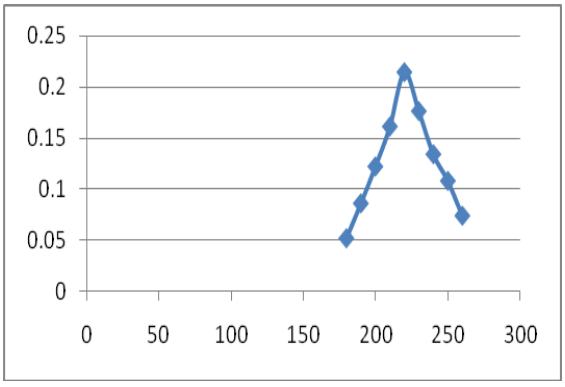

Figure.6 λ_{max} of Diclofenac sodium

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4) **PREPARATION OF CALIBRATION CURVE OF DICLOFENAC SODIUM:**

The pure compound of Diclofenac sodium 100mg dissolved in 100 ml acetonitrile. From this solution 1ml is taken in to 10ml volumetric flask and make up volume to 10ml. From this solution we have made different concentrations like 2 µg/ml, 4 µg/ml,6 µg/ml, 8 µg/ml, 10 µg/ml. The intra and inter day absorbance values are taken in to the consideration.

Data represent the absorbance of the different concentration of Diclofenac sodium.

S.no	Concentration (µg/ml)	Absorbance
1	2	0.052
2	4	0.105
3	6	0.149
4	8	0.214
5	10	0.268

Table.8

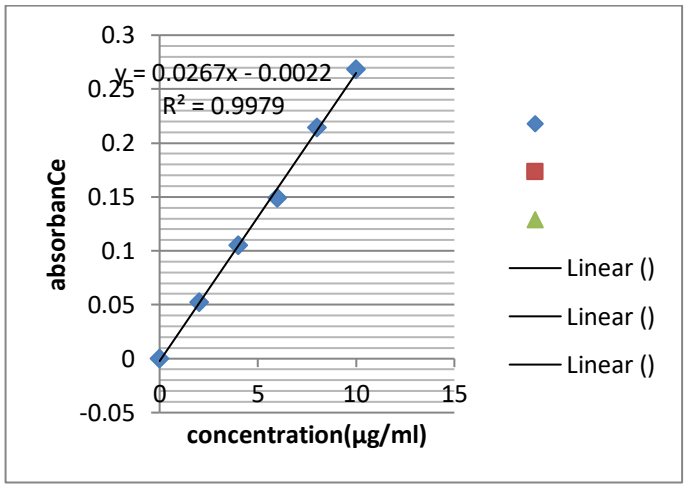

Figure.7 Calibration curveof Diclofenac sodium

4) **ESTIMATION OF DICLOFENAC SODIUM IN TABLET DOSAGE FORM:**

For the analysis of Diclofenac sodium in tablets, three different brands of 50mg[Reactin-50],[Voveran-50], [Novatab-50] strength were taken. Twenty tablets each of [Reactin-50], [Voveran-50], [Novatab-50] were weighed and powdered. The tablets powder equivalent to 50 mg of Diclofenac sodium were calculated, take double quantity for three brands and transferred to, three 50 ml different volumetric flasks. 25 ml of acetonitrile was added to each flask and shaking for 15 minutes, finally the volume was made up to 50 ml with the same solvent. These solutions were filtered through Whatmann filter paper. The absorbance's of these solutions were measured at 220nm. We found percent purity at a concentration of 6µg/ml.

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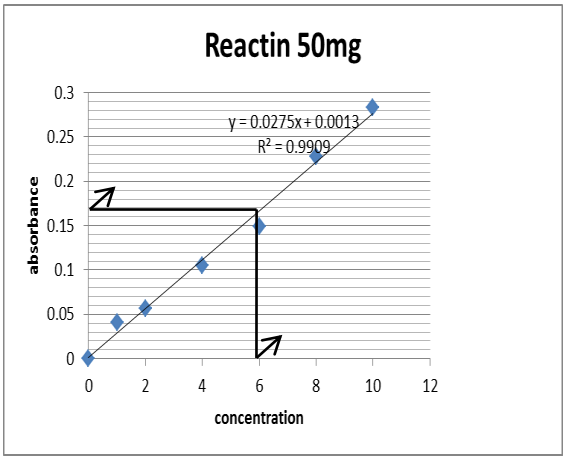

Figure.8 Absorbance curve of Reactin(50mg)

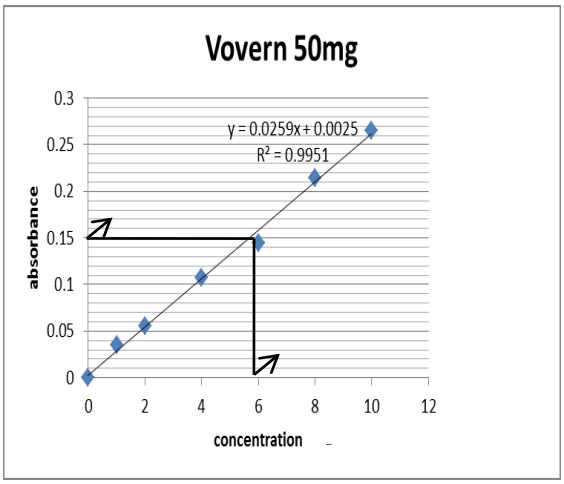

Figure. 9 Absorbance curve of Voveran (50mg)

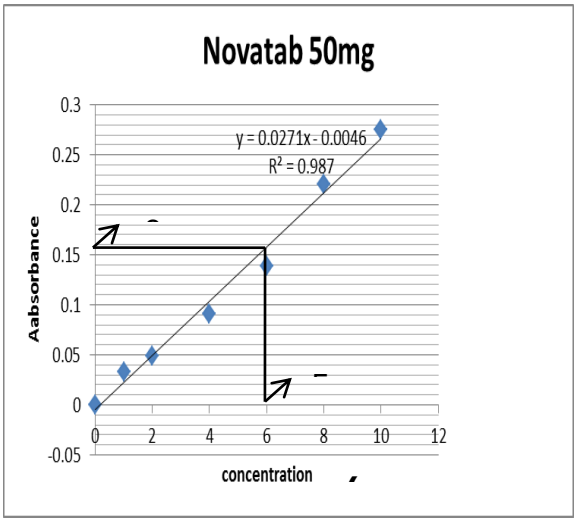

Figure.10 Absorbance curve of Novatab(50mg)

Brand name	Labelled amount(mg)	Amount found(mg)	%Purity of drug
Reactin 50	50	49.965	99.99
novatab 50	50	47.63	95.82
vovern 50	50	49.28	99.12

Table.9 percentage purity of different brands of diclofenac sodium at 6µg/ml.

RESULTS AND DISCUSSION

Diclofenac sodium is freely soluble in Acetonitrile. UV absorption spectra of diclofenac sodium at a concentration of 6µg/ml show a maximum absorbance at 220 nm in Acetonitrile. Linear correlation was observed between absorbance and concentration of Diclofenac sodium over the range of 2.0-10.0 µg /ml. The percentage recovery values indicate that the amount present in the formulation is not meeting its labeled claim. This method is useful for the determination of Diclofenac sodium in bulk and pharmaceutical dosage forms. The physical parameters like hardness, friability, weight variation are found in acceptable range as per I.P.

CONCLUSION:

Based on the results obtained in this work, the UV spectrophotometric method for determination of Diclofenac sodium is specific. The amount of drug obtained and percentage purity is less when compared to that of labeled claim. Hence it is concluded that the developed method is simple, precise and accurate method can be effectively used for the routine analysis of Diclofenac sodium. The UV spectrophotometric method is an alternative to determine Diclofenac sodium in pharmaceutical dosage forms that contain it as unique active principle with quite satisfactory results for the specific purposes of its design. Its advantages over other existing methods are its simplicity, fastness, low-cost and non-polluting conditions.

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